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# A TEXT-BOOK OF MEDICINE



*SECOND EDITION*

G. DIEULAFOY



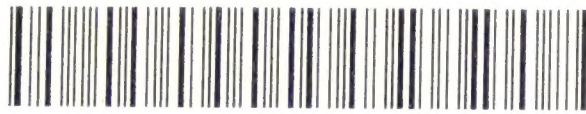
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# A TEXT - BOOK OF MEDICINE

BY

G. DIEULAFOY

PROFESSOR OF CLINICAL MEDICINE AT THE FACULTÉ DE MÉDECINE DE PARIS; PHYSICIAN  
TO THE HÔTEL DIEU; MEMBRE DE L'ACADÉMIE DE MÉDECINE

SECOND EDITION

*Authorized Translation from the Sixteenth Edition of "Manuel de  
Pathologie Interne"*

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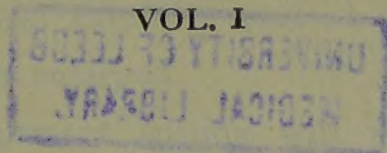
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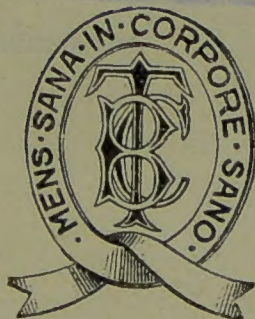
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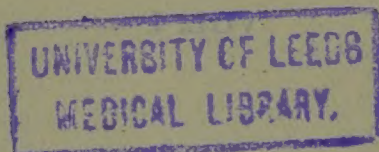
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## PREFACE TO THE SECOND ENGLISH EDITION.

THIS edition is translated from the sixteenth French edition, which was published just before the death of the distinguished author.

The following sections are entirely new: Syphilitic mediastinitis, Mediastinal syphiloma, Pachypleuritis and inexhaustible pleurisy, Pathomimia, Syphilitic meningitis of the base of the brain, Syphilitic polioencephalitis, Streptococcic septicæmia, Staphylococcic septicæmia, Gonococcic septicæmia, Pyloric stenosis, Sporotrichosis, Hæmolytic icterus.

The question of recent therapeutics is discussed in detail. A section has been devoted to the use of superheated air in gangrene, arthritis, and neuralgia. The study of vaccines, including the opsonic index and Wright's vaccines, is carefully described. The new arsenical preparations, hectine and Ehrlich's "606," complete the therapeutics of syphilitic infection; Wassermann's reaction finds a place in this section.

Every chapter has been carefully revised and brought up to date; the index has been much enlarged.

We trust that the present edition will receive the same cordial welcome extended to its predecessor.

V. E. COLLINS,  
J. A. LIEBMANN.

*August, 1912.*

28403







## AUTHOR'S PREFACE TO THE FIRST ENGLISH EDITION.

HAVING been asked for a preface to the English translation of my "Text-Book of Medicine," my first desire is to express the great satisfaction I feel at seeing my work translated into English, for that language is so widespread over both hemispheres that no other translation which has yet been made would have contributed to the same extent to the recognition abroad of this work of French origin.

I must add that in writing my "Text-Book of Medicine," which has now reached its fifteenth edition, I have given much space to the works of my eminent English colleagues, since a number of them are of the very first importance. Thus will be found the illustrious names of Sydenham, Graves, Basedow, Corrigan, Addison, Bright, Stokes, Paget, etc., whose scientific researches are known and appreciated by everyone.

Long ago my venerated master, the great Trousseau, popularized in his "Clinical Lectures" the valuable discoveries of English practitioners, and gave the names of their authors to the newly diagnosed diseases. I have followed the example of Trousseau, and have often dipped into English medical literature, so rich in precious documents. I shall be satisfied if the medical profession in England will on their side give a good reception to the work which I am now presenting to them.

G. DIEULAFOY.

PARIS.







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# TEXT-BOOK OF MEDICINE

## PART I

### DISEASES OF THE RESPIRATORY SYSTEM

#### CHAPTER I

#### DISEASES OF THE NASAL FOSSÆ

##### I. CORYZA.

**Coryza**, or nasal catarrh, is the term applied to inflammation of the pituitary mucosa.

##### 1. Acute Coryza.

**Description.**—**Acute coryza** shows itself by frontal headache and a feeling of obstruction and of tickling in the nasal fossæ. At first **sneezing** is frequently repeated, and brought on again by the slightest impression of cold. The nasal mucosa, at first dry, soon secretes a clear and irritating liquid, causing erythema of the parts over which it flows. The nose is shiny and swollen; the senses of **smell** and of **taste** are blunted or abolished. The patient constantly keeps his mouth open, whilst his breathing is difficult and noisy. Suction and deglutition are rendered difficult by the blocking of the nasal passages. These troubles are insignificant in the adult, but become serious in the infant, who cannot take the breast without suffocating.

The inflammation spreads in various directions: to the frontal sinuses (sharp pains in the head); to the conjunctivæ (injection of the eyes and lachrymation); to the mucous membrane of the Eustachian tubes (auditory troubles, buzzing noises).

General malaise and transient fever may also be present. By the second or third day the cold is **ripe**: the nasal secretion becomes thick and greenish, crusts form, the patient's nose is blocked up, his voice has a nasal twang, and herpetic vesicles often appear round the nostrils or the lips. Towards the end of the first week the coryza ceases. When the inflammation reaches the larynx and the trachea, it produces laryngitis and tracheitis; in vulgar parlance, "the cold has settled on the chest."

**Ætiology—Diagnosis.**—Cold and damp weather, the first sunshine of spring, chills of every kind, but especially of the feet, are the common causes of coryza. Influenza at its onset, measles during the period of invasion, and the iodides, produce a nasal catarrh which differs in nature from true coryza.

Some **asthmatics** are suddenly seized with violent and repeated fits of sneezing. During the fit, which lasts from a few minutes to a quarter of an hour, the eyes are injected and watery and the nose runs freely; but after this fit, which **precedes** or **replaces** the attack of asthma, everything returns to normal. True coryza differs as much from this manifestation of asthma as it does from **hay-fever**, or spasmodic rhino-bronchitis. This malady, which in England has been described under the name of hay-fever, and in which there is a nervous element besides the catarrhal one, is often the ap-panage of gouty or asthmatic patients, and usually appears at the beginning of summer. It presents two chief forms, which may follow one another or be present together—on the one hand, nasal catarrh, with abundant secretion, uncontrollable sneezing, pricking sensations in the eyes, injection of the conjunctivæ, violent headache and insomnia; on the other hand, dyspnœa, resembling that of asthma and playing the chief part, while the nasal catarrh may be of secondary moment. Hay-fever will be discussed in detail under Diseases of the Bronchi.

We must not confuse coryza with the nasal forms of diphtheria, of blennorrhagia, and of glanders. When **diphtheria** attacks the nasal mucous membrane, it produces an abundant secretion, which is sometimes sanguineous. On examining the nasal fossæ, we can usually discern the diphtheritic membrane, and bacteriological examination shows the presence of Löffler's bacillus.

**Glanders** likewise produces a specific coryza: the nasal mucous membrane is swollen, excoriated, and ulcerated; nasal respiration is impossible, and sanious, foetid liquid flows from the nostrils. This discharge, which constitutes a sign of the highest importance in animals, is much less profuse in man, and its presence would not suffice to establish the diagnosis if the cutaneous eruptions and the articular manifestations of glanders were absent.

**Treatment.**—Inhalations of vapour of iodine and of ammonia have been extolled. In infants at the breast care must be taken to cleanse the nasal fossæ of the secretions which hamper the movements of sucking and swallowing.

Some relief may be obtained from the following powder:

|                       |    |    |    |    |    |        |
|-----------------------|----|----|----|----|----|--------|
| Salicylate of bismuth | .. | .. | .. | .. | .. | 3iv.   |
| Powdered camphor      | .. | .. | .. | .. | .. | 5i.ss. |
| Cocaine hydrochlorate | .. | .. | .. | .. | .. | gr. i. |



The pricking sensations and the secretions are relieved by the use of the following ointment twice daily :

|                    |    |    |    |    |    |    |          |
|--------------------|----|----|----|----|----|----|----------|
| Menthol ..         | .. | .. | .. | .. | .. | .. | gr. ii.  |
| Lanoline ..        | .. | .. | .. | .. | .. | .. | gr. xlv. |
| Vaseline (pure) .. | .. | .. | .. | .. | .. | .. | ʒii.     |

## 2. Chronic Coryza.

**Description.**—This form may follow attacks of acute coryza, or may be chronic from the outset.

The sneezing, the frontal headache, and the fever of acute coryza are wanting, but there is no lack of other signs. The nose is stuffed up, so that the patient can only breathe with his mouth open, especially at night. Respiration is embarrassed, noisy, and sometimes accompanied by a kind of snoring sound ; the voice is nasal ; the senses of taste and of smell are blunted ; the hearing is less acute, and buzzing in the ears is frequent. The nasal secretion may be scant or abundant. In chronic **dry** rhinitis, which is most common in adults, secretion is almost absent, and patients complain of a distressing feeling of dryness. More commonly, especially in young subjects, the nasal secretion is abundant, and the nasal mucous membrane is covered with crusts and dried mucus. Chronic coryza has a slow course and an indefinite duration ; it is sometimes interrupted by acute attacks, and at other times more or less lengthy remissions give some respite to the patient. We shall see later the frequency of **ozæna** in chronic coryza.

On rhinoscopic examination, the mucous membrane appears red and hypertrophied, especially over the anterior part of the inferior turbinate bone (hypertrophic rhinitis). When the coryza is of very long duration, the mucous membrane is indurated and fibrous, while the glandular elements show a tendency to disappear.

Under the name of **posterior** coryza, Desnos has described a chronic inflammation of the naso-pharyngeal cavity. This variety is chiefly associated with glandular angina ; the local lesions can only be discerned by rhinoscopy, and its chief symptoms are snuffing and hawking.

In lymphatic children a chronic **herpetic** coryza has been described. The orifice of the nostrils and the septum are covered by crusts like those of impetiginous eczema. After the crusts fall off, they leave bare slight ulcers, which become covered with new crusts, and the duration of the illness is indefinite.

The changes in the so-called scrofulous coryza of infants are characterized by hypertrophy of the nasal mucous membrane which shows fungating vegetations, by deep ulcers which may attack the bones, by deformity and flattening of the nose. This old description must be revised, for most of these lesions are due to tubercular disease, or to **early or late hereditary syphilis**.

**Diagnosis.**—In the following chapters we shall give the diagnosis of chronic coryza from nasal syphilis and tuberculosis. It is enough for me to mention here that chronic coryza has many symptoms in common with **adenoid growths** of the naso-pharynx and with **mucous polypi** of the nose. Rhinoscopic examination will remove all doubts.

**Treatment.**—The local treatment of chronic coryza consists in cleaning the mucosa by means of appropriate douches; cauterization, powders, and snuffs are then employed. Lavage may be carried out by a siphon douche, the nozzle of which fits the nostril tightly; the fluid, introduced **without force**, into one nostril, flows back through the opposite one without passing into the pharynx. These douches consist of lukewarm saline solutions, weak boric lotions, or solutions of chlorate of potash. The cures at Challes and Mont-Dore are of much service. As a snuff, one of the following preparations may be recommended:

|                       |    |    |    |    |    |            |
|-----------------------|----|----|----|----|----|------------|
| 1. Bismuth subnitrate | }  | .. | .. | .. | .. | āā ʒii.ss. |
| Venetian talc         |    |    |    |    |    |            |
| Precipitated chalk    |    |    |    |    |    |            |
|                       | .. | .. | .. | .. | .. | gr. v.     |
| 2. Chlorate of potash | .. | .. | .. | .. | .. | gr. xlv.   |
| Powdered sugar        | .. | .. | .. | .. | .. | ʒv.        |

Arsenical preparations, taken internally, are useful when the rhinitis is associated with the gouty diathesis.

## II. NASAL DIPHTHERIA.

**Description.**—Nasal diphtheria is nearly always associated with diphtheria of the fauces, which it may precede or follow. It begins somewhat like a simple coryza, with redness of the nostrils and nasal discharge. There is but little sneezing. The nasal discharge is sanious, muco-purulent, or sanguineous, and contains membranes. Epistaxis is common, and usually precedes the formation of membranes. Repeated and profuse epistaxis was thought by Trousseau to be of evil omen.

The patient's voice is nasal, and he can only breathe with his mouth open.

The submaxillary glands are swollen. On rhinoscopic examination, the mucous membrane is swollen and coated with membrane, which is adherent, and usually localized to the posterior half of the nasal fossæ.

In some cases, especially in children with measles, the diphtheria may reach the nasal duct, and spread to the eyelids and the eyes. This **oculo-palpebral** variety shows itself by lachrymation, with redness and swelling of the conjunctiva and of the eyelids. Sero-purulent secretion appears, the eyelids and the ocular conjunctiva are covered with false membrane, and in some cases perforation of the cornea and destruction of the eye result when streptococcal infection is also present.



Nasal diphtheria, described by the term "fibrinous rhinitis," may be of such duration as to be chronic.

The **diagnosis** of diphtheritic coryza is easy when the patient is already suffering from pharyngeal or laryngeal diphtheria; if coryza be the first symptom hesitation is pardonable. The diagnosis is very difficult, especially at the commencement, and I know **only one way of settling the question**—viz., by culture and bacteriological examination of the membrane or of the nasal mucus (*vide* Diphtheritic Angina).

The **prognosis** is not very grave if the disease remain limited to the nasal fossæ, and if the diphtheria bacillus be present alone without the streptococcus. If both organisms are present, the membranes usually present a different aspect, being diffuent and gangrenous, while the discharge is profuse, persistent, and accompanied by epistaxis. Such a coryza usually points to very severe diphtheria, and is one of the manifestations of Trousseau's malignant diphtheria. Löffler's bacillus finds so favourable a soil in the nasal fossæ that its localization in the nose appears to me to have great weight in determining diphtheritic paralysis.

We often find virulent bacilli in the nasal cavities several weeks after the cure of diphtheritic angina.

Diphtheritic coryza, like all the varieties of diphtheria, should be treated with injections of **serum**. The earlier the injection, the better the chance of success; hence the importance of making a correct diagnosis from the very first.

### III. NASAL SYPHILIS—SYPHILITIC RHINITIS.

In this chapter I shall review (1) the primary lesion, the chancre; (2) secondary troubles; and (3) tertiary troubles.

#### 1. Nasal Chancre.

**Description.**—The study of nasal chancres comprises chancre of the skin of the nose and chancre of the nasal fossæ. Every syphilitic chancre is composed of a mass of embryonic cells, and forms a small tumour (primary syphiloma), developed at the expense of the skin and of the subcutaneous tissue. The appearance of the chancre differs, however, on the skin and on the mucous membrane. On the skin it is covered by a crust, due to the presence of the horny layer, which does not exist in the mucosa; the lesions of the mucous epithelium, soaked with fibrino-purulent liquid, end, not in the formation of a crust, but of a false membrane, which is flaccid, greyish, and diphtheroid.

1. Let us first consider **chancre of the skin**. It is situated on the bridge of the nose, on the nostrils, on the tip of the nose, or on the

nasolabial fold. On the bridge the chancre is flat; elsewhere it is bulky, prominent, and spread out, forming a hypertrophic papule (Fournier). It runs the same course as all cutaneous chancres: at first it is a crack, or a reddish erosive papule, then it grows larger, becomes prominent and encrusted, but always remains painless.

This encrusted or pustulo-crustaceous chancre has the appearance of ordinary ecthyma. If the crust be raised, after having been first softened, the chancre appears with all its characteristics—viz., flat or slightly convex surface, erosive, but not ulcerated, smooth, reddish, often bleeding and covered with papillæ. Sometimes a scanty purulent secretion may be noticed. The edges of the chancre are flat, not perpendicular; strictly speaking, there is no edge, because there is no ulceration. Its base is indurated and parchment-like. Adenopathy is constant and may be unilateral or bilateral; the condition is somewhat indolent, and is confined to the glands at the angle of the jaw, while one gland is often much larger than the others.

After about two months the chancre heals without cicatrization; the induration and the adenopathy, however, persist for a long while.

The nasal chancre must not be confounded with a furuncle. From the first the part on which a boil is about to develop is swollen, painful, red, and shiny; later it becomes purulent—characters which are quite different from those presented by a chancre. Chancre will not be mistaken for epithelioma, as the latter is a painful tumour, infinitely slower in its development, ulceration is delayed, the base is not indurated, and enlarged glands do not appear till much later. Epithelioma does not tend to spontaneous cicatrization, as chancre does.

2. Let us now consider **chancre of the nasal fossæ**. This chancre always occurs on the anterior or posterior part of the nasal fossa. I merely mention chancre of the posterior orifice of the nasal fossa from accidental inoculation by an infected Eustachian catheter.

Chancre of the vestibule usually arises on the cartilaginous septum. It develops as a greyish or reddish lump, which may be indurated or softened, and has roughly the appearance of a mushroom. This chancre, in its hypertrophic form, partly fills the nostril and causes an ichorous or bloody discharge. The nose is red and swollen; though it is deformed externally, no **deviation** of the septum is present. Radiation of pain to the nose or the face is common. Enlarged glands at the angle of the jaw are always found. The chancre lasts from six weeks to two months, and then heals without cicatrization.

The diagnosis between chancre and malignant tumour of the nasal fossa (sarcoma) must be based upon the following considerations: The evolution of sarcoma is much slower than that of chancre, nasal hæmorrhage



may be frequent and profuse, ulceration appears late, pain is acute, enlarged glands appear slowly; lastly, sarcoma deforms the nasal fossæ and causes deviation of the septum.

## 2. Secondary Troubles—Syphilitic Coryza.

I shall describe first cutaneous syphilides, and secondly those of the mucous membrane.

1. Cutaneous syphilides show here, as elsewhere, different forms, and may be dry, moist, or crusted. The dry syphilides are papular, papulolenticular, or papulo-squamous. They are frequent on the *alæ nasi*, where they often present the granular form (Fournier).

The moist, erosive syphilides, or mucous patches, appear as cracks and clefts in the *ala nasi* and the naso-labial fold. Syphilides which are crustaceous, papulo- or pustulo-crustaceous may invade the whole of the nose.

All these syphilides are indolent in character and do not itch. They are rarely confined to the nose alone, and are generally found on the cheeks, the chin, or the forehead.

2. Secondary syphilis of the **mucous membrane** presents a different aspect, according to whether it attack the adult or the new-born infant, as early hereditary syphilis.

In the adult we find bright red erythema of the mucous membrane and erosions, with muco-purulent or muco-sanguineous secretions, and with formation of crusts, which reform after they have been rubbed off. These crusts may block the nostrils, impede respiration, and simulate eczema.

In the **newly-born** coryza is often the **first sign of hereditary syphilis**. It appears a few weeks after birth. The child breathes with great difficulty through the nostrils, and the movements of sucking are hampered; but so far there is little distinction between syphilitic and simple coryza. Sero-purulent, and in some cases bloody, fluid exudes from the nose; the secretion becomes more and more sanious, without being profuse, irritates the *alæ nasi* and the upper lip, and produces ulcers covered by yellowish or greenish crusts, which may hide the upper lip. Nasal syphilis in the new-born, in contradistinction to what is seen at a later age, rarely results in tertiary lesions and deformities (Trousseau). Specific coryza, however, may become chronic, with thickening of the mucous membrane, ulceration and swelling of the adenoid tissue. Perichondritis of the septum, destruction of the vomer, and obliteration of the lachrymal duct, have also been seen (Ziem).

We must recognize syphilitic coryza of the new-born in order to treat it without delay, and to avoid the possible contamination of the nurse from the child. Coryza rarely exists as the only manifestation of syphilis, and is usually accompanied or followed by other troubles which are an aid to diagnosis. We must, therefore, examine the child's body for skin eruptions,

roseola, erythema of an ashy tint on the neck, hands, or feet, and patches which are scalloped, do not itch, and are often scaly. We may sometimes find mucous patches round the anus or the navel, behind the ears, upon the scrotum or the labia majora, and Trousseau used to lay stress upon unhealthy fissures and ulcers of the folds of the skin. From this time many children with hereditary syphilis show changes in appearance: their bodies take on a brownish colour, the eyebrows fall out, and are replaced by the yellowish, scaly plaques of psoriasis. These marks of hereditary syphilis will aid in the diagnosis of syphilitic coryza.

### 3. Tertiary Troubles of Nasal Syphilis.

The tertiary lesions of nasal syphilis which may result from the acquired or hereditary disease are in each case identical. As, however, syphilis may invade the nose or the nasal fossæ, it is necessary to study each form.

1. **Tertiary Syphilis of the Nose.**—The skin lesion here is essentially the **tubercle**, which much resembles the chancre in structure, and is composed of a mass of embryonic cells. The syphilitic tubercle is a cutaneous **gumma**, and forms a small tumour of the size of a millet-seed, a small pea, or a cherry-stone. This gumma, which is, as it were, set in the skin, is at first firm and resistant, and projects above the surface of the skin. The syphilitic tubercle is rarely solitary, and variable numbers are seen in the same region; they may be grouped in a mass, form segments of a circle, or sometimes fuse together and produce a true **gummatous infiltration**, “a kind of plaque of hyperplastic integuments, studded or bordered by tubercular nodules.”

These dermatoses are common upon the face, but the nose is the “victim by choice” (Fournier). They are sometimes confined to one part of the nose; at other times they invade the whole nose and spread to the face. The lesion, as we have said, appears as a tubercle (nodular syphiloma) or an infiltration (diffuse syphiloma). For several months the disease is insidious in its development; pain, fever, enlargement of glands—in short, all signs—are absent. The nose, however, loses its normal aspect, becoming deformed and increased in size, while the affected skin is studded by dull red projections, and slight desquamation of the epidermis occurs. At a more advanced stage the dermatosis may present different aspects: in rare cases the tubercles, especially those which are isolated, spread, become indurated or almost horny, and end in atrophy. As a rule, however, both the nodular and the diffuse syphiloma, when untreated, end in softening, ulceration, suppuration, and formation of crusts. These syphilides are known as **tuberculo-ulcerating** and tuberculo-crustaceous, or as the **gummatous ulcer** which is serpiginous and perforating.

This phase, in which the gummatous tissue softens and ulcerates, is



relatively rapid, and contrasts with the slowness of the preceding phase. At this period the nose is deformed, enlarged, budding out at one spot, ulcerated at another, sometimes gullied by purulent ulcers, and covered in places by brownish or greenish crusts, which are thick, stratified, and adherent. In spite of these lesions, we usually notice no functional trouble, no fever, and little or no pain.

When the lesions are deep, and especially when they have not been treated in time, tertiary syphilis leaves indelible marks: the *alæ nasi* are scalloped and destroyed; the nose is deformed, flattened, thinned, and furrowed with hard, white, and honeycombed scars.

**2. Tertiary Syphilis of the Nasal Fossæ.**—The lesions usually commence in the mucosa, and then invade the perichondrium, the cartilages, the periosteum, and the bone. The cartilage and the bone may be attacked from the outset. Sometimes the disease produces only superficial ulcers, which are not serious and readily recover, but at other times it destroys by invasion and perforation. It attacks the bony and the cartilaginous **framework** of the nose, lays bare the cartilages and the bones, gives rise to sequestra, and mutilates the organ, causing irremediable destruction.

These lesions must be studied separately, for they present different symptoms according to the region affected.

(a) *Hypertrophic Syphilitic Rhinitis.*—The picture is one of chronic coryza: nasal respiration is incomplete or impossible, the patient wipes away thick mucus with crusts, and acute attacks sometimes supervene. On examination of the nasal fossæ, we find hypertrophy of the mucosa; but this hypertrophy, which may be polypoid, must not be confounded with adenoid vegetations. The rhinitis is not always general, and may confine itself to one fossa, or to the inferior turbinate bone which fills the meatus. Ozæna often accompanies this syphilitic rhinitis.

(b) *Perforation of the Septum.*—The nasal fossæ are separated by a bony and cartilaginous partition; the bony part is formed, above by the perpendicular plate of the ethmoid, below by the vomer, while the cartilaginous part is formed by the triangular cartilage, which fills up the gap left by the bony plates. The cartilage is usually first affected by syphilis: the lesion begins in the mucosa, reaches the perichondrium, which it lays bare, sets up necrosis of the subjacent cartilage, and then causes a round or oval perforation of variable size. In some cases the lesion extends to the vomer or the ethmoid, whence sequestra and more or less extensive losses of substance result. This process goes on insidiously, without pain and under cover of the deceptive symptoms of chronic coryza.

Syphilitic perforations of the septum must be **diagnosed** from those due to other causes. We see a simple perforating ulcer, which begins simultaneously on both sides of the septum at a symmetrical spot. It

always respects the bony septum, frequently causes epistaxis, and appears to be associated with the presence of staphylococci or streptococci. The tubercular ulcer—and I speak now of primary tuberculosis exclusively—affects the nasal septum, and hardly ever its bony part. The malady appears as a fungating, mushroom-like growth, which causes perforation of the septum. The edges of this ulcer are prominent, sanious, and obstruct both nostrils. Bacteriological examination may reveal the presence of bacilli. Perforation of the septum may also result from typhoid fever.

(c) *Destruction of the Subseptum.*—The subseptum of the nose is formed by the cartilage of the septum, against which the horseshoe-like branches of the cartilages of the alæ nasi rest. When the subseptum is destroyed by a syphilitic lesion, the two nostrils have one triangular gaping opening, and, as the point of the nose is no longer supported, it bends back, and assumes the curved appearance of a parrot's beak (Fournier).

(d) *Destruction of the True Bones of the Nose.*—The root is formed by the true bones of the nose. When the bony framework is attacked by syphilitic necrosis and disappears, the soft parts sink in—the nose looks as though the bridge had been crushed in by the blow of a hammer—and the tip turns up. “This deformity is a certificate of syphilis” (Fournier).

(e) *Destruction of the Cartilaginous Vault.*—Below the true bones which form the upper framework there is a cartilaginous roof, which forms the inferior framework of the nose. When the cartilage is destroyed by syphilis, the lower half of the nose sinks in and is drawn backwards; the lower segment is invaginated into the upper segment. By pulling the tip of the nose, the normal shape can be restored for a moment, but the invagination at once recurs. Fournier has called it the “opera-glass” nose. Two remarkable examples were seen in my wards at the Necker Hospital and at the Hôtel Dieu. This special shape is said to be due, according to some writers, to the absence of the cartilaginous framework; but, according to others, it is chiefly caused by retraction of the cicatricial tissue.

(f) *Lachrymal Tumour.*—Nasal syphilis may produce an exostosis of the lachrymal bone and of the nasal process of the superior maxilla. Stenosis of the nasal canal with lachrymal tumour results.

(g) *Perforation of the Palatine Vault.*—The floor of the nasal fossæ, like the vault of the palate, is formed by the superior maxillary bones, which articulate behind with the palate bones. As the result of ulcerating syphilides, or periosteal gummata of the floor of the nasal fossæ, a part of the superior maxilla is laid bare; the bare bone necroses, a sequestrum is formed, and an abscess results on the buccal aspect, between the bone and the mucous membrane. This abscess projects into the mouth as a small indolent tumour. It may open itself or have to be opened. We then find, with the probe, that there is a sequestrum, which may be movable or fixed, and



when the sequestrum is detached, a **perforation** results. This perforation generally appears in the middle line, a little in front of the palate bones. Its shape may be round or oval, and in size it may be as large as a pin's head, a lentil, a sixpenny-piece, or even larger, for it may invade a part of the palatine vault.

There are two quite distinct phases in this process. During the first phase, which is very slow, the process is purely nasal; the lesion shows itself by chronic coryza, with muco-purulent secretion, appearance of crusts, and formation of sequestra, which are easily recognized by exploration with a probe. Ozæna is a very common symptom; the fœtor is sometimes appalling, and the stench improves, or ceases, only after elimination of the dead bone.

This **nasal** phase, which for a long time is insidious and almost painless, is followed by a **buccal** phase, in which we see the formation of the palatal abscess and perforation of the arch. As soon as perforation occurs, two new symptoms appear: the voice becomes nasal, and solids or liquids taken by the mouth pass back through the nose.\*

It was long believed that the perforation of the palate in syphilis proceeded from the mouth to the nose. This is an error. The lesion starts in the nose, and the perforation proceeds towards the mouth (Fournier, Duplay). When the perforation is small, specific treatment may succeed in closing it; when it is large, surgical intervention is necessary.

(h) *Naso-cranial Syphilis*.—Under this heading I shall describe the syphilitic lesions which, at first limited to the roof of the nasal fossæ, later attack the organs of the cranial cavity.

The vault, or upper wall, of the nasal fossæ forms a narrow gutter, composed of several bones, of which the cribriform plate of the ethmoid and the sphenoid play, in the present question, the chief part. Gummatous osteoperiostitis in this region may readily extend to the cranial cavity.

If the lesion remain limited to the upper wall of the nasal fossæ, we only find nasal symptoms. This stage is sometimes painless, at others accompanied by nasal or frontal pain and intense headache. The symptoms are those of a chronic coryza, with discharge and crusts, and, in addition, epistaxis, ozæna, and elimination of sequestra. Ozæna is, however, rarer in this variety of nasal syphilis than in the others which we have already studied. The use of the probe is not always free from accidents, and I have had under my care a woman in whom it produced giddiness, acute frontal pain, and almost syncope.

The lesion may long remain confined to the roof of the nasal fossæ, and affect the sphenoid and the ethmoid, without causing cerebral troubles; but in other cases symptoms of meningitis and of encephalitis may supervene at any moment. Frontal pain, which sometimes is very

\* For further details, see chapter on Perforation of the Vault of the Palate.

sharp, paralyses of the motor or sensory cranial nerves, vomiting, epileptiform convulsions, apoplexy, and fatal coma, are the complications which may appear, slowly or with extraordinary rapidity, and make up the cerebral phase of syphilitic naso-cranial osteitis.

Autopsies show the cause of these severe troubles. Osteitis and necrosis of the sphenoid and ethmoid bones, phlebitis of the coronary, cavernous, or petrous sinuses, phlebitis of the ophthalmic vein, intra-orbital suppuration, purulent meningitis, and encephalitis of the frontal lobes are often found.

The preceding descriptions suffice to show the great importance of nasal syphilis.

**Diagnosis.**—For want of attention, syphilitic rhinitis is often mistaken for a simple coryza. The patient thinks but little of his “cold,” and treats himself. Noting its tenacity, he seeks advice. If the physician do not recognize syphilis, he rests content with prescribing nasal douches, snuffs, and a cure at Challes or elsewhere, when he should employ mercury. As a general rule, every individual who for a long while wipes thick mucus or crusts from his nose should be suspected of syphilitic rhinitis, especially if ozæna be present. Exploration of the nasal fossæ may then show the presence of erosions, ulcerations, or more or less extensive losses of substance.

The diagnosis between tertiary nasal syphilis (hereditary or acquired) and tuberculosis of the nose is sometimes most difficult.

First, let us consider the “dry tubercular” lesions. We have two patients, the one suffering from lupus of the nose, the other from tubercular syphilis. On examination we find at first nothing but resemblances: in both cases an eruption of tubercles upon a red infiltrated base, in both cases tubercles identical in size and shape and grouped in like pattern. The diagnosis appears very difficult: the lupus tubercle, however, is more transparent, like barley-sugar, while the syphilitic one is of a dark reddish tint, like muscular tissue; the lupus tubercle is flabby and sinks in, while the syphilitic one is much harder to the touch; lastly, lupus is infinitely slower in its course than syphilis.

Let us now pass to the diagnosis of **ulcerating** lesions of the nose. We have two patients, one suffering from syphilitic ulcers, the other from those of lupus; in both cases the ulcers may be covered with crusts. How can a differential diagnosis be made? Here again the resemblances appear at first sight to outweigh the differences, but more careful scrutiny reveals points of distinction. The areola round the syphilitic ulcer is of a dull red, dark, and pigmented, while the areola round the lupus ulcer is clearer and more bluish. The edges of the syphilitic ulcer are hard, perpendicular, adherent, and not flattened, while those of the lupus ulcer are flat, thin, soft, flabby, moist, and not perpendicular. The floor of the syphilitic ulcer is hollow, anfractuous, and sloughy, while the floor of the other is almost flat.



In both cases the ulcer is covered with crusts, but the crusts of syphilis are more compact, harder, more stratified, more brownish-green and like oyster-shells than those of lupus. As regards progress, syphilis does in a month what lupus does in a year.

We now come to the diagnosis between the **destructive** lesions of syphilis and of lupus. Syphilis destroys the nose in large pieces, while scrofula eats it away atom by atom. The nose in lupus is symmetrically destroyed, and cut off as by a hatchet blow, from before backwards, from the lower level of the nasal bones to the upper lip; the aspect is that of a death's-head. Syphilis, on the other hand, causes more irregular and less symmetrical mutilation. Lupus may very rarely attack the mucous membrane, for primary lupus of the septum exists, but acquired or hereditary syphilis has a special tendency to attack this part. When lupus spreads to the nasal fossæ it is usually limited to the vestibule, the cartilages, and the floor of the maxilla; in any case it rarely attacks the nasal skeleton. Syphilis, on the other hand, does not show these restrictions, and the different bones of the nasal fossæ may fall under its blows. Flattening of the bridge, the "opera-glass" nose, and perforation of the roof of the palate, are almost always the result of acquired or of hereditary syphilis, and are not, as a rule, seen in tuberculosis.

The details into which I have entered regarding the differences between syphilitic and tubercular lesions of the nose prove that diagnosis is not always easy. The difficulty is still greater when both diseases are present together. Ricord, in his picturesque language, calls this "*Le scrofulate de verole.*" In some cases (even more frequent in the larynx and in the lungs) syphilis and tubercle successively invade the same organ, and the two infections are juxtaposed. In other cases, however, we might truly speak of a hybrid affection, as in certain syphilo-tubercular affections of glands and in certain lupoid lesions of the skin and the nasal mucosa, which are equally syphilo-tubercular. M. Leloir has quoted cases which appear to me conclusive. Treatment alone can settle the question.

All I have said of tertiary nasal syphilis is equally applicable to **late hereditary** syphilis, which supervenes at a more or less advanced age.

The nasal lesions of hereditary or acquired syphilis show themselves by troubles which are, on the one hand, temporary and curable; on the other, irremediable. These troubles—rhinitis, with its crusts and ulcers; ozæna, with its fearful stench; destruction of the soft and bony parts of the nose; perforation of the vault of the palate; nasal deformities; and the terrible consequences of naso-cranial syphilis—are the more formidable as they are usually latent in the early stages.

We cannot, then, use too much care in the **diagnosis** of nasal syphilis. These lesions, especially in dealing with the hereditary disease, are too often

taken for those of scrofula, strumous eczema, or lupus. Failing clear proof to the contrary, we must always think of syphilis and act accordingly.

**Treatment.**—The treatment of nasal syphilis does not differ from that of syphilis in general. Mercury is the essential agent. Mercury, rather than iodide of potash, is the drug for tertiary syphilis. For many years I have almost entirely given up iodide of potash and employed mercury. The most efficacious preparation is the aqueous solution of the biniodide. An intramuscular injection of this solution is given daily for a fortnight. Later the dose may be increased. The treatment is stopped for a fortnight, and then resumed again for a like period, and so on for three months. For further details, see Appendix on Therapeutics.

#### 4. Tuberculosis of the Nasal Fossæ—Lupus of the Nose.

##### (1) TUBERCULOSIS OF THE NASAL FOSSÆ.

**Description.**—Tuberculosis of the nasal fossæ is not very rare; its relative frequency is shown by the following table:

|             |    |    |    |    |           |
|-------------|----|----|----|----|-----------|
| Tongue      | .. | .. | .. | .. | 51 cases. |
| Pharynx     | .. | .. | .. | .. | 21 „      |
| Mouth       | .. | .. | .. | .. | 22 „      |
| Soft palate | .. | .. | .. | .. | 8 „       |
| Tonsils     | .. | .. | .. | .. | 4 „       |
| Nasal fossæ | .. | .. | .. | .. | 5 „       |

It may be primary or secondary.

(a) The **primary**, or at least the apparently primary, form is confined to the nasal fossæ, independently of any other region. It nearly always takes the form of a mushroom-like or polypoid excrescence, simulating sarcoma. It begins in almost every case on the cartilaginous septum, as a submucous infiltration, and the mucosa may remain unaffected for a long time. Later the mucosa ulcerates, and the growth forms a hernia of fungating, mushroom-like tissue, occupying both nostrils, which are partly obstructed.

The process spreads by eccentric growth: at the centre of the tumour softening and destruction occur; in the tissue around the tumour young granulations appear. When the process ends in perforation of the septum, the edges of the perforation are formed by fungating and encrusted masses of tissue.

The course of the lesion is slow and painless. The symptoms at first are those of chronic coryza. Later, when the fungating mushroom is formed, the question of diagnosis from chancre and from sarcoma arises. Chancre is unilateral, indurated at its base, accompanied by early enlargement of glands, and rapid in its growth, while the tubercular mass is bilateral, flabby, and very slow in its growth.

The diagnosis from sarcoma is clinically very difficult; careful search



must be made for the tubercle bacillus, which is sometimes only found after making many preparations.

The diagnosis of nasal tuberculosis must also be made in the ulcerated stage. To avoid repetition, I would refer the reader to the preceding chapter on Nasal Syphilis.

(b) **Secondary** tuberculosis of the nasal fossæ is much more frequent than the primary form. It supervenes in the course of pulmonary or laryngeal phthisis, and may also accompany tuberculosis of the mouth and pharynx. It rarely shows the polypoid, fungating appearances of the primary form. It usually gives rise to multiple ulcers, which are confined to the vestibule and the anterior part of the inferior turbinate bone. The ulcers vary in size; the edges are usually jagged, and the floor is greyish and covered with purulent secretion or crusts. Bacilli are rare in the primary form, but very abundant in the ulcers of the secondary variety. These ulcers are generally but little painful, and thus differ from bucco-pharyngeal tuberculosis, which gives rise to very severe pain.

Tuberculosis of the nasal fossæ *per se* is not of much gravity. In some cases, however, it forms the starting-place of tubercular meningitis.

Treatment which is ineffective in the secondary forms may be efficacious in the primary form. Extirpation, cauterization, scraping, and application of a concentrated solution of lactic acid would seem to be the best methods.

## (2) LUPUS OF THE NOSE.

Speaking generally, lupus vulgaris (Willan's lupus) is an attenuated tuberculosis of the skin and the adjacent mucosæ. It may be said that the varieties of cutaneous tuberculosis have but little virulence, and lupus is remarkable for its slow course and attenuated virulence. Lupus vulgaris, nevertheless, belongs to the tubercular group. Lupoid tissue contains very few bacilli, seeing that they are only found once in every eleven cases (Cornil and Leloir), and yet Leloir has nearly always succeeded in producing tuberculosis in animals by inoculation of the anterior chamber of the eye with fragments of lupus. Furthermore, the local reactions of tuberculin are evident in lupus, just as they are in true tuberculous lesions; this is a fresh proof of the tubercular nature of lupus vulgaris.

Lupus may invade the skin in diverse places, but the **nose is the "seat of election."** Sometimes the nose alone is invaded; at other times lupus extends to the cheeks or the lips.

**Description.**—Lupus vulgaris of the nose is characterized by the development of small intradermal nodules. These nodules, or lupus tubercles, form the primary element of the disease. They vary in size from a millet-seed to a lentil, or larger.

The tubercle is fairly transparent. In colour it looks like barley-sugar or apple-jelly. Its consistency is somewhat soft and easily appreciable to the touch. It runs a very slow course. In certain cases it undergoes interstitial absorption, and gives place to white depressed scars, which deform the nose ; but more often it undergoes ulceration, and produces loss of substance. It is by no means rare to find cicatrized tubercles, ulcers, and tubercles in full growth in the same area.

The tubercles often form a patch. In the simplest form of nasal lupus, the patch, I think, occupies the ala nasi ; it is reddish, round or oval, but the tubercles are not prominent. This type is known as **flat** or **macular** lupus, in which the ulceration is usually slight and superficial.

In other cases lupus, instead of being flat, is exuberant, the tubercles being prominent and highly coloured. This form, unlike the flat kind, has a great tendency to ulcerate, and is called lupus vegetans, exuberans, or exedens. From its invading nature it is sometimes named lupus vorax ; it attacks the nose, the cheeks, the lips, and the eyelids.

The ulcerations of lupus are indolent and of a reddish colour ; they have an irregular floor, carpeted by flabby granulations, bathed in fluid and covered with more or less thickened crusts. The ulcers are irregular, with infiltrated and livid edges. The crusts are greyish, sometimes brownish, thickened and stratified ; when they fall off, an ulcer, which is often studded with tubercles, is left uncovered.

The course of lupus is extremely slow, and the disease lasts years or decades. The ulcers are replaced by dark scars, which later become whitish and are traversed by prominent bands. These ulcers slowly determine the destruction of the soft parts of the nose, and the scars cause stenosis of the nostrils.

The **diagnosis** between lupus and syphilis has been given in the preceding chapter. The cure of lupus, either spontaneously or by treatment, is rarely permanent, and the lesions often reappear after several years of apparent cure.

Lupus vulgaris may invade the nasal mucosa primarily, or may be secondary to lupus of the skin. Primary lupus of the septum and of the subseptum is seen. Lupus of the nasal mucosa has the same slow and indolent course as lupus of the skin, and, while it may affect the cartilages, it is quite exceptional for it to attack the bones.

I must now say a few words about lupus erythematosus, which some consider to be a very mild cutaneous tuberculosis, while others deny its tubercular nature. It is still called acneiform lupus. In most cases it occupies the bridge of the nose and the adjacent part of the cheeks, having somewhat the shape of a butterfly. It begins as a red stain, and is covered with scales ; it spreads, and finally becomes colourless and atrophied with



the formation of glossy, whitish, superficial scars, without ulceration. Lupus erythematosus may be associated with lupus vulgaris.

The **treatment** of nasal lupus has benefited by the employment of phototherapy in dermatological practice (Finsen).

### 5. Ozæna.

**Ozæna** is the fœtid odour which proceeds from the nose in some people. It is, however, important not to confuse ozæna—that is to say, the smell which has its origin in the nose—with the fœtid odour which the breath acquires from the affections of the mouth and throat. Carious teeth, alveolar periostitis, inflammatory conditions of the gums, secretions which accumulate during the night on the buccal and lingual mucous membranes, also cause fœtor of the breath. The caseous products which block the tonsillar crypts are a cause of fœtor. I would mention, too, the foul breath which accompanies fœtid bronchitis and dilatation of the bronchi. Ozæna must not be confounded with these different conditions.

**Ætiology.**—Though we do not know the exact cause of ozæna, clinical cases teach us that in certain people ozæna exists, although ulceration, deformity, and atrophic rhinitis are not present in the nose. We might say that the nasal secretion in these people, just as the vaginal or pharyngeal secretions in others, and in yet others the perspiration of the axilla or of the feet takes on a fœtor, the primary cause of which is unknown.

In some individuals, chiefly herpetics, this tendency to fœtor of the nasal secretions may be absent in the normal state, but is at once awakened by coryza, and especially by chronic coryza. On the other hand, we see patients with ozæna in whom an acute coryza causes the fœtor to disappear—for a time, at any rate. Certain persons, from infancy or from puberty, suffer from ozæna which may be called “constitutional”; it is associated with a sero-purulent nasal secretion, persists with alternations up to adult life, and only diminishes at a very advanced age.

Ozæna is sometimes associated with ulcers of the mucosa, with lesions of the cartilages and the bones of the nose, and with the nasal deformities which arise from hereditary or acquired **syphilis**. This important variety of **syphilitic ozæna** has been studied under Nasal Syphilis.

In certain individuals ozæna may be considered as a true morbid entity, having its origin in a congenital malformation of the nasal fossæ (atrophic rhinitis). This variety would be true ozæna, other kinds being considered symptomatic. This malformation consists in a considerable enlargement of the nasal fossæ, with atrophy of the turbinate bones (especially the inferior ones) and thickening of the mucosa (Zaufal). This gaping of the nasal fossæ allows such a free passage to the current of expired air that the secretions are imperfectly swept away, the mucus becomes stagnant, and

crusts form. In this alkaline medium a special micro-organism (a large **diplococcus**) has been described by M. Loewenberg, who considers it to be the specific cause of the fœtor.

Ozæna does not always develop in the same way. Its evolution is more or less rapid. True ozæna, which seems to be due to a congenital malformation of the nose, generally shows itself during late infancy, when the nasal cavities are growing; it takes months and years to develop. The fœtor varies in different subjects, but is especially marked when crusts and mucus are expelled, and is so intense in certain patients that a whole ward may be tainted. As the sense of smell is lost, the patient is unconscious of the fœtor, but the disgust which he causes to those around him makes life miserable. The social consequences of this affection are terrible; life in common with others becomes difficult, marriage is impossible for young girls, and the tendency to suicide is not rare. The patient often speaks with a **nasal tone**, which results from the resonance of the voice in the abnormally enlarged nasal cavities. The nose is often saddle-backed, the tip is tilted, and the nostrils gape.

The infection which produces ozæna may be propagated in different directions, and give rise to dacryocystitis, conjunctivitis, keratitis, or sinusitis. M. Luc has described tracheal ozæna.

The **treatment** of ozæna consists in the daily use of a nasal douche of bichloride of mercury (1 part of mercury in 10,000 of water). After the douche boric powder (Loewenberg) is carefully blown into the nasal fossæ and upper parts of the pharynx. Aspirations of very hot saline solution morning and evening for several months give excellent results (Bonnier). In the case of syphilis mercury as described in the preceding chapter must be exhibited.

## 6. Epistaxis.

**Description.**—**Epistaxis** is hæmorrhage from the nasal mucosa. The bleeding, which is usually slight, consists in the flow of bright blood, drop by drop, from one nostril, rarely from both.\* If the bleeding be abundant, and especially if it occur while the patient is lying down with the head back, the blood flows through the posterior nares, and may then pass along the pharynx and œsophagus into the stomach, to be rejected by vomiting. A mistake may thus occur if the patient does not recognize the epistaxis, and is content with saying that he has vomited blood; hence the rule to explore the nasal fossæ carefully in doubtful cases of hæmatemesis. I have several times seen such errors in diagnosis.

This year, at the Hôtel-Dieu, one of my patients vomited fluid blood

\* Galen has noticed that the blood generally comes from the right nostril in diseases of the liver.



and clots; the hæmatemesis resulted from nasal hæmorrhage diverted into the stomach. On depressing the tongue we saw the blood flowing down the posterior wall of the pharynx, and examination of the septum revealed the source of the hæmorrhage, which was at once arrested.

The quantity of blood lost is very variable. In some cases the loss is slow, lasts from ten minutes to a quarter of an hour, and the subject does not lose more than one or two ounces of blood. In other cases the flow is rapid and profuse, appears from the nose and mouth at once, and may last several hours if efficacious treatment do not intervene; the loss may then amount to more than a pint. Furthermore, as the bleeding readily recurs—since any effort, such as blowing the nose or sneezing, is enough to detach the clots—it follows that in predisposed subjects (hæmophilia) the loss of blood may assume large proportions. When the epistaxis is repeated and abundant, the patient becomes pale and weak, and is liable to fits of dizziness, vertigo, and syncope, as is customary after any great loss of blood.

Nasal hæmorrhage has a most irregular **course**; it may appear daily, several times in the twenty-four hours, or months and years apart. It sometimes assumes an intermittent form, or returns at certain seasons of the year.

**Ætiology.**—Epistaxis is very common at the age of puberty, but is more rare in old age. The abundance and the distribution of the vessels on the surface of the mucous membrane explain the frequency of these hæmorrhages. Capillary aneurysms have been met with. Epistaxis may be active or passive. It is active when it is the result of an afflux of blood. I may quote, among other examples, the epistaxis favoured by the hypertrophy of the heart in aortic insufficiency; the supplementary epistaxis of the menstrual periods and of piles; the hæmorrhages which supervene after the suppression of a cutaneous affection, such as erysipelas (Sore); and those which are the result of sunstroke and of sudden changes of temperature or of atmospheric pressure. Epistaxis is frequent at the onset of typhoid fever and of measles, and is seen in the course of articular rheumatism (Trousseau) and of phthisis (Leudet); it is sometimes the herald of hæmoptysis. **Passive** epistaxis is seen in blood stases, when the venous tension is increased (mitral and tricuspid lesions), or when the return of blood from the head is impeded (compression of veins by tumours of the neck and the mediastinum).

Epistaxis in icterus simplex and icterus gravis, in the hæmorrhagic forms of the eruptive fevers, in malarial infection, purpura, and leucocythæmia results from **changes in the blood**. Epistaxis is frequent in **diabetes** and in **Bright's disease**; indeed, diabetes predisposes to great bleeding. In the chapter on Bright's Disease I shall dwell in detail on the epistaxis of chronic nephritis and on **severe epistaxis**, which is sometimes one of the first symptoms of Bright's disease. Epidemics of epistaxis, which could only

have been a larval fever, have been noted. The epistaxis which follows a fall upon the head sometimes indicates a fracture of the base of the skull, involving the upper wall of the nasal fossæ.

I must make special mention of epistaxis of local origin in ulcers of the mucosa, varices, angiomata, polypi, and vascular tumours of the septum.

Epistaxis is rarely the result of hæmorrhage from a wide area. Rhinoscopy shows in most cases that the rupture of the small vessels which gives rise to epistaxis occurs in certain defined regions—to wit, the antero-inferior and the central portion of the septum and the inferior turbinate bone, where erectile tissue exists.

**Diagnosis—Prognosis.**—It is necessary to trace the cause of the bleeding to ascertain if it depend on some general state or on a purely local lesion (ulcer, varix, erectile tumour, angioma of the septum), to find out whether it be symptomatic of some affection of the liver, the heart, or the kidney, or whether it be supplementary to a suppressed hæmorrhage, and to ask if it may not denote the onset of enteric fever, or if it be not the first sign of a hæmorrhagic form of some eruptive fever. The **prognosis** depends on the quantity of blood lost, the strength of the patient, and the cause of the bleeding. Each of these factors must be carefully considered. Epistaxis is sometimes a grave complication in measles and in typhoid fever.

**Treatment.**—To arrest epistaxis the simplest measures, such as injection of very hot water (especially oxygenated water) into the nasal fossæ or compression of the nose, sometimes suffice; but in obstinate cases we must resort to plugging, which exerts direct pressure upon the seat of the hæmorrhage. It is first necessary to make out the site of the bleeding by direct examination. In twenty-five cases of epistaxis Chiarri has shown that the hæmorrhage in twenty-two of them came from the anterior part of the septum. It is the “seat of election” in epistaxis, a seat easy to recognize, and we have only to look for it. “Raise the ala nasi, so that the orifice of the nostril is turned outwards as much as possible, follow an oblique line upwards and backwards, and at a distance of 1 inch the zone of bleeding will be found.” We then apply a plug of wool at the point in question, and we can exert efficient pressure by keeping the plug in place with a stop-clamp applied astride the bridge of the nose, or by introducing into each nostril one jaw of the clamp forceps, previously wrapped with gauze (Rangé). If this plugging do not suffice, or if the seat of the bleeding be higher up or farther back, we introduce into the nostril, by means of forceps, a series of plugs of absorbent wool soaked in oxygenated water: perchloride of iron must not be used. After plugging, we survey the posterior orifice of the nasal fossæ, so as to be certain that the blood does not continue to flow backwards into the pharynx. We can also perform plugging by means of a bladder of gold-beater’s skin. For this purpose fix the bladder on a rigid



urethral sound of small calibre, introduce the empty bladder into the nose, inject water through the sound, so as to distend the bladder and plug the sound. This apparatus is easily kept in place, and has often yielded me the best results.

If, in spite of these means, the epistaxis continue, posterior plugging must be employed. To plug the posterior nares use Belloc's sound, or a flexible urethral bougie. Introduce the sound, smeared with vaseline, into the inferior meatus, push it horizontally till it emerges behind the soft palate, seize it with forceps, and draw it out of the mouth. By means of a strong thread of sufficient length, suspend from this end of the sound a plug of wool smeared with boric vaseline, 1 inch long and half as broad. Next pull back the sound introduced into the nose. The gauze plug may be thus fixed in the posterior opening of the nose, while the index-finger, introduced into the mouth, helps the passage of the plug behind the soft palate. The thread is fixed to the cheek by means of diachylon or collodion; it is kept in place by anterior plugging, which is usually combined with posterior plugging.

The plug, however, soon becomes painful to the patient. The nose swells, breathing is impeded, and the patient can rarely tolerate the plugs for more than twenty to thirty hours. To withdraw them, moisten by injecting tepid water into the nostrils. The anterior plugs come out easily, while the posterior ones are detached by means of the injection, and are finally spat out by the patient.

In some cases (erectile tumour, angioma) plugging is not the best treatment, and the bleeding surfaces must be cauterized with nitrate of silver, or preferably with the galvano-cautery at a dull red heat. By this means M. Luc was able at one sitting to arrest profuse epistaxis which threatened the life of one of my patients admitted for an angioma.

Adrenalin must enter into the therapeutics of epistaxis. The bleeding mucosa is touched with a plug soaked in a solution of adrenalin (1 in 1,000). A plug of wool saturated with a weaker solution (say, 1 in 5,000 or 1 in 10,000) may also be left *in situ*. The vaso-constriction which results sometimes produces paleness from the decoloration of the mucosa.

When the hæmorrhage has been abundant, give iced drinks, use subcutaneous injections of ergotin, and administer, in tablespoonful doses, the following hæmostatic draught :

|    |                        |    |    |    |         |
|----|------------------------|----|----|----|---------|
| Rx | Acid. sulphuric. dil.  | .. | .. | .. | ℥ x.    |
|    | Tinct. ferri perchlor. | .. | .. | .. | ℥ x.    |
|    | Tinct. opii            | .. | .. | .. | ℥ v.    |
|    | Aquam                  | .. | .. | .. | ad ʒss. |

Employ **injections of serum** (see Appendix on Therapeutics).

If these means fail to stop the bleeding, and if the patient's life be in

danger, **transfusion of blood** must be employed. It is an operation which has often yielded me excellent results.

Some years ago I performed transfusion upon two patients : the one was suffering from epistaxis of diabetic origin, the other from bleeding due to hæmophilia. The former was an adult, the latter a child. In both cases the bleeding was exceedingly serious, on account of its amount and its persistence. The bleeding was at once arrested by transfusion.

Periodic epistaxis, even though it is not due to a larval fever, is successfully treated with sulphate of quinine. Lastly, it is necessary to remember that certain supplementary or critical bleedings must be respected, especially in old people, in whom they are often a safety-valve.



## CHAPTER II

### DISEASES OF THE LARYNX

#### I. GENERAL SURVEY OF THE ANATOMY AND PHYSIOLOGY OF THE LARYNX.

VOCAL and respiratory troubles comprise nearly the whole of laryngeal pathology. As we cannot understand aphonia and dysphonia, with all their shades, and cannot grasp the mechanism of spasm of the glottis and the genesis of paralyses of the vocal cords, or diagnose changes in the recurrent and external laryngeal nerves, if we have not a mental picture of the normal functions of the laryngeal apparatus, I must briefly indicate the principal points in the **physiology** of the larynx.

The skeleton of the larynx is formed by the cricoid, thyroid, and arytenoid cartilages. The cricoid is shaped like a ring, and is much broader behind than in front.

The thyroid has been compared to a shield : it protects the vocal cords, which are inserted in the angle of its posterior surface.

The arytenoids, which resemble a funnel in shape, play a most important part. They swing on their base at the crico-arytenoid articulation, and the opening or closing of the vocal cords is produced by their different movements.

**Function of the Glottis.**—The glottis is the space limited by the vocal cords (interligamentous glottis) and by the arytenoid cartilages (intercartilaginous glottis). The inferior cords alone merit the name of vocal cords, for sound is produced at their level ; the superior vocal cords have usurped the term “ cords,” which must be replaced by that of “ ventricular bands.”

All the parts of the larynx, including its cartilages and joints (passive organs), its muscles and nerves (active organs), combine for one purpose—*i.e.*, for the movements of the vocal cords and the different shapes of the glottis.

The glottis plays two chief parts—the one, which concerns the individual's life, is the passage of air into the respiratory channels ; and the other, which is an attribute of species, is the emission of sound, from the simple cry to the modulations of the human voice.

Respiration, therefore, on the one hand, and emission of sound on the other, are functions which show clearly the importance and gravity of diseases of the larynx.

**Muscles of the Larynx.**—The muscles of the larynx may be classified in several groups. The posterior crico-arytenoids form the first group. They are inserted into the posterior surface of the cricoid, and into the external and posterior process of the arytenoid. Their contraction draws the vocal processes outwards, especially if inspiration be very deep.

The posterior crico-arytenoids, then, are the muscles of **respiration** ; their rôle is to open the vocal cords, allow free passage to the air, and keep the glottis open during respiration. By their contraction they oppose the natural tendency of the lips of the glottis to come together, like two valves, during the aspiration of air into the lung, an event which at once happens when they are paralyzed.

Such is the isolated action of the posterior crico-arytenoid muscles as muscles of respiration. When they contract synchronously with the arytenoideus, they form a muscular band, which straightens the two arytenoid cartilages upon the cricoid. They thus become the antagonists of the thyro-arytenoid muscles, and free the arytenoids from the traction of the cords which would draw them forwards.

They may also be looked upon as **tensors** of the vocal cords. This tension is effected by their means, and without them would be impossible. Furthermore, by acting in concert with the adductors, the posterior crico-arytenoids fix the arytenoid cartilages on the cricoid, make their movements firm, and thus allow the vocal cords at their posterior insertion to follow the movements of the cricoid. They are, therefore, muscles of **inspiration, tension, and fixation**.

The second group is formed by the lateral crico-arytenoid muscles, which are the constrictors of the interligamentous glottis and the single ary-arytenoideus, which constricts the intercartilaginous and, consecutively, the interligamentous glottis. These muscles, by drawing the vocal cords together, shut the glottis, and place it in the desired position for the production of vocal efforts.

For this act the glottis is quite shut, while in the production of the voice the cords do not come quite in contact, but leave a space of variable shape and size between one another.

By bringing the vocal cords together the constrictor muscles of the glottis assist in the production of sound—they prepare it. Sounds can only be produced if the vocal cords be sufficiently approximated; if they be too open, the expired air escapes, and is wasted without being used in the production of sound. This fact can easily be verified upon the larynx of a corpse.

The third group comprises the muscles of **phonation**; these are the thyro-arytenoid and crico-thyroid muscles.

The thyro-arytenoid muscles are composed of two chief bundles, the one situated in the thickness of the vocal cord, the other outside it. This latter bundle, which is much the larger, is flattened against the thyroid cartilage, terminates at the outer edge of the arytenoid, and blends with the muscular loop, which the arytenoideus completes behind. The contraction of this muscular loop shuts the glottis. The thyro-arytenoid is, then, a muscle of **effort**; it is an **adductor**, like the lateral crico-arytenoideus, and it also plays a part in phonation, by bringing together the vocal cords, which the column of expired air seeks to open. It regulates the power of the voice by its opposition to the expiratory muscles of the thorax.

The internal bundle of the thyro-arytenoideus (the muscle of the vocal cord) draws the arytenoid and the thyroid cartilages together. It is not, therefore, a tensor of the vocal cord, neither does it stretch the cord at all; it shortens the vocal cord and loosens. instead of stretching it; but, as other muscles—the distensors of the vocal cords—begin to contract, it can no longer shorten, and as it cannot thicken or swell, it hardens and acquires a firmness and resistance which regulates the periodicity of the vibrations. The more the firmness is increased the greater become the vibrations and the higher the sound. This internal bundle is, then, the muscle of the **diapason**, as the external bundle is the muscle of the **intensity** of the sound. This muscle may thus be looked upon as a **tensor** of the cords, the word "**tension**" signifying the state of varying rigidity which results both from the passive distension of the cords and from their active retraction (Bonnier).

The crico-thyroid muscle is the antagonist of the thyro-arytenoid, but it is only so by acting with the muscles which straighten the arytenoids upon the cricoid. The combined action of these muscles does not, properly speaking, produce the tension of the vocal cords, but only their **distension**. When the elevator muscles of the larynx draw the thyroid cartilage upwards and forwards, and with it the anterior insertion of the vocal cords, the latter, in their turn, pull on the arytenoids and the postero-



superior part of the cricoid. The cricoid cartilage would then swing forwards, if the crico-thyroid muscle, taking the thyroid for its fixed point, did not straighten the cricoid by carrying the posterior insertions of the cords backwards. In that case the cords can be stretched. The crico-thyroid muscle is, then, one of the distensors of the cords; its paralysis, without producing complete aphonia, affects the voice and necessitates the assistance of the other distensor muscles.

A fourth group forms the **extrinsic** musculature of the larynx. Some are **elevators** of the larynx (mylo-hyoid, genio-hyoid, digastric, stylo-hyoid), and their action is continued by the thyro-hyoid muscle. The others are **depressors** of the larynx, towards the sternum (sterno-hyoid and sterno-thyroid) and the shoulder-blade (scapulo-hyoid). Lastly, another muscle draws the larynx towards the vertebral column (inferior constrictor of the pharynx); it is a **retractor** and completes the musculature of the apparatus of phonation.

In fact, all these intrinsic and extrinsic muscles are simultaneously but unequally in play during phonation. In intonation—that is, in the emission of voluntary sound—the thyroid prominence occupies a fixed level between the sternum and the chin. In modulation—that is, when the intonation is varied—the prominence rises for high sounds and descends for low ones; as the neck and the head keep the same attitude, or as the chin remains at the same distance from the sternum, the *pomum Adami* always occupies the same level for the same intonation.

When the head is bent or straightened—that is, when the relations between the chin and sternum vary—the levels occupied by the thyroid prominence vary equally, but a fixed height of the thyroid prominence corresponds for each sound to a fixed attitude of the head. This prominence answers to the anterior insertion of the vocal cords. The function which we have just studied is due to the combined action of all the extrinsic muscles of the larynx.

Phonation is in harmony with the act of expiration. In expiration the trachea, the cricoid, and with it the posterior insertion of the vocal cords, are raised, in proportion as the air escapes and the chest is emptied. It suffices to prolong a sound during the whole period of expiration to verify the ascent of the larynx. If the cricoid be raised, the thyroid must also be raised to maintain the same degree of tension of the cords, and it is only raised by the action of the elevators, which is modified by the depressors and the retractor. All the musculature, therefore, is in play.

When we modulate sounds (high sounds) up the scale, the cords are stretched and the larynx is raised, while the contrary takes place when our intonation becomes lower.

The elevators of the thyroid, drawing the anterior insertion of the cords upwards and forwards, are therefore tensor muscles, just as the muscles which straighten the arytenoids upon the cricoid, and as the crico-thyroideus, which draws the posterior insertion of the cords downwards and backwards (Bonnier).

**Nerves.**—The muscles of the larynx are supplied by the recurrent and the external laryngeal nerves.

The recurrent nerve, formed by the spinal accessory and vagus nerves, supplies all the muscles except the crico-thyroidei; the fibres from the vagus seem more especially reserved for the posterior crico-arytenoid muscles, which open the glottis, while those from the spinal accessory supply the other muscular groups.

The crico-thyroid muscles, or indirect tensors of the vocal cords, are supplied by the external laryngeal branches of the superior laryngeal nerve, which takes origin from the gangliform plexus of the vagus.

The extrinsic musculature is innervated by the motor branch of the trigeminal, by the facial, the glosso-pharyngeal, and especially by the hypoglossal nerve.

Bonnier has recorded seven cases of hysterical aphonia in which the muscles, innervated by the laryngeal nerves, were working perfectly. The want of tension in the cords was due to the inertia of the elevators supplied by the hypoglossal nerve.

The larynx receives its sensory nerves from the vagus by the superior laryngeal nerve, which makes the upper part of the organ exquisitely sensitive, and by the external laryngeal nerve, which gives a more obtuse sensibility to the subglottic portion.

**Respiration—Voice—Effort.**—Respiration goes on freely, thanks to the contraction of the posterior crico-arytenoid muscles, which dilate the glottis and keep it open during respiration. The laryngoscope shows that the glottis forms a triangle, with its base backwards, at the arytenoid cartilages.

The production of the voice is much more complicated. The air in the chest is expelled with a variable force, which is regulated by the contraction of the expiratory muscles on the one hand, and by the muscles of effort on the other, and differs in speaking or in singing.

When the voice is to be produced, there is first accommodation on the part of the glottis—that is, the ary-arytenoid, lateral crico-arytenoid, and external thyro-arytenoid muscles draw the vocal cords together to the proper degree, so that they may vibrate under the pressure of the expired air. At this moment the glottis presents the appearance of a fusiform cleft, which may attain as much as 2 or 3 millimetres in its greatest diameter.

The external muscles fix the thyroid at a given level; the crico-thyroideus carries the cricoid backwards and downwards; the posterior crico-arytenoid muscles, assisted by the ary-arytenoideus, draw the arytenoids backwards; and the cords would be extended, without being really stretched, if the internal bundle of the thyro-arytenoideus did not struggle against the passive distension of the cords by their active retraction. The latter thus acquire a physiological aptitude for vibration, which may be looked upon as a tension peculiar to these organs.

The height of the sound is, then, produced by the tension of the vocal cords. In the chest-voice the sound is produced by the rapid periodic variations of pressure of the air at the glottis: the quicker the vibrations, the higher the sound; the greater the resistance to expiration—that is, the greater the difference of pressure—the more intense the sound.

The sound is not produced by the resonance proper of the vocal cords, but by the variations of tension which their vibrations give to the column of expired air at the level of the glottis.

The mechanism of the production of the head-voice is still imperfectly known.

According to some (Lermoyez), the mucosa of the cord is said to be the only vibratory part; according to others (Bonnier), the larynx is said to act like a whistle of variable shapes, the column of air which comes out of the glottis breaking against the edge of the ventricular bands. The height of the sound may be equally produced by the pressure of the expired air; greater and greater pressure may thus raise the sound by a fourth, and even a fifth, the tension of the vocal cords remaining the same. Furthermore, some authors admit that the tension of the vocal cords may be compensated, in a certain measure, by their approximation—that is to say, almost the same effect is obtained with the vocal cords when little stretched but close together and with the cords more open but very tense. Thanks to these different combinations of length, tension, opening, and pressure, the larynx gives the infinite shades and modulations of the voice in the acts of singing, lecturing, and conversing.

The shape of the glottis, the vibrations of the cords, and the limits of these vibrations, can be verified by means of the laryngoscope. During the emission of a high sound the anterior part of the vocal cords vibrate, and, in proportion as the sound becomes deeper, we see the glottis assume the ellipsoid shape, the vibrations being produced in the posterior parts of the vocal cords, and even in the interarytenoid glottis, which not only takes part in respiration, as was long supposed, but which also contributes to the production of low notes. These few remarks on the production of the human voice will help us to understand how a simple ulceration, the presence of a false membrane,



the **paralysis** of a muscle, the **ankylosis** of an articulation, or **œdema** of the arytenoid, reacts at once on such a delicate organ in different ways.

The **timbre of the voice** varies in each individual, according to conditions which were unknown until the researches of Helmholtz. I will briefly recapitulate them. Every sound is formed of a **fundamental** note, and certain accessory notes, called **harmonies**, which have a fixed relation to the fundamental note. The harmonics are always higher in the scale than the fundamental note. When they do not bear a regular ratio to the fundamental note, the sound is simply a noise. When, on the contrary, they are in regular proportion, a musical note is produced. The **timbre** of the note, then, depends upon the grouping and the number of the harmonics. In the human voice the fundamental notes and the harmonics are produced at the vocal cords, but other harmonics are also produced in the pharynx, the nose, and the mouth. Lesions of the mouth, the nose, and the pharynx, as well as those of the larynx, are therefore able to modify the **timbre** of the voice, which becomes **throaty** or **nasal** in different cases.

The **act of effort** is only possible when the framework of the thorax, on which almost all the muscles of the trunk and some of the muscles of the upper limbs are inserted, is firmly fixed. The thorax then becomes the fixed insertion-point of the muscles which are to be brought into play in the effort. This initial fixation of the thorax is obtained by means of a deep inspiration, but on condition that the firmly closed lips of the glottis oppose the issue of the previously inspired air. In **paralysis** of the constrictor muscles of the glottis the **effort** is impossible.

## II. ACUTE CATARRHAL LARYNGITIS.

**Description.**—**Acute catarrhal laryngitis** may occur as a distinct illness, or be secondary to some other ailment. In both cases it begins with a feeling of tickling in the larynx, and, as the sensibility of the mucosa is increased, the inspired air appears too cold, and its passage into the respiratory channels is painful. The cough, which is at first dry and slight, becomes more severe as mucus accumulates on the lips of the glottis.

Phonation and respiration soon become difficult; the emission of sounds is painful, the high notes are lost, the voice is hoarse, altered in its low timbre, and almost inaudible, because the inflamed and paretic vocal cords are no longer in their normal state of tension and vibration. Trifling laryngitis only provokes hoarseness; when it is intense, when the paralysis of the vocal muscles is very marked, and when the ventricular bands are œdematous and cover the vocal cords, **aphonia** becomes complete. The voice, which is deep and raucous, is sometimes interrupted by sharp sounds, like those of the **falsestto**; this phenomenon can be explained by the vibratory nodes which are formed on the cords, thickened and covered with mucus.

Respiration is free in the adult. In the child, whose glottis is much narrower, dyspnœa is frequent, and often complicated by suffocative attacks, which result from spasm of the glottis. The expectoration, insignificant at first, is composed later of thick greyish sputum, which is less the result of the laryngeal inflammation than of the bronchitis and the tracheitis which are so often present.

With the laryngoscope we see that the mucosa has taken on a dark colour

at several spots; the epiglottis, the aryteno-epiglottic folds and the arytenoids are red and swollen; the vocal cords have lost their pearly, shining aspect, and are covered with rosy striations, though they escape the swelling which frequently attacks the ventricular bands. The secretion from the mucous glands is abundant; it sometimes has a gummy aspect and coats the different regions. In the benign form the fever is insignificant. The illness does not last more than a week or a fortnight, and the different symptoms show rapid improvement, with the exception of the vocal troubles, which **are much slower** in their disappearance.

We also see acute laryngo-tracheitis **a frigore**, which is more **intense**: the fever is sharp; coughing and swallowing cause much pain; the sputum is sometimes streaked with blood; vocal troubles are very marked; and the breathing is as much hampered as it is at the onset of œdema of the glottis, a fact which is explained by the swelling of the inflamed parts. With the laryngoscope we discover, in addition to the lesions previously described, ecchymoses, which are most marked on the anterior part and the free edge of the vocal cords, and which testify to the severity of the inflammation.

**Ætiology—Diagnosis.**—Acute laryngitis is provoked by the direct contact of cold air with the larynx, and by its indirect action upon some other part of the body (cold to the feet). It is set up by irritant vapours, accompanies tracheitis, bronchitis, or coryza, and is one of the chief symptoms of influenza. In measles it assumes a special type.

The severe form may simulate **œdema of the glottis**; the slight form, which is much more frequent, must not be confounded with **nervous aphonia**. Every individual who is seized with dysphonia or hoarseness, following a chill, has not perforce laryngitis; he may have vocal paralysis (**nervous aphonia**) (Krishaber). In this case the laryngoscope reveals no trace of phlegmasia, and the vocal cords have kept their whiteness, but are incompletely stretched, and the paralysis, which is usually unilateral, causes dysphonia. These vocal troubles are due to paralysis of the external laryngeal nerve, which supplies the crico-thyroid muscle, by which the vocal cords are made tense.\*

Laryngitis, especially in children (laryngeal cough and hoarseness), is often the chief symptom of the invasion of measles; the presence of pharyngeal, ocular, and nasal catarrhs will settle the diagnosis.

It must not be forgotten that laryngeal troubles of **syphilitic** origin, such as hoarseness and loss of voice, due to erythema and erosive syphilides

\* This paralysis of the external laryngeal nerve is not more astonishing than that of the facial or radial nerves from the same cause. It is also curious to see that the same agent—cold—destroys the function of the motor nerves by causing paralysis, and exaggerates that of the sensory nerves by causing neuralgia ("Aphonie Nerveuse," *Thèse de Paris*, 1865).



of the larynx, have the closest resemblance to simple laryngitis. This fact has an important bearing upon **treatment**.

**Prognosis—Treatment.**—Acute catarrhal laryngitis, which is not formidable in the adult, is more serious in children, because it produces suffocative attacks. It is subject to relapses, and is a serious misfortune in people whose larynx is a “professional instrument” (Peter and Krishaber), as in singers, barristers, or public speakers, who are sometimes obliged to abandon their profession because the voice is slow to regain its normal character, and is lost afresh under the influence of similar causes.

Sweating, hot drinks, soothing gargles, powders, inhalations, blisters placed on the front of the neck, topical applications to the larynx, and, lastly, local blood-letting when the inflammation is acute, form the general **treatment**.

### III. CHRONIC LARYNGITIS.

I shall describe three varieties of chronic laryngitis—catarrhal, glandular, and hypertrophic. I would, however, remark that these varieties are not always distinct clinically; in fact, they are **often associated**.

**Chronic Catarrhal Laryngitis.**—This variety is hardly ever primary; it usually succeeds one or several acute attacks, and, like every laryngitis, is kept up by the efforts of singing, by excess of tobacco, and of drink. Pain is absent, cough is moderate, and hoarseness is almost uniform, but rarely goes as far as aphonia. The laryngoscope shows redness and swelling of the mucosa, with varicose vessels and glandular projections. The inflammation is sometimes localized to certain spots, which are, in order of frequency, the posterior surface of the epiglottis, the aryteno-epiglottic ligaments, the ventricular bands, and the vocal cords.

**Gouty Laryngitis.**—Gouty persons often suffer a form of chronic laryngitis with exacerbations, which are accompanied by rhinitis, tracheitis, and bronchitis. The bronchitis is at times limited to the bases of the lungs; if it occupy the apices, the affection may be considered of a tubercular nature. This laryngitis is chiefly seen in singers during the first years of study, and is more frequent in summer than in winter. Most often it disappears spontaneously, when other manifestations of the diathesis appear, unless vocal strain has provoked definite hypertrophy of the cords (Bonnier).

**Glandular Laryngitis.**—**Glandular or granular laryngitis** is generally chronic from the outset, and does not, like the preceding form, follow attacks of acute laryngitis. It is often associated with granular pharyngitis, which opens the scene in many cases; the condition might, therefore, be termed “granular pharyngo-laryngitis.” Herpetic and arthritic subjects are predisposed to it, but abuse of drink or tobacco, and especially the immoderate use of the voice, are its chief causes. The inflammation is limited, as a

rule, to the clusters of glands on the posterior surface of the epiglottis, in front of the arytenoid cartilages, where the glands form a vertical ridge, and to those on the ventricular bands and on the vocal cords, where their mission is to moisten the papillary region of the vocal cord, and thus prevent loss of function. The inflammation attacks the glands of the arytenoids, of the base of the epiglottis, of the vestibule of the larynx, and of the vocal cords. The hypertrophy of the glands, together with the increased **vascularity** of the mucosa, alters the voice; the singer can no longer sound the low notes, and soon loses the clear tone of the **high notes**. The loss of the high notes, which is one of the first symptoms of glandular laryngitis, is easily explained.

In the normal state the high notes are produced as follows: The anterior processes of the arytenoid cartilages, by their approximation, bring the vocal cords into exact contact, and the lips of the glottis vibrate, especially in their anterior third, when a sound is emitted. The complete approximation of the vocal cords can no longer take place if the interarytenoid mucosa be swollen and thickened. The high sounds, therefore, are defective or suppressed. Later the patient loses the low notes, and the middle notes are also affected if the lesion reach the vocal cords.

When the lesions are general, the laryngoscope shows the granular condition and the vascularity of the ventricular bands and of the vocal cords, in addition to the glandular hypertrophy described above. Erosions are sometimes seen at different points of the mucosa.

**Hypertrophic Laryngitis.**—This form is usually associated with the preceding variety. The hypertrophy may be general or local, and, in the latter event, involves the epiglottis, the aryteno-epiglottic folds, and the vocal cords. Türk has described a variety of hypertrophy attacking the vocal cord, **chorditis tuberosa**. The hypertrophied parts are rigid, often deformed; the epiglottis bends backwards, and partly hides the entrance of the larynx; the aryteno-epiglottic folds are thickened and shortened; the arytenoids resemble an irregularly shaped nipple; and the vocal cords are much enlarged.

Besides the various changes in the voice, hypertrophic laryngitis is sometimes accompanied by **dyspnœa**, due to the swelling of the affected parts, which may obliterate the orifice of the larynx and gradually lead to asphyxia.

In chronic laryngitis both local and general **treatment** are employed. The direct application of a 10 per cent., or even 20 per cent., solution of nitrate of silver, insufflations of bismuth, inhalations of iodine and sulphur vapour, and preparations of arsenic internally, are the therapeutic agents most employed. Good results are obtained from the cures of Eaux-Bonnes, Cauterets, and La Bourboule. Krishaber has successfully applied igni-puncture to the granulations of the larynx.



We must also prove that the nasal fossæ are clear, and must restore nasal respiration. Gouty laryngitis often yields to hot local applications to the neck and to nasal douches of hot saline solutions. It is ameliorated by very warm gargles. The best of these gargles can be made with wine, adding infusion of cinnamon, which makes the gargle astringent; the gargle must be used as hot as the patient can bear it. As a rule, all the symptoms yield very rapidly (Bonnier).

#### IV. SYPHILIS OF THE LARYNX (SECONDARY LARYNGOPATHIES—TERTIARY LARYNGOPATHIES).

The frequency and the importance of syphilitic lesions of the larynx deserve our careful attention. Lewin says: "Next to the skin and the throat, syphilis most frequently attacks the larynx." The epiglottis, the aryteno-epiglottic folds, the ventricular bands, the vocal cords, and the skeleton of the larynx, may show most diverse changes due to syphilis. These changes may be superficial or deep. The superficial lesions, which comprise laryngeal catarrh, erythema, mucous patches, erosions and ulcers, with or without œdema, are seen in the secondary stage.

The deep submucous infiltrations, which comprise diffuse or circumscribed syphilomata, ulceration, hypertrophy, vegetations, changes in the cartilages, formation and elimination of sequestra, cicatricial retractions, progressive laryngo-stenoses, permanent deformities, and perilaryngeal phlegmon, are found in the tertiary period.

##### Secondary Laryngopathies.

**Description.**—The chancre has never been seen in the larynx. The secondary lesions—erythema, catarrh, erosion, and ulceration—with or without œdema, first demand notice.

**Erythema** appears a few weeks after infection, at the same date as the mucous or cutaneous syphilides, and is a very frequent trouble. Many people become hoarse within some weeks or months after infection, and ascribe the trouble to a chill, or speak of "loss of voice," when the mischief is really syphilitic. With the laryngoscope the erythema is characterized by a rosy, ash-coloured, or reddish tint of the mucosa, and often coincides with erythema of the throat, which of is a vermilion colour, especially on the velum and the anterior pillars. The voice becomes hoarse, and may be almost lost. The erythema disappears in a few months, but is prone to relapse; slaty coloration of the vocal cords sometimes remains. •In some cases erythema is accompanied by secretion, and laryngeal **catarrh** is found.

Secondary **syphilides** of the larynx may be erosive or ulcerative. The

erosions are superficial ulcerations, which are opaline, rounded, or oval, and surrounded by a red border ; on the vocal cords they are usually elongated and situated on the free edge. Erosive syphilides sometimes coincide with erythema and provoke vocal troubles, but are not, as a rule, accompanied by œdema or dyspnœa. Swallowing is painful when the aryteno-epiglottic folds are involved. These erosive syphilides form part of the early secondary troubles, whilst the ulcerative syphilides appear later. The latter (and I do not allude to the deep ulcerations of the tertiary period) are much broader and more hollow than the erosive syphilides, their edges are sometimes prominent and of a vivid red, while the surrounding mucosa is œdematous ; they occupy the epiglottis, the arytenoid region, the ventricular bands, and the vocal cords.

It must not be thought that the superficial lesions of the larynx belong solely to the first year after infection ; catarrh, erythema, erosions, and superficial ulcerations, with or without œdema, although they be slight and part of the "secondary troubles," may still appear many years after the primary infection. Most of these lesions, which may be called "benign," only cause vocal troubles.

Pain is absent, and cough, so frequent in other forms of laryngitis, is often wanting in these cases. The voice is, however, **altered** and **roughened** ; this is the most constant symptom. Hoarseness, dysphonia, and sometimes aphonia, may supervene slowly or rapidly, and may be very obstinate. Persons suffering from syphilis of the larynx are certainly more sensitive to the action of cold, and chills are no doubt to blame for first attacks, and also relapses, of these secondary troubles. This knowledge will prevent us considering a laryngitis in which syphilis plays the chief part as a simple laryngitis a frigore.

**Dyspnœa** is rare in these cases ; nevertheless, superficial and **apparently benign** lesions are sometimes accompanied by **œdema** of the larynx and **respiratory troubles**. This fact is very important ; Krishaber lays great stress upon it. Although dyspnœa may be exceptional in secondary laryngopathies, the oppression may rapidly increase, and tracheotomy would be necessary if the dyspnœa did not speedily yield to treatment. I have several times proved this statement, and M. Mauriac, like Krishaber, affirms that "almost insignificant erosions may become a dangerous inflammatory centre, around which œdema of the glottis suddenly develops."

In dealing with a laryngitis apparently due to chill, or with an apparently simple loss of voice, syphilis must always be thought of, and due investigations made, to avoid an error in diagnosis. The discovery of the chancre and its satellite glands, the presence of syphilides of the skin (roseola), and of the mucosæ (mucous patches), headache, and alopecia, will all help to settle the pathogenic diagnosis.



### Tertiary Laryngopathies.

**Description.**—The tertiary lesions are much rarer, but much graver, than the secondary ones. Though they do not appear, as a rule, within two years from the date of infection, they may be very early (during the first year) or very late (after the twentieth year). The tertiary laryngopathies coincide fairly often with specific lesions of the trachea and the lungs (“they are, as it were, the root”), while the secondary laryngopathies are especially associated with specific lesions of the velum palati and of the throat.

1. **Ulcerative Syphiloma—Gummata.**—The gumma may be superficial or deep, and may attack the soft parts or the skeleton of the larynx. In some cases it forms a small swelling, and is the gumma, properly speaking; in others it infiltrates the tissues, and is the diffuse syphiloma (syphiloma en nappe).

The **Gumma** is the most typical lesion of tertiary laryngeal syphilis. It may vary in size from a pin’s head to a hazel-nut. Several isolated or confluent gummata in different stages of growth may be met with. As a rule, the gumma, seen with the laryngoscope, forms a rounded projection, which is of sombre hue and surrounded by reddish œdema. As softening takes place, it becomes yellowish at the centre, and in a few days an **ulcer** is formed, which shows little tendency to spontaneous healing. The edges of the ulcer are perpendicular, the floor is greyish, the surrounding tissues are hard and elastic. The area on which the ulcer is situated is œdematous.

The gummatous neoplasm is not always circumscribed; it may infiltrate the mucosa and submucous tissue in a diffuse manner, and is then termed “**syphiloma en nappe**.” This syphiloma is often capricious, like the **phagedænic** lesions, and produces serpiginous ulcers, which not only destroy the mucosa, but also attack the perichondrium, cartilages, and joints; they may even reach the extralaryngeal tissues, producing inflammation of the neck (Mauriac). This diffuse syphiloma commences in the submucosa; it may, however, start in the skeleton of the larynx.

We see, lastly, other ulcerations which do not result from the necrobiosis of a gumma. In addition to the gummatous ulcers which I have just described, we may see tertiary ulcers, which at first look like a simple congestion of the mucosa or superficial erosions. We must not, however, trust to the “benign appearance,” for this lesion, though apparently congestive, may be the prelude of severe phagedæna.

The gummatous and ulcerative lesions which I have just described present the most varied pictures with the laryngoscope. The epiglottis, which is so frequently attacked by syphilis, is thickened, hyperplastic, œdematous, and deformed; it forms a dull red tumour, which obliterates the

vestibule of the larynx to a variable extent. In other cases the epiglottis is indented, loopholed, or covered by ulcerations "from the slight erosion of its surface and of its edges to the serpiginous and gangrenous ulcers which reduce it to shreds"; it is sometimes converted into an irregular stump. Phagedæna of the epiglottis is also frequent.

The arytenoid region and the aryteno-epiglottic folds are deformed, œdematous, and hypertrophied by the gummatous tissue; they block up the vestibule and hide the vocal cords. Vegetations are not rare. The folds are the seat of ulcerations with greyish floor and dark perpendicular edges.

The vocal cords undergo the most varied changes: they are red, hypertrophied, and ulcerated. The ulceration begins in the free border, eats into the cord, making it look like a saw; the cord may be reduced to shreds, or the ulcer may end in vicious scars and adhesions.

**2. Non-Ulcerating Syphiloma.**—The diffuse infiltration does not always go on to ulceration; it may run a very slow course, and may cause in the larynx (as in the lips and the prepuce) fibrous thickening which has no tendency to ulcerate. This thickening, which is rarely general, narrows the cavity of the larynx; it affects the subglottic part of the organ, the epiglottis, the ventricular bands, and may be confined to one of these parts. The laryngoscope shows the affected part to be deformed and of a dull red; its surface is granular, mammillated, and sometimes covered by vegetations. In an old syphilitic patient whom I have just seen hypertrophy of the left ventricular band and two vegetations in the arytenoid region were present.

**3. Vegetations.**—The neoplasm often takes the form of a **vegetation**. The vegetations are rarely solitary; they may be isolated or confluent, sessile or pedunculated, and may vary in size from a millet-seed to a pea. Their "seat of election" is the vocal cords and the ventricular bands. They develop on the surface, on the edges, and at the angle of the junction, but rarely invade the epiglottis, in distinction to ulcerations which have a marked preference for it. These vegetations, as a rule, develop in mucous membrane which is already diseased, and may, by reason of their number or of their size, narrow or block the glottis and the cavity of the larynx.

**4. Lesions of the Skeleton.**—The syphiloma may primarily attack the cartilages, or only invade them after the soft parts. The affected cartilages are, in order of frequency, the arytenoids and the cricoid. The thyroid is usually respected. The first result is calcification of the cartilage; the calcified tissue then necroses, and the separation of the sequestrum is accompanied by suppuration, fistulæ, and œdema. The necrobiotic process, which begins on the inner wall of the cartilage, may invade its whole thickness, and, after eating through it, set up inflammation in the front of the neck.



Dislocations and ankyloses of the joints result from the syphilomatous process. In some cases a fragment of cartilage, or an entire arytenoid, may be cast off through a fistula, be rejected by the mouth, or fall into the air-passages and provoke asphyxia.

The changes in the cartilages of Wrisberg and of Santorini are perfectly visible with the laryngoscope. Crico-arytenoid ankylosis is almost always unilateral, and fixes the vocal cord on the affected side.

5. **Œdema.**—Œdema of variable colour often accompanies tertiary laryngopathies. Laryngeal œdema **plays a considerable part** in these cases; it may be more or less extensive, and invades, in order of frequency, the epiglottis, the ventricular bands, the aryteno-epiglottic folds, the vocal cords, the parts below the cords, and the trachea. The symptoms present are stridor, sucking-in, and dyspnœa which borders on asphyxia. The laryngoscope reveals the deformity and the swelling of the œdematous parts; the epiglottis “is rolled up like a paper-twist, and swollen so as to resemble a chestnut or the cervix uteri,” while the arytenoids form large pads, which obliterate the supraglottic region.

Syphilitic œdema of the larynx will be studied in detail in Section 8.

6. **Tracheo-Laryngeal Adenopathy.**—The numerous glands which are present in the trachea and the larynx are sometimes attacked by syphilis, and we therefore find the symptoms which accompany lesions of the recurrent nerves (spasm of the glottis, or paralysis of the vocal cords).

7. **Cicatrices—Laryngo-Stenosis.**—Ulcerous lesions of the larynx sometimes leave deformities, adhesions, and retractions, which affect both the voice and the breathing. The epiglottis may be displaced or deformed by the cicatricial bands, which draw it towards the aryteno-epiglottic folds or towards the lateral wall of the pharynx; the result is complete deformity of the aperture of the larynx. The vocal cords may be converted into fibrous bands, which dislocate the vocal process; the axis and the calibre of the laryngeal cavity are altered, and gradual laryngo-stenosis results. In some cases the stenosis arises from the welding of the ulcerated vocal cords, which then form a kind of diaphragm, and partially obliterate the glottis.

8. **Paralysis.**—The existence of syphilitic **paralysis** of the laryngeal muscles can only be recognized with the laryngoscope. The paralysis is often unilateral, and chiefly affects the left cord. Paralysis of the posterior crico-arytenoid muscles is characterized by immobility of the vocal cords at the moment of inspiration; the glottis, instead of being open during inspiration, tends to close, and asphyxia becomes imminent. When one lateral crico-arytenoid muscle is paralyzed, the cord on the corresponding side does not approach the other cord during phonation. Syphilitic paralysis of the ary-arytenoideus is very rare; the laryngoscope shows that the intercartilaginous glottis remains open during the emission of sounds.

Laryngoplegias are very often isolated. Their pathology is still imperfectly known : they may be due either to lesions of the glands adjoining the recurrent nerves, or to an intracranial lesion, though it is far more likely that these partial paralyses have a peripheral origin. They may be compared with the paralysis of the oculo-motor nerves and with the various forms of facial paralysis which are seen in all stages of syphilis.

**Symptoms.**—**Vocal** troubles are often the first to appear, and may vary from hoarseness to complete and persistent aphonia. **Cough** is rare and not important. **Respiratory** troubles, from shortness of breath to the most violent dyspnœa, are practically constant. Many patients suffer from breathlessness, to which they become accustomed by avoiding violent efforts. In some the attacks of dyspnœa are very severe ; in others the dyspnœa may improve for a while, only to return in a worse form ; lastly, in some cases, especially when œdema is present, the dyspnœa is so rapid in its onset that asphyxia becomes imminent, and intervention is imperative.

**Stridor** and **sucking-in** accompany almost every case of severe laryngeal dyspnœa.

This dyspnœa is due to the stenosis of the larynx ; the narrowing may be rapid (acute laryngo-stenosis) or slow (progressive laryngo-stenosis). Œdema and abscesses are the commonest causes of **acute laryngo-stenosis**.

**Progressive laryngo-stenosis** is due to many causes, such as development of gummata, diffuse hypertrophic syphiloma, presence of vegetations, œdema, abscesses, cicatrices, deformity of the aryteno-epiglottic region, dislocations of the arytenoids, and adhesions of the vocal cords. This enumeration explains sufficiently the diversity, the continuous or intermittent character, and the intensity of the respiratory troubles.

I may remind the reader that asphyxia may follow the fall of a necrosed cartilage into the trachea.

Swallowing is very painful, or almost impossible, in some patients, especially when the edges of the epiglottis and the arytenoids are inflamed and ulcerated. Sharp pains in the ear may accompany the dysphagia. Fœtor of the breath is not so common as in cancer.

**Diagnosis.**—There is often difficulty in diagnosis, because so many lesions of the larynx (hypertrophic laryngitis, syphilitic, tubercular, and cancerous disease, and œdema in Bright's disease) may cause the same vocal and respiratory troubles. Another difficulty in diagnosis results from the fact that such lesions as hypertrophic infiltrations, polypoid excrescences, ulcerations, and œdema may show an identical appearance with the laryngoscope. The signs and symptoms which may help us in diagnosis demand mention.

1. **Chronic Hypertrophic Laryngitis.**—General health excellent, no loss of flesh, appetite good, dyspnœa absent or trifling, obstinate cough



and dryness of the throat ; for a long time past gradual change of the voice, loss of high sounds, hoarseness, and muffling of the voice.

With the laryngoscope : Hypertrophy and granulations on the ventricular bands ; redness and granulations on the vocal cords, especially on their free border ; hypertrophy of the glands of the larynx, chiefly in the arytenoid region. Result : incomplete approach of the arytenoids ; insignificant erosions of the mucosa ; pharyngo-laryngeal catarrh, with abundant secretion.

**Gouty** laryngitis presents during each acute attack marked erythema of the larynx, the pharynx, and the trachea. The periodicity of the crises, however, eliminates syphilitic erythema. The vocal cords have sometimes a blood-red appearance.

**2. Tubercular Laryngopathy.**—Wasting, loss of appetite, bronchitic cough, hæmoptysis, or previous pleurisy, pulmonary lesions in course of development. In some exceptional cases primary laryngeal tuberculosis ; vocal troubles of every degree ; dyspnœa absent or marked, according to the site and the severity of the lesions ; pain and dysphagia, which may be very acute.

With the laryngoscope : **Unusual pallor of the palate and of the vestibule of the larynx** ; isolated or combined lesions of the larynx, which present the following forms :

(a) **Tubercular infiltration, without ulceration**, confined especially to the interarytenoid region, the epiglottic folds, and the ventricular bands ; hypertrophy and deformity of these parts, notably conical swelling of one or both arytenoids ; epiglottis enormous ; great swelling of the ventricular bands ; vocal cords dull and rough ; epiglottis usually healthy ; enlarged glands ; laryngeal catarrh.

(b) **Tubercular vegetations**, which may be large, are found in the interarytenoid region. They are dull, livid, covered with purulent mucus, and often implanted upon an ulcerated base ; surrounding œdema pale.

(c) **Tubercular Ulcerations.**—Their edges are jagged and flat ; they are not deep, and the surrounding œdema is soft and pale. These ulcers in time destroy the ventricular bands, the vocal cords, and the folds of the epiglottis. By curetting the ulcers, liquid which sometimes contains the tubercle bacillus may be obtained.

(d) **Paralysis**, or paresis of a vocal cord, a fairly frequent sign at the commencement of laryngeal tuberculosis (Libermann).

**3. Syphilitic Laryngopathies.**—Health excellent ; no loss of appetite ; no wasting ; cough absent or trifling ; previous syphilis ; vocal troubles, from simple hoarseness to complete and persistent aphonia ; respiratory troubles, varying from shortness of breath to dyspnœa, with stridor and sucking-in ; pain on deglutition and in the ears, according to the lesion.

Examination with the laryngoscope : Pallor of the palate, the pharynx, and the larynx, so common in tuberculosis, is **not seen**. The laryngeal lesions, whether isolated or combined, are :

(a) Rounded, prominent and reddish **gumma**, situated on the epiglottis, the folds, or the ventricular bands, projecting into the larynx and causing deformity.

(b) **Diffuse syphiloma** of the hypertrophic form, situated in different regions, and often coinciding with excrescences and ulcerations.

(c) **Ulcerations**.—Slightly different according as they are consecutive to gumma, diffuse syphiloma, or tertiary non-gummatous ulcerations. The syphilitic ulcer has generally thick perpendicular edges, whereas the edges of the tuberculous ulcer are jagged and flat. The syphilitic ulcer is usually deeper and more excavated than the tubercular one ; the œdematous tissues which surround the syphilitic ulcer are usually red and hard (in tubercular ulceration they are pale and soft). The syphilitic ulceration rapidly hollows out channels and perforates the tissues (tubercular ulceration is **slow** in its progress). Syphilitic ulceration is found in every part of the larynx, but has a marked predilection for the epiglottis, and especially invades its laryngeal surface. Syphilitic ulcers are less numerous than tubercular ones, and leave scars behind them ; tubercular ulcers do not.

(d) **Vegetations**.—They are rarer in syphilis than in tuberculosis. In syphilis vegetations are chiefly found on the base of the epiglottis, on the ventricular bands and the vocal cords (in tuberculosis they are usually found in the interarytenoid region). Syphilitic vegetations show a peculiar tendency to bud out ; they are usually associated with ulceration, tend to disappear and give place to a cicatrix. Tubercular vegetations continue to grow.

If tuberculosis complicate syphilis, the diagnosis is exceedingly difficult. This association is not rare. It is remarkable that tuberculosis, if it be associated with syphilis, is less severe and less rapid than when it is present alone in the larynx.

4. **Cancerous Laryngopathy**.—The diagnosis is extremely difficult, because cancer of the larynx may be almost latent for a year or two ; it does not affect the general health, the appetite remains good, the patient does not waste away, and the glands are not yet infected. Vocal troubles alone are present, but they have no distinctive character. Later, when the cancer has ulcerated, the nature of the lesion, the ready bleeding, the fœtor of the breath, the acute pains in the throat and the ear, and the bloody expectoration, are signs in favour of cancer. But during the first period—and this period may be lengthy—how can a diagnosis be made ? In the first place, if the patient have no syphilitic nor tubercular taint, the question is somewhat simplified. Cancer at the outset is always unilateral, and the lesion is clearly



circumscribed; the "seat of election" is the ventricular band or the vocal cord. When the cancer is polypoid, it is distinguished from other vegetations by the fact that the ulcer deepens in proportion as the tumour grows (Krishaber). The fungating mass of cancer resembles a cauliflower; it is ulcerated and bleeds easily. We find nothing similar in syphilis or tuberculosis.

**5. Polypi of the Larynx.**—The polypoid growths of syphilis must not be confounded with the vegetations of tuberculosis and with papillomata. Tubercular vegetations are colourless, covered with muco-pus, implanted on an ulcerated surface, and surrounded by more or less œdematous mucosa; they are usually situated on the posterior part of the cord, or on the anterior process of the arytenoid. The papilloma may be as large as a millet-seed or a hazel-nut; it may be pedunculated or sessile; it is pearly, wrinkled, and sometimes covered by whitish mucus. Its "seat of election" is the anterior third of the free edge of the vocal cords, because this part is rich in papillæ and glands. These polypi cause vocal and sometimes respiratory troubles.

**6. Œdema of the Larynx.**—Œdema of the larynx, due to many other causes, may simulate that due to syphilis. Dyspnœa, stridor, and sucking-in exist in both cases, and therefore careful examination should always be made as to the cause, in order to institute antisiphilitic treatment without delay if it be required.

**Treatment.**—The recognition of syphilitic changes in the larynx is most important, because, except for certain tertiary lesions and cicatricial stenoses, syphilitic affections of the larynx yield very readily to specific treatment. Local treatment is of only secondary importance; specific treatment is imperative. Inunctions or solutions of mercury and iodide of potassium in large doses give very good results. The vocal and respiratory troubles disappear—sometimes slowly, at other times quickly—and in many instances specific treatment has averted troubles which seemed to demand tracheotomy. We must bear these facts clearly in mind. Krishaber has published some absolutely remarkable cases.

For some years I have exclusively employed oily or aqueous injections of biniodide of mercury, and I find this preparation so superior that I treat syphilis by means of these injections alone, and hardly ever use iodide of potassium. This treatment will be found in the Appendix on Therapeutics.

In some cases, however, this treatment does not at first succeed. **We must persevere**, and severe cases which have resisted treatment for twelve days or a fortnight begin to yield, provided we keep on. I have seen patients in a state of asphyxia from syphilis of the larynx. They had already taken mercury and iodides, but still the lesion had made progress, because the doses were **neither sufficiently increased nor continued for a sufficiently long period**. I have had the satisfaction of seeing these patients recover.

We must not be too ready to perform tracheotomy. Even when asphyxia appears imminent, death rarely occurs from syphilis of the larynx, and, if we push the treatment which I have just indicated, we shall generally succeed in averting the danger without operation.

When tuberculosis and syphilis are present together, it seems, according to the most recent statistics, that it may be advantageous not to treat the syphilis. Tonic treatment must especially be employed. In addition to the fact that mercury and iodides weaken a tubercular patient, tuberculosis progresses in proportion as syphilis improves; indeed, it appears to assume an exceptionally serious character. On the other hand, the two affections appear to neutralize one another to a certain degree—at least, for a time.

### **Hereditary Syphilis of the Larynx.**

The larynx is no more secure from the lesions of **early or late hereditary syphilis** than other organs. “The laryngeal manifestations of hereditary syphilis are much more common in youth than we usually think. Mackenzie has met with a great number of cases, some in the first year (early hereditary), and others up to twelve and fifteen years (late hereditary).” The lesions attack the epiglottis (perichondritis), which is red, thick, ulcerated, and adherent to the neighbouring parts (laryngeal atresia). The arytenoid regions and ventricular bands are infiltrated and covered with vegetations (Moure). The vocal cords are sometimes normal, at other times thickened and ulcerated. Vocal and respiratory troubles are the chief symptoms. The voice is husky or inaudible, and in the child who cannot talk the cry presents the same changes (Sevestre). Respiration is embarrassed from simple breathlessness to the most severe dyspnoea.

I am convinced that many infants with laryngeal troubles which simulate inflammation or œdema of the larynx are really suffering from hereditary syphilis. I saw, with Dr. Bonin, a young infant who had been seized with such severe attacks that tracheotomy was discussed. The attack yielded after some days, thanks to Van Swieten’s liquor. M. Sevestre has reported several cases of the same kind. We cannot pay too much attention to the **pathogenic diagnosis** of laryngeal affections.

## **V. TUBERCULOSIS OF THE LARYNX—LARYNGEAL PHTHISIS.**

**Pathological Anatomy.**—Each organ or each tissue reacts, in its own way, to the invasion of the tubercle bacillus. The large nasal tubercle, which might almost be taken for a sarcoma, and the small tubercular granulations scattered over the intestine or the meninges look quite different, and yet the same micro-organism is the cause in both cases. The larynx, too, has its



own peculiar kind of tuberculosis, and we find three varieties of lesion, either alone or in combination: **infiltration**, **ulceration**, and **vegetations**, are the usual lesions in tuberculosis of the larynx.

In acute cases, and especially in acute **pharyngo-laryngeal** tuberculosis, the miliary granulations are scattered over the surface of the larynx; but in chronic cases, which are much more frequent, the granulations are, so to say, hidden in the base or around the ulcerations. The chief feature in tuberculosis of the larynx is infiltration; ulcerations and vegetations may be present or absent.

**Tuberculous infiltration** may attack all the layers of the larynx. It invades the glands, and develops at the expense of the connective tissue and of the epithelial cells; it surrounds the vessels like a muff; the tunica adventitia is involved, and the lumen of the vessel is obliterated. In the muscles it produces an interstitial myositis. Lastly, it becomes **diffused** throughout the different tissues, and provokes hypertrophic indurations, which, though bordering on fibrosis, only look like oedema (Doléris).

In the cadaver the **tuberculous infiltration** appears as a greyish or yellowish **swelling**, with uniform or uneven surface; it is firm to the touch, and fairly hard on cutting. For a long time this swelling was mistaken for oedema (Gougenheim).

The interarytenoid region, the arytenoids—which stand out like sugar-loaves—the ventricular bands, the aryteno-epiglottic folds, and the epiglottis, take part, separately or simultaneously, in the swelling, which may be considerable. The vocal cords are more rarely swollen; they are reddish, streaked with vessels, and sometimes resemble cushions in appearance.

In some regions of the larynx, as in the vocal cords, the process is characterized by a fibrous or fibro-caseous condition, which closely resembles lupus.

Laryngeal tuberculosis is often **ulcerative**. The ulcers begin in the superficial layer of the mucosa, often in a caseous gland. Small caseous foci are seen on the interarytenoid mucous membrane, the epiglottis, the folds, and the vocal cords. The lesion develops in the corium of the mucosa. First, congestion of the part, and, later, multiplication of the connective cells, are seen. This infiltration, which is primarily submucous, and especially active in the glandular acini, extends and undergoes caseous degeneration, followed by ulceration.

These ulcers often become more extensive by the coalescence of secondary ulcers. The edges are jagged and infiltrated with tubercles; the floor is covered with large polypoid vegetations, especially in ulcers of the interarytenoid region.

The arytenoid region is generally first affected; the ulceration may deepen and attack the crico-arytenoid articulation and the perichondrium. The arytenoid and cricoid cartilages are frequently attacked, while

the thyroid cartilage usually remains free. In some cases the lesion starts in the perichondrium or in the joint, and forms a "tumor albus" containing bacilli. The invasion of the cartilage is preceded by calcareous infiltration and ossification, and is followed by necrosis, expulsion of the sequestrum, suppuration, fistula, and œdema. The sequestrum is reddish, dry, and situated at the bottom of a foul-smelling anfractuous cavity. The elimination of sequestra provokes migratory abscesses, and the pus finds its way into the larynx or the pharynx, or outwards through the skin. Œdema of the larynx, subcutaneous emphysema, and laryngeal fistulæ result from this process.

Tubercular ulcerations also attack the aryteno-epiglottic folds, and may destroy the ventricular bands, obliterating Morgagni's ventricles. The epiglottis is more rarely ulcerated. When it is attacked, the base and the laryngeal surface suffer, whereas syphilis usually affects the lingual surface. The vocal cords are often ulcerated, toothed like a saw, or completely destroyed.

The **vegetations** in laryngeal tuberculosis may show two forms. In the first form the vegetation develops in an ulcer, and forms a bud, which is limited to the ulcerating surface and has a papillomatous appearance. These papillomata, which are chiefly found at the arytenoid region and the posterior part of the vocal cords, may grow to a large size. Sometimes they look like cauliflowers, partially obstruct the orifice of the glottis, and, as they are lacking in firmness, are easily detached. If they should fall into the respiratory passages, they may give rise to the gravest accidents.

In the second form the vegetation does not begin in an ulcer, but develops on its own account, and is chiefly met with in primary tuberculosis of the larynx (Mandl).

In tubercular laryngitis the peritracheal and bronchial glands are often attacked by inflammation, and cause lesions of the recurrent nerves.

**Symptoms.**—Tuberculous infiltration may exist at certain points in the larynx without causing any symptoms. As a rule, the lesion is slow in its progress. At first it invades the arytenoid region, the ventricular bands, and the vocal cords, and for some time the only symptom is hoarseness and muffling of the voice; neither respiratory troubles nor pain are present at this stage.

In some cases, however, even from the first the patient experiences a tickling in the larynx, which is very disagreeable, and causes incessant jerky cough. The sufferer firmly believes that a particle of food has been arrested in the larynx.

In some cases the voice remains almost normal, but the breathing is rather short and quick, and inspiration is more noisy than in the normal state, because the inspired air meets with resistance at the swollen glottis.



If the larynx be examined at this stage, which may be of indefinite duration, we are struck by the unusual **pallor** of the **velum palati**, the pharynx, and the vestibule of the larynx. We find swelling of the interarytenoid tissues, which are specially affected by tubercular lesions; the cartilages of Santorini, the aryteno-epiglottic folds, the ventricular bands, and the epiglottis are all swollen. In some cases the vocal cords are greyish, or rosy and enlarged.

At a more advanced stage these lesions are more marked; the infiltration causes the arytenoids to stand out like sugar-loaves, and the interarytenoid space is covered with vegetations, while the ventricular bands are enormous and cover the subjacent vocal cords, which are no longer visible. The aryteno-epiglottic ligaments, which are fixed and swollen, narrow the aperture of the larynx; the vocal cords are streaked with red, especially on their posterior third, have a skin-like appearance, and show erosions and indentations, while one cord is paretic and does not approximate well. Finally, the larynx as a whole is irregular, funnel-shaped, and covered with thick mucus. At this time the vocal troubles are very marked, yet aphonia may not be complete and dyspnoea may not be severe.

The disease may remain for an indefinite period in the stage of tubercular infiltration without ending in ulceration; if ulceration ensue, dyspnoea and acute pain supervene. The voice is almost **completely lost**; sometimes the aphonia is complete, and it cannot well be otherwise, since the vocal cords are hypertrophied, ulcerated, covered by the enlarged ventricular bands, and fixed by the ankylosis of the arytenoids.

The cough depends rather upon the changes in the lung than upon the lesions of the larynx; it is stifled and **belching** (Trousseau and Belloc), so that the patient, when coughing, appears to be making attempts at belching.\* Acute perichondritis is revealed by sharp pains, with respiratory embarrassment. If an abscess forms, the dyspnoea becomes excessive, and the abscess, which is situated in the arytenoid, opens into the pharynx or the larynx.

Ulcers on the base of the epiglottis and on the aryteno-epiglottic folds are often associated with similar lesions of the base of the tongue and of the pharynx; they render **swallowing** extremely painful. This dysphagia is so painful that in some cases the patient feels as though "a live coal had been swallowed." Not only is the pain made worse by the least attempt at swallowing, but in some cases the patient experiences constant hyperæsthesia of the back of the throat. The saliva, which is abundant, cannot be swallowed, and drips from the mouth; the patient, deprived of rest, sleep,

\* This phenomenon is easily explained. In the physiological condition the sudden opening of the vocal cords by a jerky expiration gives the cough its peculiar tone; but in laryngeal phthisis, as the wide-open glottis no longer offers any resistance, the expired air is belched out.

and nourishment, becomes marasmic and disheartened if relief be not forthcoming.

Aural pain, which may be very intense in all laryngopathies, is sometimes present.

**Dyspnœa**, which is so often seen in advanced tuberculosis of the larynx, is due to infiltration, swelling of the tissues, **œdema** of the larynx, polypoid vegetations, and to perichondritis, with its resultant abscesses. Every variety occurs : it may be slow or sudden in its appearance, and is sometimes accompanied by attacks of suffocation and spasms of the glottis. The breathing, which is harsh or noisy, sometimes takes a **strident** character. Laryngoscopic examination is often difficult by reason of the abnormal positions of the epiglottis, the swelling, the œdema, and the muco-purulent secretion which covers the larynx.

The **course** and **duration** of the malady are very variable. In some cases the march of events is rapid, and life is threatened by an abscess following acute perichondritis, by œdema of the glottis, or by pulmonary phthisis, which complicates the laryngeal mischief and hastens events. The patient becomes marasmic, the wasting is extreme, and hectic fever closes the scene.

**Catarrhal Form.**—I have just described the most common form of laryngeal tuberculosis, which runs a slow course ; in some cases, however, the disease begins abruptly, like simple catarrhal laryngitis, with cough, sudden hoarseness, and aphonia, which may be complete and last for several days. Laryngoscopy reveals nothing but redness, swelling of the mucosa, and more or less abundant secretion. If the patient already have tubercular lesions in the lungs, though he have only slight signs, the laryngitis is open to discussion. Some writers say that it is tubercular, other writers call it “laryngitis in tubercular subjects.” I do not say that tubercular patients cannot have catarrhal laryngitis, but I think that most cases of so-called catarrhal laryngitis in phthisical subjects are tubercular. They are more persistent than cases of simple catarrh, they are subject to relapses, they leave infiltration of the mucosa behind them, and though they are sometimes completely cured, yet they may end in infiltration and ulceration. This laryngitis, therefore, in a patient with hæmoptysis, pleurisy, or tubercular lesions in the lungs, is really tubercular, even though it be apparently benign and catarrhal. Though it may be cured, and may rarely end in the other forms of laryngeal phthisis, I consider it as a manifestation of tuberculosis, just as are those cases of pleurisy or of hæmoptysis which sometimes supervene during excellent health. They are, nevertheless, the first sign of tubercular mischief that will be fully developed some months or years later.

So-called catarrhal laryngitis presents in some cases frankly tubercular lesions, which prove that bacillary infection may occur, not only in the



depth of the tissues, but also in the superficial layers. Heintze, quoted by Héring, saw a trifling infiltration of the ventricular band, and found bacilli in it, between the epithelium, in the glands, and in the cylindrical epithelial cells of the glands. The bacillus, says M. Héring, may be introduced by the glands, and thus provoke an infection of superficial origin.

**Diagnosis.**—Let us first establish the diagnosis at the **start** of the malady. The symptoms of laryngeal tuberculosis in its first period—namely, cough, hoarseness, and dysphonia—are common to every chronic laryngitis. These symptoms, therefore, cannot give sufficient data either for or against tuberculosis.

Laryngeal tuberculosis may begin with paralysis of a vocal cord, the appearance of a papillomatous vegetation, or an attack of catarrh. An affirmative diagnosis, therefore, is difficult at the outset. Certain signs, however, are of very great importance. One of these signs is the **unusual pallor** of the velum palati and of the epiglottis. This pallor is sometimes as marked as in most characteristic anæmia; the mucosa of the palate and the parts of the larynx which are usually pink take on a dull, greyish, and sometimes opaque tint in tuberculosis. This pallor is not seen in non-tubercular laryngitis, and is therefore a valuable sign.

Another sign in laryngeal tuberculosis is the initial localization of the lesions. The interarytenoid and the arytenoid regions are the seat of election in tubercular lesions; swelling, granulations, and vascularization of the above regions, together with the anæmic appearance just described, are strongly in favour of tuberculosis.

At a more advanced stage we must distinguish phthisis from **syphilis** and from **cancer of the larynx**. This question is fully discussed under Syphilis of the Larynx. Syphilis rarely attacks the vocal cords; it seizes the parts which adjoin the pharynx—*i.e.*, the epiglottis and the posterior surface of the arytenoids. Furthermore, syphilitic ulceration is usually limited to one point, and does not invade the rest of the larynx. In tuberculosis the ulcerations are multiple, and accompanied by more or less intense laryngitis. Tubercular ulcerations are frequently covered by polypoid growths, which are much rarer in syphilis. Syphilitic ulcerations are quickly improved by treatment, whilst remedies have practically no effect on tubercular ulcerations. Syphilis, like phthisis, may attack the cartilages, but in eighteen cases out of twenty necrosis of the larynx is of tubercular origin. By scraping the ulcer we can remove some particles of secretion and find the **tubercle bacillus**.

**Cancer of the larynx**, and especially epithelioma, presents the peculiarity of being almost indolent, and of having a very much **slower** course in the larynx than in other organs; indeed, a patient with cancer of the larynx may live for two or three years after tracheotomy (Krishaber). Vocal

troubles are for a long while the only symptom, and when other local and general symptoms supervene, such as hæmorrhage, fœtor of the breath, dysphagia, pain, dyspnœa, etc., the laryngoscope generally reveals the cancerous vegetations.

**Ætiology.**—According to Heintze, the larynx of adults who are suffering from pulmonary tuberculosis is affected in 50 per cent. of the cases. In some cases the laryngeal trouble is **primary**, and appears as the first manifestation of tubercular infection. As a rule, laryngeal tuberculosis is secondary to pulmonary disease, and the infection of the larynx takes place either by the deep path of the vessels and lymphatics or by the superficial path along the surface of the mucosa, or through the excretory ducts of the glands. Louis supposed that the constant passage of infected sputum from the lungs infected the larynx. As for the other causes, they closely follow the ætiology of pulmonary phthisis. Males appear more predisposed than females, and the malady occurs in the former between twenty-five and forty years of age.

In the **child** laryngeal tuberculosis deserves special mention: the younger the child the rarer the disease. Parrot found tubercular laryngitis in only 3 per cent. of autopsies on infants under two years. In the infant pulmonary tuberculosis is usually of the miliary form; the child does not expectorate, and its larynx is not bathed in pus swarming with bacilli; this factor, no doubt, helps to explain the rarity of tubercular laryngitis in children.

In my experiments with Krishaber we studied the results of inoculation and contagion in the ape.\* We were struck by the fact that animals which had been inoculated and had died from tuberculosis showed no tubercular changes in the larynx.

**Treatment.**—Laryngeal tuberculosis is not incurable. It sometimes remains stationary, and several cases (Héring) prove that the tubercular ulcers may be cured. Every effort must be made to relieve the dysphagia and the hyperæsthesia of the back of the throat, which cause such torture to the patient.

For this purpose hypodermic injections of morphia should be given morning and evening. At meals the painful parts should be touched with a brush or a sponge, mounted on a curved handle and soaked in the following solution:

|                       |    |    |           |
|-----------------------|----|----|-----------|
| Cocaine hydrochlorate | .. | .. | 1 part.   |
| Water                 | .. | .. | 50 parts. |

With a little practice, the patient himself can paint the painful parts with the solution, but he must avoid swallowing it. Inhalations and sprays of

\* Dieulafoy and Krishaber, *Arch. de Physiologie*, March, 1881, No. 3.



water containing a little sulphur (Allevard) may render some service. Painting the ulcers with a 10 per cent. or 20 per cent. solution of lactic acid, and later with the pure acid, sometimes yields good results. Ruault employs phenol and sulphur in castor oil.

The cough may be relieved by careful intratracheal injections of menthol in oily solution, but this method demands much judgment. The gargles of very hot and astringent wine which Bonnier employs for gouty laryngitis are also of service, by soothing the cough and the pharyngo-laryngeal irritation.

Bonnier prefers insufflations which the patient can use without the physician's aid, such as iodol, 1 part; sodium benzoate, 6 parts; tolu, 2 parts; tannin, 1 part; gum arabic, 3 parts; cinnamon, 0.20 part, to sprays of menthol or eucalyptus.

Curettage of the ulcers and cauterization has rendered good service in experienced hands.

The "altitude cure" is not contra-indicated in cases of tubercular laryngitis. I have seen the larynx, as well as the lung, improve by the cure at Davos-Platz.

## VI. LARYNGEAL DIPHTHERIA--CROUP.\*

**Definition.**—Croup, or pseudo-membranous laryngitis, is characterized by the presence of membranes in the larynx and trachea. These membranes may exceptionally be due to a non-diphtheritic lesion, the diphtheria bacillus taking no part in the process. Clinical medicine had created a simple croup, said to be neither contagious nor infectious, and, indeed, clinical medicine was right. Bacteriology has shown that false membranes in the larynx and pharynx may be produced by microbes which have nothing in common with the diphtheria bacillus. A small diplococcus may give birth to membranes in the larynx or the pharynx, although it has none of the toxic properties of the diphtheria bacillus. We shall return to this point later.

We find, then, diphtheritic croup, which is the rule, and non-diphtheritic croup, which is the exception.

**History.**†—In 1765 Home, a Scotch physician, published an interesting monograph on croup. He was the first to indicate clearly the characteristics of this malady, and to separate it from certain affections of the pharynx with which it had been previously confounded; but he was doubly wrong in mistaking the identical nature of diphtheritic angina and of croup, which other observers had previously established, and in giving a single description for two distinct maladies—*i.e.*, false croup and true croup. Bretonneau,

\* To avoid repetitions, see articles on Diphtheria and Diphtheritic Angina.

† To complete the history, refer to the chapter on Diphtheria.

in his memorable work on **diphtheritis**, re-established the identity which Home had failed to recognize. He applied the term "**laryngite striduleuse**" to a malady which simulates croup, but which has nothing in common with it. We know how brilliantly Trousseau completed the teachings of his master, Bretonneau, on diphtheria and croup, and with what success he made the operation of tracheotomy popular.

The bacteriological researches of recent years have given valuable precision to the diagnosis and the prognosis of croup, while **serotherapy** has completely modified the treatment.

**Division—Ætiology.**—Croup is **primary** when the diphtheria which engenders it attacks the patient while in good health. It is **secondary** when the diphtheria supervenes as a complication in the course of some other malady, such as measles, scarlatina, whooping-cough, or typhoid fever.

Croup usually follows diphtheria of the pharynx, and statistics prove that the larynx is chiefly invaded from the second to the fifth day of the angina. Croup sometimes follows **diphtheritic coryza**—that is to say, the diphtheria begins in the nose and spreads to the larynx. Cases of croup secondary to diphtheria of the bronchi have been seen, and have been called "ascending" croup. Diphtheria may also invade the larynx before other parts, and the disease is then called **primary**. This form, however, is very rare, and, in order to vouch for this fact, we must be certain that the throat and the nasal fossæ are normal.

In some cases croup appears though false membranes in the throat are absent, and yet cultures made from the mucus on the tonsils may show the diphtheria bacillus. We see, therefore, how rare **primary** croup must be.

The chief causes of croup are epidemicity and contagion. **Contagion** is only too well proved by the numerous examples of physicians who have contracted the disease from their patients. In certain parts—Paris, for example—croup is **endemic**. **Epidemic** croup sometimes rages with terrible severity, as witness the epidemics which ravaged Europe in the sixteenth and seventeenth centuries—the **enfermedad del garrotillo** in Spain, the **morbis strangulatorius** in Italy. Croup spares no age, though it chiefly attacks infants between the age of two and seven years.

**Symptoms.**—The present description refers to diphtheritic croup in children. Whether croup at once invade the larynx, or whether it be preceded by diphtheritic tonsillitis or coryza, the growth of membrane in the larynx and the trachea is immediately announced by vocal and respiratory trouble. The **false membrane** may be said to **sum up** almost the whole history of croup, for its presence on the vocal cords and in the larynx alters the normal sounds, narrows the glottis, and hinders or prevents the entry of air into the lungs.



The pathological rôle of the false membrane, however, is essentially a mechanical one. In some cases this mechanical rôle is of secondary importance, and the gravity of the illness does not arise from the obstruction of the larynx, but comes from the preceding angina, from the poisoning of the economy by the diphtheritic toxine, from the concomitant bronchitis and broncho-pneumonia, from the addition of secondary infections, from the association of the streptococcus with the diphtheria bacillus, etc.

**Cough** is the earliest sign of croup. Slight and trifling at first, it returns in very short fits; during the next few days it acquires a dull and muffled quality, and becomes as inaudible as the voice. As the disease progresses the cough becomes less frequent, and the fits only recur every quarter or half hour, and even at longer intervals (Trousseau). The **voice** is at first hoarse, but after some days it becomes inaudible, and the aphonia is complete. *Vox nihil significat*, said Aretæus.

In the child the respiratory troubles are early and marked, because the larynx is narrower than in the adult. The dyspnœa, which is at first slight, begins at night, and is ushered in by slight whistling on inspiration; it increases as the aperture of the glottis becomes narrowed by the false membrane, which forms chiefly on the aryteno-epiglottic ligaments and the vocal cords, and as the air meets an obstacle, the inspiration is changed into a shrill and prolonged whistling. At the same time depression of the epigastric hollow and of the suprasternal fossæ occurs. This "sucking-in" is due to the tendency each inspiration has to produce a vacuum in the chest, and to the compensatory ascent of the diaphragm.

As the dyspnœa grows worse, the breathing becomes sawing, like the noise which a saw makes when it cuts stone (Trousseau). Every two or three hours at first, then every hour, and at yet more frequent intervals, we observe terrible attacks of suffocation; they result from spasms of the glottis, and resemble those seen in œdematous or spasmodic laryngitis. This struggle may last several days, and, if the illness do not take a favourable turn, asphyxia ends the scene. "The bloated and cyanosed face, the hollow and shining eyes, express the most painful anxiety, and at length the death struggle begins, without there being from this time forward any attacks of suffocation, such as those which have already occurred might lead one to expect" (Trousseau).

**Auscultation** of the chest, when there is no pulmonary complication, reveals nothing but the echo of the laryngeal whistling. The respiratory rhythm is altered, and expiration becomes longer than inspiration, from the difficulty which the expiratory muscles experience in driving the air through the narrowed glottis.

The **expectoration** is often characteristic, and about the third or the fourth day the patient coughs up shreds of membrane—flat if they come from

the larynx, tubular and branching if the bronchi be invaded. False membranes are coughed up in half the cases.

The **fever** in croup is not, as a rule, high, and the temperature varies between  $101^{\circ}$  and  $103^{\circ}$  F. **Albuminuria** is frequent (Sée, Barbosa), and is due to nephritis, caused by the diphtheritic toxins.

Multiple **eruptions** have often been noted in croup, as in diphtheritic angina (Sée); they show different forms and simulate the exanthems of measles and of scarlet fever.

In **adults** the symptoms of croup present some differences, because of the shape and size of the larynx. The modifications of the cough and voice are the same, but dyspnoea and asphyxia supervene slowly, and are not, as a rule, accompanied by stridor and attacks of suffocation.

**Course—Duration—Prognosis.**—The course of croup may be divided into two periods—the first of dyspnoea, the second of asphyxia—while the two together may last from three days to a fortnight; but many exceptions, especially during an epidemic, are seen in the course and the succession of symptoms, and the disease may be fatal on the third or even on the second day. Trousseau's lectures on diphtheria and croup will suffice to warn us against the surprises of this disease, and to show us how suddenly death may appear. These fulminant forms are chiefly seen in adults, and are due rather to the virulence of the infection than to the lesions in the larynx. A terrible attack of suffocation may occur, and be followed by continuous dyspnoea, or dyspnoea may come on without attacks of suffocation, and the patient succumb to the infection (malignant diphtheria).

**Abortive** croup has been described. I have seen such an example in a little boy who, some days before, had been attacked by nasal diphtheria. In abortive croup the false membrane remains limited to the vestibule of the larynx, or, at least, the cords are scarcely affected. The vocal troubles are absent or insignificant, and the respiratory symptoms are not severe.

The **progress** of croup varies a little, according to the virulence of the infectious element. In general terms, when we see a relative calm between the attacks of suffocation, it means that the child is not yet threatened by the infectious element. If the little patient, however, be infected by the disease (malignant diphtheria), the truce is not complete: no lull is seen between the attacks, or after the expulsion of false membranes; permanent dyspnoea, prostration, thready pulse, and marked albuminuria bear witness to the virulence of the infection and the gravity of the prognosis.

Cases of **prolonged** croup must be recognized. M. Cadet de Gassicourt has cited cases of croup which lasted fifteen, twenty, or twenty-five days, and recovered; there was no asphyxial stage, and tracheotomy was not performed.

During the course of the disease remissions are sometimes seen: the



voice regains its tone and the breathing its freedom; but this improvement, which is due to expulsion of membrane, is too often transient, and must not be mistaken for recovery. Croup is an extremely serious malady, which formerly, even with surgical intervention, often ended in death. Injections of serum have recently improved the prognosis to a striking extent.

**Complications.**—**Diphtheria of the bronchi** may precede or follow the laryngeal disease. In every case it increases the danger, because it adds a fresh obstacle to that already present in the larynx. The patient brings up membranes, which may be tubular, rolled up, or like coagulated mucus.

**Broncho-pneumonia** is a still worse complication, which appears at all periods, both before and after tracheotomy, but, as a rule, from the third to the sixth day of the disease. It is sometimes accompanied by gangrene of the lung. Its onset is followed by a rise of temperature and violent dyspnœa, so that in a child the respirations may exceed sixty (Millard). Diphtheritic broncho-pneumonia is nearly always lobular, and not pseudo-lobar. Anatomically it is characterized by a large amount of fibrin and by hæmorrhages into the pulmonary lobules. Klebs' bacilli, and many other microbes—streptococci, pneumococci, and staphylococci—are found in the alveoli. In this variety, as in other kinds of broncho-pneumonia, these secondary infections play a most important part in the production of broncho-pulmonary lesions.

**Pleurisy** is sometimes a complication, but is of moderate importance.

**Diphtheritic coryza**, considered by Trousseau as of evil augury, is often present in the malignant form of diphtheria.

In children **tuberculosis** is often associated with diphtheria of the respiratory tract. "The gravity of tuberculosis is such," says Variot, "that in fifty-four deaths which occurred in January and February at the Bretonneau Annexe, we found tubercular lesions in the thoracic organs sixteen times."

After croup, as after all the local manifestations of diphtheria, we sometimes see **paralysis**; but, as croup is rarely the only manifestation of diphtheria, these paralyses are chiefly due to the concomitant angina. Lastly, the eruptive fevers may appear in the course of croup, and give rise to serious complications.

**Diagnosis.**—In cases where the diagnosis is difficult the laryngoscope must be employed, especially in adults. This method has often confirmed a doubtful diagnosis by revealing the presence of membranes in the larynx.

Acute laryngitis, œdema of the larynx, and spasmodic laryngitis are the diseases which most resemble croup. In **simple acute laryngitis** the vocal and respiratory symptoms are not so severe, and do not gradually get worse, as they do in croup. In **œdema of the glottis**, which is very often but a part of some other disease, the antecedents at once enlighten us. In any

case, the respiratory symptoms are worse than the vocal ; expiration is easier, less prolonged than in croup, and sometimes accompanied by a *bruit de drapau*. The supraglottic œdema, which causes the dyspnœa, may also be visible.

The diagnosis between croup and laryngitis due to hereditary syphilis demands special notice. The symptoms may simulate those of croup so closely that errors have been made. In my lectures at the Faculté I reported the case of a young child who was about to undergo tracheotomy for asphyxia set down to croup. Van Swieten's solution was given as soon as hereditary syphilis was diagnosed, and we succeeded in curing the child in a few days. M. Sevestre has reported several analogous cases. The possibility of laryngeal syphilis must always be considered in a young child who, in the absence of previous diphtheritic angina and of glandular enlargement, presents symptoms analogous to those of croup.

The diagnosis between croup and **false croup** is somewhat difficult. The two diseases have a different onset. The invasion of croup is more insidious, and the respiratory symptoms gradually become intense. The invasion of false croup is more sudden. A child who has gone to bed in good health wakes suddenly, in the middle of the night, suffering from dyspnœa that reaches its height at once, and, this attack over, the patient appears next day to be in good health. In croup the cough and the voice are muffled ; the presence of membrane on the vocal cords explains the aphonia. In false croup the voice and the cough are harsh and noisy, and resemble the barking of a dog. They are not muffled, as in croup, at any rate, between the attacks. During the attack of false croup, however, they may be muffled.

Besides the signs peculiar to each disease which I have just enumerated, careful inquiry should be made into the antecedents of the patient : information should be sought as to whether diphtheritic angina have been recently present, and we must try to discover if some signs of it, such as enlargement of the submaxillary glands, do not remain ! we must not forget that croup is the only malady of the larynx which may be accompanied by the rejection of false membranes.

**Bacteriological Diagnosis.**—The clinical diagnosis of croup is in many cases insufficient, and must be completed by the bacteriological examination of the membranes. Full details are given under Diphtheritic Angina, for diphtheritic angina and laryngitis are closely associated. I recapitulate here the points referring to croup. As I have already said, the diphtheria bacillus is not the only microbe capable of producing false membranes. Just as there are pseudo-diphtheritic anginae, so there is a pseudo-diphtheritic croup. This form of croup is neither contagious nor infectious ; it is not accompanied by toxic symptoms, and is not marked by malignancy ; it is due to a small diplococcus (Brisou's coccus).



Martin records seven cases of croup due to this diplococcus, with membranous angina of the same nature, and twelve cases of croup due to the same coccus, without previous angina. Non-diphtheritic croup, due to the diplococcus in question, is infinitely less serious than diphtheritic croup. It is not toxic; recovery usually takes place without secondary infections and without tracheotomy. In these attenuated forms it may simulate stridulous laryngitis; indeed, I am of opinion that certain cases of false croup are nothing else than a mild laryngitis, due to the diplococcus.

In other cases the bacteriological examination of the membrane in croup reveals both the diphtheria bacillus and the small coccus. This association of the diplococcus and the bacillus is not usually grave, and M. Martin's monograph shows that these cases recover without secondary infections.

On the other hand, when examination reveals the association of the diphtheria bacillus with the staphylococcus or the streptococcus, the prognosis is bad. The same remark applies in the case of diphtheritic angina. The presence of the streptococcus in croup, as in angina, should make us fear grave results. In such a case the symptoms comprise marked glandular enlargement ("the proconsul's neck," Saint Germain), nasal discharge, diarrhoea, and albuminuria, which lead us to fear the death of the child as much from the general poisoning as from the croup.

These few data, with which every physician must to-day be familiar, sufficiently indicate, in my opinion, the importance of bacteriology in the present question. If we cannot make a direct examination of the membranes from the larynx, the membranes and the mucus in the throat must be examined, and, even in the absence of membrane in the throat, the culture from a piece of mucus removed from the tonsil or from the pharynx may often allow accurate **diagnosis** and **prognosis** to be made.

**Secondary Croup.**—This variety supervenes in the course of some other disease, such as measles, scarlatina, whooping-cough, or typhoid fever. The details will be found under these different diseases. In general terms, the secondary forms are less characteristic in their ways than primary croup and more readily take on an infectious character.

**Croup in Measles.**—Diphtheria has a liking for measles. Croup in measles is sometimes primary, and may appear about the same time as the rash. The disease is often mild, and the laryngeal symptoms are slight; yet the prognosis is extremely grave, because of the broncho-pneumonia which accompanies it, and of the double infection of measles and diphtheria. The membranes in the larynx are softer and more diffuent, and the lesions are more of the ulcerative type.

**Croup in Scarlatina.**—Croup is much rarer in scarlatina than in measles; it rarely occurs alone, and more often coincides with diphtheria of the pharynx and the nasal fossæ.

**Croup in Whooping-Cough.**—Secondary croup in whooping-cough comes next to that of measles in frequency.

**Croup in Typhoid Fever.**—Croup is excessively rare in the course of typhoid fever.

**Pathological Anatomy.**—In croup the catarrhal inflammation of the mucosa, the congestion and the oedema of the submucosa, are of secondary moment; the false membrane is the chief lesion. It covers the different parts of the larynx, and especially the aryteno-epiglottic ligaments and the vocal cords, either as a continuous membrane or in isolated patches. In colour it is a yellowish-white, sometimes tinted with minute hæmorrhages. In some cases it is very thin, but in other cases its thickness may be as much as 2 millimetres, owing to the stratified layers which grow on its deep surface. The younger these layers are the greater their resistance, while the older layers, which are pushed towards the surface, have become friable. The false membranes are composed, not only of fibrin, but also of pus corpuscles and epithelial cells from the mucosa, which undergo a colloid infiltration, taken by Wagner for an albuminoid substance. These epithelial cells, converted into refractile blocks, are deformed and branched like a stag's horn (Wagner). Each stratum of the false membrane develops at the expense of the corresponding epithelial layer, and becomes more superficial as a fresh subjacent layer is produced. "It has been debated whether the false membrane is above or below the epithelium; from what has been stated above, we see that it is formed exactly in the superficial layer of the epithelium, and partly at its expense. Its structure, too, seems to differ according to its age; at the outset the epithelial network appears to be prominent, but a little later the fibrinous and purulent elements are in excess" (Leloir).

The mucosa beneath the false membrane is usually intact, and rarely ulcerated. The diphtheria bacilli, the poison which they elaborate, and their association with other bacteria, are described in detail under Diphtheritic Angina.

The diphtheria bacillus undoubtedly gives rise to the membranes, and to the poison which provokes intoxication and paralysis.

**Treatment.**—I cannot here enter into all the details of serotherapy, and the reader is requested to consult the chapter on **Diphtheritic Angina** for further details. I shall here describe the application of serotherapy to croup.

The results of treatment largely depend upon the previous performance of tracheotomy. The results are likewise very different, if the croup be due to the diphtheria bacillus alone, or if it be due to the bacillus associated with the staphylococcus or the streptococcus.

Let us consider these results.



1. A child who is suffering from croup has not yet been tracheotomized. The cough is raucous, the voice is inaudible, the breathing difficult, and the sucking-in well marked. Severe attacks of suffocation occur at brief intervals. At first sight tracheotomy seems unavoidable, but an injection of 20 centimetres of serum is given. Twelve hours later a second injection is given, and in the great majority of cases the injections of serum arrest the formation of fresh membranes, favour the rapid disappearance of those already formed, and recovery is rapid. "In 169 children admitted for diphtheritic angina, fifty-six showed laryngeal trouble, and in twenty-five tracheotomy appeared unavoidable. Under the influence of injections of serum every twelve hours, the sucking-in decreased, and afterwards only returned in fits. The child brought up the false membrane, and after two or three days the breathing was natural, to the great astonishment of the house-physicians and attendants in the diphtheria pavilion, who, from their large acquaintance with children suffering from croup, quite thought the operation could not be avoided" (Roux and Martin).

2. In children suffering from croup who have undergone tracheotomy the success of serotherapy diminishes, according to the nature of the associated microbes. These associations are the same in croup as in angina.

(a) The association of diphtheria with Brisou's **small coccus** is favourable both in croup and in angina. In the statistics of Roux and Martin, the mortality was only one in ten cases, and this was due to broncho-pneumonia following tracheotomy. The quantity of serum injected amounted to 50 c.c. in divided doses.

(b) The association of diphtheria with the **staphylococcus** is to be feared. It is not deadly in the case of angina, but is very fatal in cases of croup after tracheotomy, because it gives rise to pulmonary complications, such as broncho-pneumonia, which often follow the operation, and against which injections of serum are often impotent. In eleven cases Roux and Martin recorded seven deaths, a mortality of 63 per cent. The mean quantity of serum employed in divided doses amounted to 60 c.c. In these patients the membranes are pultaceous and very extensive; the temperature was always above 103° F., and the breathing was much quickened.

(c) The association of the diphtheria bacillus with the **streptococcus** is most formidable, both in croup and in angina. In spite of serotherapy, the mortality in cases of croup which have been operated upon has been 63 per cent., according to Roux and Martin. Broncho-pneumonia and pseudo-membraneous bronchitis are responsible for most of the deaths.

Since many cases of secondary infection can be set down to tracheotomy, this operation must in future be replaced, when possible, by intubation.

**Intubation** of the larynx was extolled by Bouchut, but his instrument rendered the results of intervention so hazardous that physicians followed

Trousseau's advice, and abandoned intubation, tracheotomy being infinitely superior. Great progress, however, has been made in the apparatus for intubation. The method has been so perfected, thanks to Collin's instruments and to Bayeux's technique, that hesitation is no longer possible. In the very great majority of cases intubation will replace tracheotomy.

The writings of Variot and Bayeux and of Martin give us the most complete information on this subject. As Bayeux says: "We run the risks of grave results—nay, even of the death of children—by delay. We risk nothing by intubation when carried out betimes."

## VII. STRIDULOUS LARYNGITIS—FALSE CROUP.

**Ætiology.**—**Stridulous laryngitis** (Bretonneau), or **false croup** (Guersant), is simply an acute catarrhal laryngitis of infancy, which derives its spasmodic character from the tender age of its victims. In small children the intercartilaginous glottis is rudimentary, and the aperture of the glottis is short and narrow. Changes in the larynx are, therefore, readily accompanied by dyspnœa, which in children takes the form of fits. This laryngitis is most frequent from two to six years, is often seen during the invasion of **measles**, and may be the forerunner of broncho-pneumonia.

**Description.**—Trousseau has given so complete an account of this malady that I cannot do better than quote it *in extenso*: "A child between the age of two and five years is seized in the middle of the night—about eleven, twelve, or one o'clock—with an attack of dyspnœa. He wakes out of his sleep in a very uneasy and feverish state. His cough is harsh and very frequent, but strong and noisy; his breathing is jerky, panting, and accompanied by inspiratory stridor. His voice, which is modified in its timbre, is inaudible during the fits, but rough and hoarse in the intervals. There is, however, one capital fact—it is never extinguished, as in true croup."

Oppression and anxiety are sometimes excessive; the face is congested, and the eyes express profound terror. This alarming crisis may last from half an hour to about three hours, but the attack then ends. The child becomes quiet, sleep returns, and the pulse-rate falls. The skin is slightly moist. Then the patient wakes up. The cough is still croupy, though looser. During the day it is still more catarrhal, the breathing is less whistling, and the voice has almost regained its natural tone. As a rule, the attacks recur several nights in succession, but they always decrease in violence, while the days are good, the patient having but little fever or malaise, and suffering from a loose and less croupy cough. On questioning the parents we learn that the child went to bed quite well and fell into a peaceful sleep. Sometimes, on the contrary, we are informed that he had



been out of sorts for some days, that he had taken cold, but had retained his usual spirits. Lastly, if the throat be examined, the most careful examination shows that false membranes and glandular enlargement are absent. This sudden onset, with symptoms which are more alarming in appearance than those at the commencement of croup, is in most cases characteristic of false croup. This malady results in cure, fatal cases being very rare.

**Diagnosis—Treatment.**—The **diagnosis** of stridulous laryngitis is given under Croup. It is always necessary to think of hereditary syphilis of the larynx, which simulates both croup and false croup.

If the reader will refer to **Croup**, he will see, under Bacteriological Diagnosis, that in some children the laryngeal troubles are due to the presence of a small diplococcus, and may resemble both true and false croup. I think that, even in a condition which has every appearance of false croup, we should not neglect bacteriological examination of the mucus or of the pharyngo-laryngeal secretions. A case which is taken for a false croup may be really laryngitis, or pharyngo-laryngitis, due to the diplococcus; membranes may be present or absent. False croup recovers without active treatment. It is sufficient to apply blisters, or a sponge soaked in very hot water, to the child's neck (Graves). A moist atmosphere from a steam-kettle should be maintained around the patient's bed and emollient drinks given. Tracheotomy is only required in exceptional cases. False croup, however, has proved fatal.

## VIII. ŒDEMA OF THE LARYNX—ŒDEMA IN BRIGHT'S DISEASE.

### Syphilitic Œdema.

**Definition.**—The terms “**œdematous laryngitis**,” “**œdematous laryngeal angina**” (Trousseau), “**laryngeal infiltration**” (Jaccoud), and “**œdema of the glottis**,” have been used to describe infiltrations of the larynx, which differ slightly in nature, but present almost identical symptoms. Bayle, who first described œdematous laryngitis (1808), considered it, with good reason, to be dropsy of the larynx, analogous to dropsy of the cellular tissue. Bouillaud and Cruveilhier, on the contrary, endeavoured to show that it had nothing to do with dropsy, but was really an inflammatory lesion, causing the formation of purulent fluid. Both opinions are true. In some cases it is a question of œdema in the true sense of the word, the infiltration being purely serous (nephritis, scarlatina, œdema from cold), but at other times the infiltration is sero-purulent (ulcerative laryngitis, laryngeal infections, erysipelas of the pharynx, tumours of the pharynx and of the tongue, laryngo-typhus, etc.).

In this chapter, however, I shall not consider purulent infiltrations of

the larynx, or, at least, only in an incidental way. I have only in view œdema properly so-called—that is, infiltration of the larynx comparable to œdema of the cellular tissue. I have, therefore, headed this chapter “**Œdema of the Larynx.**” I shall first give a general survey of the question and then discuss œdema as seen in Bright’s disease and in syphilis.

**Site of the Œdema.**—The laryngeal mucosa is not everywhere adherent to the subjacent fibro-elastic tissue. The adhesion is lax at the glosso- and aryteno-epiglottic folds; in the arytenoid region it is quite slight, and it is not close on the vocal cords. Certain of these parts are rich in cellular tissue, and therefore œdema readily forms there.

According to its site, the infiltration may be supraglottic, glottic, or subglottic. The term “**œdema of the glottis**” is therefore improper, and comprises but a small part of the question, because, in most cases, œdema affects parts other than the glottis. **Supraglottic** infiltration is the most frequent, because of the situation of the lesions which produce œdema, and especially because the submucous connective tissue is abundant in this region. The aryteno-epiglottic and glosso-epiglottic folds, the epiglottis, the ventricles of Morgagni, the interarytenoid tissue, and the pharynx itself, participate in the trouble, and the cushions of œdema can be felt by the finger. The œdema is said to be **glottic** when it occupies the vocal cords. When the infiltration is **subglottic**, the laryngoscope reveals a reddish swelling on the side of the trachea.

**Pathological Anatomy.**—The mucosa which covers the œdematous parts is sometimes pale and anæmic, at other times red and injected. The vestibule is much infiltrated; the aryteno-epiglottic folds, which, from the abundance and laxity of their connective tissue, present the chief lesion, are œdematous, and may be enormously swollen.\* The epiglottis has lost its shape, and may be three times as thick as normal. These lesions readily explain the almost complete obliteration of the orifice of the larynx. The œdematous parts, on incision, are found to be infiltrated with serous fluid; œdema sums up the whole lesion. When the infiltration of the larynx is secondary to deep ulcerations—perichondritis, necrosis of cartilages, or sequestra—we find, in addition to the serous or sero-purulent infiltration, the lesions described under Syphilis, Tuberculosis, and Cancer of the Larynx.

**Ætiology—Pathogenesis.**—Œdema of the larynx may be primary, accidental, or secondary. Primary œdema exists as a distinct disease, and may be produced by a chill. Trousseau reports a remarkable case: “A

\* Sestier, by injecting water into the carotids of a cadaver, has produced artificial œdema of the larynx, studied its distribution and compared the size of each of the affected parts. Experimental research, in connection with pathology, shows that the **aryteno-epiglottic folds** are chiefly affected.



drunken man, who slept in the street one cold night, was seized by acute œdema of the larynx." The condition here was "true œdema"; the swelling of the laryngeal mucosa is comparable to that of the nasal mucosa in coryza. I think, however, that laryngeal œdema *a frigore* is very rare, and, on closer inspection, some other factor, such as Bright's disease or syphilis, is often found.

**Accidental** œdema follows wounds and burns. Sestier has collected sixty-nine cases. Œdema secondary to some lesion of the larynx, or of a neighbouring part, is quite common. "Every inflammatory process," says Trousseau, "determines in its neighbourhood œdema which varies in direct ratio with the amount of loose connective tissue. In the eyelids, for example, and on the prepuce, where the connective tissue is abundant and loose, a pustule of variola, or a patch of erysipelas, may cause enormous swelling. This œdema, which Virchow called collateral, and which is sometimes sero-purulent, finds a most favourable soil in the abundant lax cellular tissue of the supraglottic region.

**Laryngeal tuberculosis** may cause œdema of the larynx. The condition in some cases is a false œdema, and is really a tubercular infiltration which invades the folds, the ventricular bands, and the epiglottis; these parts are hypertrophied and indurated by infiltration of tubercular tissue, and the condition is certainly not œdema. Nevertheless, true œdema may appear in the course of laryngeal tuberculosis; it is provoked by ulceration, and especially by tubercular perichondritis and by lesions of the cartilages. The œdema in tuberculosis of the larynx chiefly occupies the arytenoid region; it is usually soft and pale, because of the anæmia of the mucous membrane.

**Cancer of the larynx** causes œdema, which nearly always begins on one of the ventricular bands. It remains unilateral, reaches the arytenoid and the corresponding aryteno-epiglottic fold; it rarely attacks the epiglottis, does not spread far, and develops side by side with the cancer.

**Laryngo-typhus** may determine severe œdema. In the decline of typhoid fever infiltration, due to the necrosis of the cartilages, appears; its onset may be slow or rapid. Scarlatina, especially during defervescence, may set up œdema of the larynx. Trousseau has reported several examples. Acute or chronic nephritis (including saturnine nephritis) may bring on œdema of the larynx. Œdema of the edge of the vocal cords is extremely frequent in young girls who are learning to sing.

### Œdema of the Larynx in Bright's Disease.

The following example will give an exact idea of this condition :\* A man was admitted into my wards with symptoms of asphyxia. His chief complaint was noisy, laboured breathing, with **stridor** audible from one end of the ward to the other. Expiration little affected ; each inspiration required much effort. The inspired air finally entered the lungs, but caused a sawing noise. Inspection showed considerable sucking-in, with depression of the suprasternal and epigastric hollows ; the diaphragm was apparently drawn upwards. The voice was slightly hoarse, a proof that the vocal cords were but little affected.

The man stated that he had been sick for the past month. The dyspnœa was not the first symptom, and the illness began with dysphagia, which gradually became so severe that the passage of food and drink was almost impossible. Ten or twelve days later the patient experienced a feeling of strangulation, which he compared to a foreign body blocking the entrance of the air-passages. The dyspnœa was at first uniform, but later interrupted by paroxysms. These symptoms finally made life almost unbearable. He was worn out, and could not take food. Breathing required the greatest effort ; each inspiration was a struggle for life, and the prognosis was most alarming.



FIG. 1.—ŒDEMA OF THE LARYNX.

*e*, Epiglottis—very œdematous, irregular, and red. It forms an enormous eyebrow above the vestibule of the larynx, and is continuous with the aryteno-epiglottic folds, which are very œdematous.

*a*, Arytenoid region—also swollen, deformed and retracted towards the œsophagus.

*b*, Ventricular bands, which are very œdematous, and mask the vocal cords for the greater part of their extent. The anterior extremity of the vocal cords is visible.

The obstruction was evidently in the larynx, but we had to ascertain its nature. M. Bonnier examined with the mirror. On opening the mouth, we at once noted a reddish œdema of the uvula, of the pillars, and of the velum palati. The uvula was bulky and tremulous, the pillars were œdematous, and the isthmus of the gullet was narrowed. Red œdema was visible at the base of the tongue and in the vestibule of the larynx. The epiglottis was bulky and much deformed, the aryteno-epiglottic folds were enormous, and the œdematous ventricular bands blocked the orifice of the glottis. The anterior part of the vocal cords could be seen momentarily, and their free edge was slightly œdematous. The illustration above represents the condition.

It is now easy to follow the march of events. The dysphagia was caused by the œdema of the palate, the pharynx, and the epiglottis. The dyspnœa was due to œdema of the vestibule and of the ventricular bands ; these parts, by opposing the

\* “ Œdème Brightique du Larynx ” (*Clinique Médicale de l'Hôtel-Dieu*, 1897, 3<sup>me</sup> Leçon, p. 49).



passage of the inspired air, made the breathing difficult and stertorous. Spasms of the glottis, which are almost inseparable from such lesions, caused the fits of suffocation.

The diagnosis of œdema of the larynx was therefore established. The patient had neither tumour, laryngeal polypus, cicatricial stenosis, nor paralysis of the posterior crico-arytenoid muscles, which may all cause suffocation, stertor, and sucking-in, but was suffering from progressive œdema, which had started in the velum palati and the isthmus of the fauces, and had reached the larynx.

The diagnosis, however, was not complete, for laryngeal œdema, whether it be white or red, and local or diffuse, may be due to many causes. Cancer, tuberculosis, and, above all, syphilis, may give rise to œdema of the larynx, while the existing lesion may be almost hidden. The œdema in this case was due to none of these causes; it was not the result of cold, but of **Bright's disease**.

The case was one of Bright's disease. It was not a case in which œdema was severe and uræmic symptoms marked; it was one of those cases of latent Bright's disease. In this patient we found all the minor troubles of "Brightism"—*i.e.*, frequent micturition, cramp in the calves, dead fingers, cryæsthesia, and itching. The arterial tension was exaggerated, the urine contained albumin, and the depuration was incomplete, as examination of the toxicity of the urine showed lowering of the toxic coefficient. We

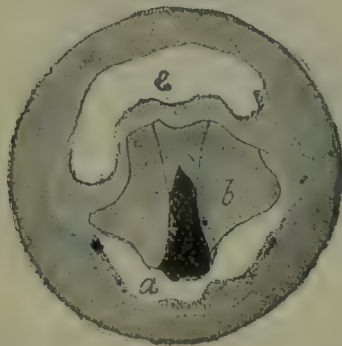


FIG. 2.—LARYNGEAL ŒDEMA.

*e*, Epiglottis—still œdematous, but regaining its normal shape somewhat.

*a*, Arytenoid regions still remain œdematous and deformed.

*b*, Ventricular bands. The swelling has diminished, the glottis is more patent, the vocal cords can be better seen, but their free edge is still irregular and swollen.

had, then, to deal with œdema of the larynx, due to Bright's disease, and producing increasing asphyxia, which might well end fatally.

His condition was so grave that I discussed the question of tracheotomy, or, better still, of intubation. I was prepared, therefore, for any eventuality, but I began by applying several leeches to the front of the neck. The result was not long delayed. On the next day the improvement was manifest: the œdema of the palate had diminished, the dysphagia was less, the stertor was not so noisy, the breathing was easier, and the œdema of the larynx was on the road to recover, as may be seen from Fig. 2.

Milk diet was ordered from the first, and two days later the improvement was still more marked. The annexed illustration (Fig. 3) gives a good idea of the improvement which rapidly followed in the œdematous parts. Six days later the patient was cured.

œdema may supervene in the course of acute or of chronic nephritis. When nephritis is very acute (as in scarlatinal and early syphilitic nephritis), it produces œdema, which tends to become general, and the anasarca is often

considerable. The serous cavities—notably the pleuræ—contain effusions, and serous exudate may be present in the brain, the lungs, or other organs. Œdema of the larynx is then associated with these various exudations.

In chronic nephritis, on the contrary, the œdema has much less tendency to become diffuse; it is more isolated and more localized to one region, such as the face, the lower limbs, or a single organ, and we see patients suffering from slowly progressive nephritis in whom pleural effusion or acute œdema of the lung supervenes, while œdema of the extremities is absent or slight. The same remark applies to œdema of the larynx in chronic nephritis. Œdema of the larynx may occur in the course of insidious Bright's disease, while other parts of the body are free. This patient had never shown œdema in any other part before œdema of the palate and of the larynx appeared. In a patient of Fauvel's no trace of œdema had been seen, and the œdema of the eyelids only appeared after that of the larynx.

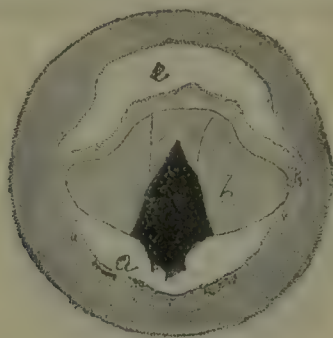


FIG. 3.—LARYNGEAL ŒDEMA.

*e*, The epiglottis has lost all trace of œdema.

*a*, The arytenoid region has regained its normal aspect.

*b*, The ventricular bands still hide the glottis to a slight extent, but they meet only during effort and phonation. The vocal cords are a little irregular along their free border, and the anterior half alone is visible during inspiration, but the air enters the larynx freely.

In Jones's patient slight œdema of the lower limbs was present, when œdema of the larynx supervened. A patient of Fraenkel's had never had any œdema, so that in his case the œdema of the larynx was considered to be the first sign of nephritis. Hanot's patient had never shown the least trace of œdema, when œdema of the palate and of the larynx appeared.

We see, therefore, that in the course of slowly progressive and more or less insidious chronic nephritis œdema of the larynx (like that of the lung) may appear suddenly, without being preceded by peripheral œdema. Accordingly, several authors (Fauvel, Fraenkel) have stated that œdema of the larynx may appear unexpectedly as the first sign of Bright's disease. I do not hold this view. I admit that œdema of the larynx may appear as the first manifestation of œdema in a "Bright" case, but I cannot accept the statement that it may be the first symptom of Bright's disease.



In fact, at the present day we no longer look upon Bright's disease as our ancestors did, and we do not wait for the appearance of œdema to make our diagnosis. We hunt out "Brightism," which may not be evident at first sight. We know that there is a mild form of uræmia which is insidious in its course, but yet perfectly recognizable. The study of the minor troubles of "Brightism," the estimation of the arterial tension, and the examination of the toxicity of the urine, reveal latent Bright's disease. We are thus able to affirm that, although an attack of epileptiform fits, of coma, of super-acute œdema of the lung, or of œdema of the larynx, may appear to be the first symptom of nephritis, it is in reality only the first apparent, and not the first real, symptom, and the nephritis has been already shown by signs which would not have passed unnoticed had trouble been taken to look for them.\*

I must insist on the fact that œdema of the larynx in Bright's disease is almost always **preceded by œdema of the velum palati**. It might truly be said that œdema commences in the throat and the uvula, and afterwards spreads downwards to the base of the tongue, the epiglottis, and the larynx. As a result, **troubles in swallowing** very often open the scene. At first the patient seems to have only a painful angina, but respiratory troubles soon appear. The œdema spreads downwards, causing a succession of complications which I have found in most of my cases. In a patient at the Hôtel-Dieu dysphagia and œdema of the throat preceded œdema of the larynx and dyspnoea by ten or twelve days. In a patient at the Necker Hospital œdema of the throat and dysphagia occurred twenty-four hours before attacks of suffocation. In the cases reported by Abate and Jones œdema of the larynx and œdema of the palate, with dysphagia and suffocation, appeared almost simultaneously. In M. Fauvel's patient the œdema of the palate preceded that of the larynx, and deglutition was impaired before respiration. In M. Hanot's patient the succession and the delimitation of the œdema was even more marked. In the first phase the œdema remained localized to the isthmus of the fauces and to the uvula, as an œdematous angina, causing acute dysphagia, which was cured by milk diet. Two months later, however, œdema of the palate reappeared, and also œdema of the larynx, causing acute attacks of suffocation. Hanot, in his notes on this case, has been careful to add: "Albuminuric œdema, limited to the uvula, may be the starting-point of œdema of the glottis."

In a patient with painful angina the throat must be carefully examined, and if red or white œdema of the uvula, of the pillars, or of the velum palati be present, nephritis must be suspected, the urine examined, the patient carefully questioned, search made for symptoms of "Brightism," and the

\* Dieulafoy: "Étude sur le Brightisme," *Bulletin de l'Académie de Médecine*, séance des 6 et 20 Juin, 1893.

diagnosis of œdema of the throat due to Bright's disease be made. This œdema then becomes an important element in diagnosis, prognosis, and treatment. It is an element in diagnosis, because it helps us to trace Bright's disease which at first might not be suspected; it is an element in prognosis, for it leads us to foresee the probable extension of the œdema to the larynx; it is an element in treatment, for it invites prompt action in order to check the disease and to avoid œdema of the larynx.

Œdema of the larynx is one of the most formidable complications of Bright's disease. In some cases, it is true, it may **remain limited**, and **for several days and weeks** betray itself only by a trifling stridor during inspiration; or, again, it may invade the larynx but slowly, so that complications may be averted. In other cases, however, the œdema may already be most acute, and the patient may rapidly succumb before help can be given. The situation is aggravated by the fact that œdema in Bright's disease does not always remain confined to the larynx, but invades the pleura and the lung. A patient of mine in the Charité had very extensive œdema of the lung, and succumbed some hours after tracheotomy—not so much from the laryngeal lesions as from the pulmonary œdema. Hanot's patient, who had both œdema of the larynx and of the lung, with 2 pints of fluid in the pleural cavity, died some hours after tracheotomy—not so much from the lesions in the larynx as from the pleuro-pulmonary mischief. It is often difficult, I grant, to gauge correctly the state of the lung in a case of œdema of the larynx, because the extreme dyspnœa, and the echo of the "sawing sound" in the larynx do not always allow perfect auscultation of the lung.

What treatment should we employ in this condition of œdema? If death appear imminent, tracheotomy may give some chance of recovery. So many failures have been noted that I would rather advise intubation, which has been tried and proved. Scarification of the œdematous swellings does good. Bleeding is an excellent means of treatment. A dozen leeches are applied to the front of the neck, so as to produce copious bleeding. Hot compresses, warm gargles, and sprays are sometimes of use.

If asphyxia be imminent, we can facilitate the entrance of air into the larynx by firm traction on the tongue. Do not forget that œdema of the lung or uræmia may also be present. Tracheotomy was unsuccessful in the three cases which I have reported, because two patients succumbed to pulmonary œdema, and the third to uræmic convulsions. If pulmonary œdema or uræmia be present, both general and local bleeding are necessary.

The nephritis must also be treated, and absolute milk diet is therefore prescribed. In place of milk by the mouth, we may give injections of lactose (150 grammes of water and 20 grammes of lactose). No chlorides should be taken, and injections of serum must not be given.



### Syphilitic Œdema of the Larynx.

Œdema is fairly rare in the course of secondary lesions of the larynx. Although they are superficial, and apparently benign, they are sometimes accompanied by œdema of the larynx and respiratory troubles. This fact is most important, and has been emphasized by Krishaber. Although dyspnœa may be exceptional in secondary lesions of the larynx (or, at least, it is very slight), yet in some cases the distress becomes suddenly acute, and tracheotomy would become necessary if the condition did not yield rapidly to appropriate treatment. I have often verified this fact, and Mauriac, like Krishaber, affirms that "quite trifling erosions may become a dangerous centre of congestion, around which œdema of the glottis rapidly develops."

Œdema of different colours, with pallor or redness of the mucosa, often accompanies tertiary lesions. A red tint is sometimes the sign of sero-purulent infiltration. Laryngeal œdema **plays a considerable part** in the history of tertiary lesions, and is one of the commonest causes of dyspnœa and of threatening asphyxia. It may be more or less extensive, and may invade the aryteno-epiglottic folds, the epiglottis, the ventricular bands, the vocal cords, the space below the cords, and the trachea. The laryngoscope shows the deformity and the swelling of the parts invaded. The epiglottis is twisted up and swollen like a chestnut or the cervix uteri. The arytenoids form large cushions, which obliterate the supraglottic region.

All tertiary lesions, etc., may at any moment cause œdema of the larynx, which may be limited, and not serious, or quite general. In the latter event, it may be slow or rapid in its progress. Krishaber and Mauriac have studied syphilitic œdema of the larynx, and have shown that its intensity is not always in proportion to the severity of the exciting lesion. As a matter of fact, we meet with shallow lesions which, though scarcely appreciable with the laryngoscope, may yet cause alarming œdema of the larynx. I have several times verified the correctness of these assertions.

In my ward at the Necker Hospital, I had two patients with laryngeal œdema. One of them had stridor and sucking-in, and was almost in a state of asphyxia. The danger was averted by energetic specific treatment. The other patient also had stridor, sucking-in, and feeling of strangulation, and with orthopnœa, which left no doubt as to a laryngeal lesion. It was of several weeks' duration, and had previously caused only vocal trouble. Bonnier found œdema of the larynx. The œdema had, in the case of one vocal cord, the polypoid appearance which can be seen in the subjoined figures. No suspicion of syphilis existed until I discovered on the right wrist a pustulo-crustaceous syphilide, which gave me the key to the case. I ordered active specific treatment, and in three weeks, as the Figs. 4, 5, 6, and 7 indicate, the œdema of the larynx disappeared, and with it all the troubles.

Syphilis may produce œdema of the larynx in yet another way—not by lesions limited to the larynx, but by setting up early nephritis, which is rapidly followed by peripheral œdema, anasarca, effusion into the serous membranes, and œdema of the lung and of the larynx.

This sketch of the importance of syphilitic œdema of the larynx proves how carefully syphilis must be looked for, in order to give injections of biniodide of mercury without delay.



FIG. 4.—ŒDEMA OF THE LARYNX.

*December 21.*—Day of patient's admission. Appearance of the glottis during forced inspiration.

*e*, Swollen and turgid epiglottis.

*a*, Œdematous arytenoid region.

*b*, Ventricular bands, which are markedly œdematous. A small portion of the vocal cords can be seen in front.



FIG. 5.—ŒDEMA OF THE LARYNX.

*January 12.*—*e*, Epiglottis, still swollen, but less flabby.

*a*, Arytenoid region, which is less prominent.

*b*, The ventricular bands have diminished in size, allowing the vocal cords and an œdematous polypoid mass to be seen.

**General Description.**—I have given the special characters of certain varieties of œdema of the larynx, and will therefore recapitulate the general description. I would call attention to the fact that the **march of events** is sudden or slow, according to the cause. The palato-laryngeal infiltration, which is secondary to nephritis, syphilis, scarlatina, or a chill, may rapidly become general, and asphyxia soon appears, while the progress is much slower when the œdema is secondary to certain lesions of the larynx. In



the case of generalized œdema, **respiration** is rapidly compromised. The patient has the sensation of a foreign body which is strangling him, and makes desperate efforts to get rid of it. During the course of the increasing dyspnœa attacks of suffocation supervene, "in which," says Trousseau, "the patient, with livid face, open mouth, gaping nostrils, eyes starting from the sockets, and skin running with sweat," grasps any support for his inspiratory muscles. These attacks last from ten to fifteen minutes, and are repeated in fatal cases at shorter intervals till death occurs. They are



FIG. 6.—œDEMA OF THE LARYNX.

January 16.—*e*, Epiglottis normal.

*a*, Arytenoid region normal.

*b*, Ventricular bands, which are still œdematous, though they allow the whole of the glottis to be seen. The polypoid swelling is implanted on the right vocal cord; it is notably smaller.



FIG. 7.—œDEMA OF THE LARYNX.

January 18.—The larynx has resumed its normal appearance, with the exception of the right vocal cord, on which a remnant of the polypoid excrescence is seen. It may well have originated in a syphilitic papule.

the result of spasm of the glottis, and are only seen in 60 per cent. of cases. Inspiration, which is noisy and accompanied by **stridor**, is usually much more painful than expiration, because the aryteno-epiglottic cushions at the moment of inspiration are said to act as a valve which blocks the upper orifice of the larynx. These cushions, pushed back like floating bodies by expiration, produce a characteristic *bruit de drapeau* (Sestier) in some cases.

Dyspnœa is not so rapid and so severe in many cases. In persons with syphilitic, tubercular, or cancerous lesions of the larynx, œdema supervenes as a complication, and increases gradually and slowly before compromising life. In some cases, however, dyspnœa due to œdema may be rapid and terrible.

The **voice** and the cough are not greatly altered, as in croup, but still may be raucous or muffled if the vocal cords are involved, or if the œdematous parts come in contact.

**Deglutition** is painful, because of the swelling of the laryngeal folds and epiglottis, and also because of the palato-pharyngeal œdema. The œdema of the pharynx and of the vestibule may be recognized both by **sight and touch**.

œdema, when consecutive to syphilitic, tubercular, or cancerous lesions, usually remains limited to the larynx, but œdema due to nephritis is often **palato-pharyngo-laryngeal**: the uvula looks like a lump of jelly, the mucosa of the throat is œdematous, and dysphagia precedes dyspnœa.

Laryngoscopy is sometimes dangerous, for it may produce a severe fit of choking.

The symptoms which I have just enumerated readily explain the gravity of œdema of the larynx. Death often occurs unless we employ active intervention. œdema of the larynx in nephritis is very dangerous. In ten cases of œdema appearing at the **outset** of nephritis, five died, two before and three after tracheotomy; and in ten cases of œdema supervening in the **course** of nephritis, nine died, four before and five after tracheotomy (Améro).

**Diagnosis.**—Foreign bodies and polypi in the larynx, spasms of the glottis caused by aneurysm of the aortic arch, croup and stridulous laryngitis, abscess of the pharynx, paralysis of the posterior crico-arytenoid muscles, and tumours of the mediastinum, may simulate œdema of the larynx, for all these conditions give rise to stridor and sucking-in. They have, however, the distinctive signs which I shall now enumerate. **Polypi** are easily recognized with the laryngoscope. The same remark applies to paralysis of the posterior crico-arytenoid muscles. **Aneurysm of the aortic arch** shows dullness, blowing murmurs, and expansile pulsation if the tumour is large. With the laryngoscope, lesions of the larynx are absent, or limited to the paralysis of a vocal cord, from the compression of the recurrent nerve by the tumour. In **croup** we may note the presence or the remains of the diphtheritic angina which nearly always precedes it, the false membranes which are so often coughed up, the early and gradual alteration of the voice, the slow and difficult expiration, and often the glandular enlargement in the neck. **Stridulous laryngitis** chooses early life, and appears suddenly. The dyspnœa at once reaches its limit, and after the suffocative attack, the relief is complete, and the voice and the breathing recover their



normal character. **Retro-pharyngeal abscess** is visible on the posterior wall of the pharynx, and if the base of the tongue be well depressed, a swelling which may fluctuate and is of a deeper colour than the surrounding tissues is discovered. Œdema of the larynx being recognized, its cause must be diagnosed. The œdema may be primary, but before we admit the existence of primary œdema, or of œdema *à frigore*, careful search must be made for any causes or the lesions which may, sooner or later, give rise to laryngeal œdema. It is important to exclude Bright's disease and syphilis, which are the two chief causes of œdema of the larynx.

I shall not go into the **treatment**, which has already been studied in each special case.

## IX. SPASM OF THE GLOTTIS.

**Definition—Ætiology.**—Spasm of the glottis results from tonic contraction of the constrictor and tensor muscles of the vocal cords. It lasts some seconds, and causes suffocative attacks, which may result in death. The spasm is either symptomatic or idiopathic.

The **symptomatic** spasm arises from stimulation of the recurrent nerves by a neighbouring tumour—*e.g.*, aneurysm of the arch of the aorta, tubercular or cancerous glands, etc. How can excitation of one recurrent nerve alone provoke spasm of the glottis? Krishaber has shown experimentally that stimulation of one recurrent nerve acts upon both lips of the glottis, just as stimulation of one vagus arrests or slows the heart-beat. Symptomatic spasm is also seen in certain diseases of the larynx, such as croup, false croup, or œdema of the glottis, and it is remarkable that the spasm is **intermittent**, although the exciting cause is continuous. This intermittence is met with in other spasmodic or painful actions of the nervous system (hepatic and renal calculi, neuralgia in cancer), though it is not easy to give the reason.

Spasm of the glottis may appear alone, or be associated with other laryngeal troubles, as at the outset or during the course of locomotor ataxia.

**Idiopathic** spasm of the glottis is not, like the preceding form, a symptom appearing in the course of another disease, but a real morbid entity which occurs in children from four to eighteen months old, and it is this form that chiefly merits description here. This disease has been improperly called thymic asthma, because it was thought to be connected with hypertrophy of the thymus. It is really a neurosis, which may be independent or related to dentition or to digestive troubles. Heredity may play some part, as several infants in the same family may be affected (Romberg).

**Description.**—The idiopathic spasm of infancy, which may or may not be preceded by prodromata, such as pains in the back and hands, or convul-

sions, begins suddenly in the middle of the night, like false croup. The glottis closes convulsively; the breathing becomes more and more difficult, and is soon completely suspended, so that asphyxia is imminent. The distress is extreme; the chest is immobile, and the vesicular murmur can no longer be heard. The face is cyanotic and bathed in sweat, the heart-beats are tumultuous, and we ask ourselves if the scene is not about to end in death. After fifteen or twenty seconds of complete apnoea, "the child takes a quiet breath again, and the attack ends by a high, sonorous inspiration that is quite characteristic, and does not resemble the croupy cough or the whoop in pertussis" (Tardieu). It is more like a shrill and very sharp hiccough (Hérard).

The attacks are not always complete, and the asphyxial period may be wanting. The usual duration is from a few seconds to two minutes, but in some cases they recur at very short intervals, and are prolonged for an hour or more (Gaspari). At first these attacks are only repeated once or twice a week; later, they reappear daily, and as many as twenty-five to fifty have been counted in a day (Hérard). Between the attacks the child's health is good; fever, cough, and loss of appetite do not occur, and cachexia does not appear till late. The total duration of the malady is very variable, but its limits lie between a few weeks and several months (Hérard). The prognosis is most gloomy, and recovery is the exception.

The symptomatic spasm in the **adult** presents some differences. The shape of the larynx and the resistance of the interarytenoid glottis explain why the spasms are less formidable than in the infant, and also why the inspiration is wheezing, some air being able to pass through the glottis.

**Diagnosis.**—Stridulous laryngitis, which also results from a spasmodic contraction, resembles spasm of the glottis; but the former attacks children from one to six years old, while the latter is seen between the ages of three and twenty months. The former is preceded or accompanied by catarrh, hoarseness, cough, and coryza; the latter comes on so suddenly that asphyxia threatens in a few seconds.

**Hereditary syphilis** often produces in young children laryngeal troubles which are so like spasm of the glottis that I am convinced that in many cases (treatment proves the point) syphilis is the cause.

**Treatment.**—In the attack the face should be sprinkled with cold water and friction to the body should be employed. The spasm often yields when the child is forced to breathe through the nose. Antispasmodics are indicated. Change of air and a stay in the country are advisable. The possibility of hereditary or acquired syphilis must be thought of, and mercury and iodides employed.



## X. PARALYSIS OF THE MUSCLES OF THE LARYNX.

Laryngeal paralysis may be classed as of peripheral, bulbar, or cerebral origin, according to the seat of the lesion.

**Peripheral Lesions.**—The laryngeal nerves may be directly injured by traumatism (Neumann), by surgical intervention, and by compression. Compression is more frequent in men than in women, and affects the left recurrent nerve more often than the right (Avellis). It may be due to tumours of the thyroid body or of glands, abscess, cancer of the œsophagus, aneurysms of the great vessels of the mediastinum and the root of the neck, pericarditis, and pleural effusions. Tumours of the base of the skull and fractures may compress or injure the vagus and the spinal accessory nerves. Tabes and diphtheria are accompanied by lesions of the laryngeal nerves, and the same holds good in certain intoxications and infections (alcoholism, saturnism, morphinism, syphilis, and diabetes). Finally, more or less lasting paralysis of the muscles of the larynx may be set down to extra- or intra-cervical action of cold.

**Bulbar Lesions.**—The nuclei of the spinal accessory nerves may be affected by the following lesions: Syphilitic growths, tumours, caries, softening, hæmorrhage, acute or chronic inflammation of the bulb, pachymeningitis, disseminated sclerosis, amyotrophic lateral sclerosis, labio-glosso-laryngeal paralysis, and tabes.

**Cerebral Lesions.**—The foot of the third frontal convolution (Garel), the subjacent white matter (Dejerine), the external part of the internal capsule (Garel and Dor), the caudate nucleus, and the claustrum (Picot and Hobbs), have been found diseased in cases of laryngeal palsies. The same troubles may be due to hysteria, or may be provoked by suggestion.

The palsies may attack several groups, a single group, or a single muscle.\*

**Paralysis of the Dilators.**—Isolated paralysis of the posterior crico-arytenoid muscles is very rare (Ziemssen), though it has recently been noted in laryngeal phthisis (Gougenheim). When both muscles are paralyzed, the vocal cords no longer open, but fall together during inspiration from the action of the constrictor muscles, and, since very little air enters through the glottis, **intense dyspnœa** results. Vocal effort is possible, and the voice is slightly altered through imperfect fixation of the arytenoids. If the paralysis is unilateral, the immobility of the vocal cord at the moment of inspiration may be seen with the mirror.

**Paralysis of the Constrictors.**—Paralysis of the constrictors is much more common than the preceding form. It may be bilateral (hysteria and diphtheria), but is more often unilateral, and almost always consecutive to a

\* To complete this study, see p. 23 *et seq.*

direct lesion of the recurrent or of the spinal accessory nerves. The length of the course and the relations of the recurrent nerves explain the relative frequency of this palsy. The causes of the lesion include aneurysm of the carotid artery (Mackenzie), double paralysis from a cancerous tumour at the posterior lacerated foramina (Türk), cancer of the recurrent nerve (Heller), double paralysis by aneurysm of the subclavian and innominate vessels (Ziemssen), aneurysm of the arch of the aorta (numerous cases), cancer of the

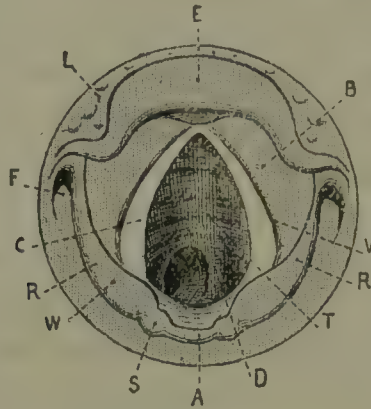


FIG. 8.—NORMAL LARYNX: APPEARANCE OF THE GLOTTIS DURING DEEP INSPIRATION.

E, Epiglottis; L, base of the tongue; B, ventricular bands; V, ventricle of Morgagni; R, aryteno-epiglottic fold; A, interarytenoid region (distended); S, cartilage of Santorini; W, cartilage of Wrisberg; C, vocal cords, opened to their maximum; F, hyoid fossa; T, trachea, with superposed rings; D, bifurcation of the bronchi.

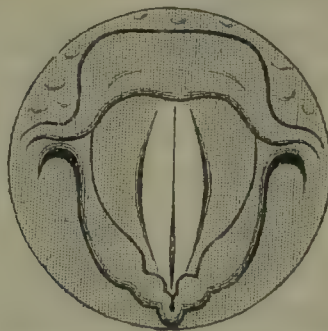


FIG. 9.—NORMAL LARYNX: APPEARANCE OF THE GLOTTIS DURING PHONATION.

The vocal cords are tense, and nearly touching in their anterior two-thirds. The cartilages of Wrisberg and of Santorini are approximated, and the interarytenoid region is partially effaced. The epiglottis is straightened, and the larynx is elongated in the antero-posterior direction.

oesophagus (Braune), chronic pleurisy of the right apex (Gerhardt), chronic pericardial effusions (Baümmler), and mediastinal tumours (Guéneau de Mussy).

In paralysis of the constrictor muscles the glottis is constantly open, so that respiration is easy; but the voice is lost, and effort is incomplete. These symptoms are less marked when the paralysis is unilateral. The laryngoscope shows that both cords (or one of them, according to the case) remain immobile, and do not come together.



**Paralysis of the Crico-Thyroidei.**—These muscles are supplied by the external laryngeal nerve. The paralysis induces no respiratory trouble, because the dilator muscles are intact. Effort is complete, because the constrictors are healthy, and the voice is not completely lost, because the other muscles of phonation are intact; but the voice is altered, and dysphonia is present, because the tension of the vocal cords is insufficient, and their

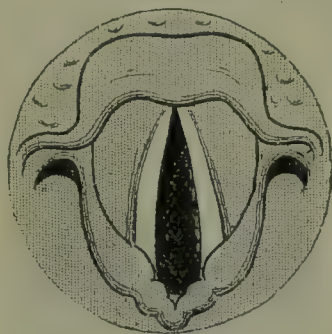


FIG. 10.—PARALYSIS OF THE ADDUCTORS DURING AN EFFORT OF PHONATION.

The lateral crico-arytenoid muscles and the ary-arytenoid muscles are paralyzed. The vocal cords are relatively tense, but they cannot approximate to the middle line. The orifice of the glottis is triangular.

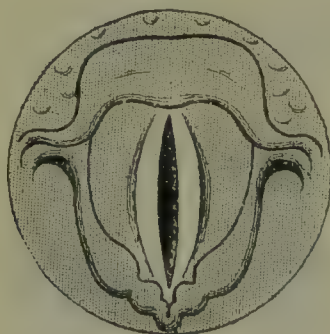


FIG. 11.—PARALYSIS OF THE TENSORS: APPEARANCE OF THE GLOTTIS DURING AN EFFORT OF PHONATION.

The vocal cords appear curved, and sometimes beaded at their free edge, and the glottis gapes slightly. This appearance corresponds to paralysis of the crico-thyroid muscle, or to paralysis of the elevator muscles of the larynx.

vibration is incomplete during the emission of sound. This paralysis occurs in hysteria. It comes on after great vocal effort, and often follows a chill, like paralysis of the facial or of the radial nerve.

**Paralysis of the Elevators.**—In order that the crico-thyroid muscles may produce the tension of the vocal cords necessary for phonation, the thyroid cartilage must be fixed above and in front by the elevators of the larynx—*i.e.*, genio-hyoid, mylo-hyoid, digastric, stylo-hyoid, and thyro-hyoid muscles. If these muscles, which are chiefly innervated by the hypoglossal nerve, are paralyzed, the larynx remains depressed, the tension is incomplete, and the

aphonia is almost absolute. The voice has a low, monotonous tone, due to the passive tension of the vocal cords under the strain of the expired air.

**Diagnosis—Prognosis—Treatment.**—Laryngoscopic examination simplifies the diagnosis of paralysis of the larynx, and the discovery of the paralysis may give a clue to its cause. Mediastinal tumours may at first give no symptoms save vocal troubles due to paralysis of one vocal cord (compression of the recurrent nerve). We are not liable, thanks to direct examination, to mistake paralysis of the dilators for **spasm** of the constrictors,



FIG. 12.—PARALYSIS OF THE LEFT RECURRENT NERVE: APPEARANCE OF THE GLOTTIS DURING AN EFFORT OF INSPIRATION.

The left cord reaches beyond the middle line, being pulled over by the overaction of the muscles supplied by the right recurrent nerve.

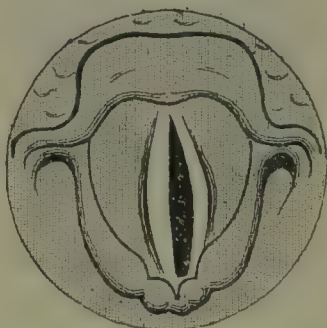


FIG. 13.—PARALYSIS OF THE LEFT RECURRENT NERVE: APPEARANCE OF THE GLOTTIS DURING AN EFFORT OF PHONATION.

The left vocal cord remains in the cadaveric position. The right vocal cord passes slightly beyond the middle line, and is alone tense.

although in both cases the respiratory troubles are almost similar. Again, paralysis of the muscles of phonation will not be confounded with **atrophy** of the vocal cords, or **ankylosis** of the arytenoid articulations, though the vocal troubles present great analogy. For similar reasons, laryngeal phthisis, cancer, and polypi of the larynx can be excluded. The diagnosis between paralysis of the crico-thyroid muscles and catarrhal laryngitis has been given in the description of the latter malady.



The **prognosis** is only grave in paralysis of the dilator muscles (posterior crico-arytenoidei), when dyspnoea may be so acute as to necessitate tracheotomy.

**Treatment** varies with the cause of the paralyses. When they are primary, electricity is the best curative agent, especially in paralysis of the crico-thyroid muscles. The voice often reappears at the first sitting. Cure is obtained by applying the electrodes over the region of the larynx, or even at any two parts of the body (Krishaber).

## CHAPTER III

### DISEASES OF THE BRONCHI

#### I. ACUTE BRONCHITIS

**Acute bronchitis** is a catarrhal inflammation of the large and medium-sized bronchi. It is often associated with inflammation of the trachea, and, indeed, in some cases **tracheitis** is the chief trouble. Tracheo-bronchitis is often preceded by laryngitis, and "The cold has settled on the chest" is quite a common saying.

Inflammation of the bronchioles will be studied later, under **capillary bronchitis**.

**Description.**—Acute bronchitis shows different degrees of severity. Thus, the **slight** form, commonly called a cold on the chest, is almost apyretic, and is only accompanied by slight malaise and headache. The cough at first is painful, dry, and spasmodic, and the expectoration is serous and greyish; but later the cough becomes loose, the sputum is thick, and in a week the illness is at an end.

In the **severe** form bronchitis begins with general malaise, shivering, and fever, which rises at night. The patient complains of headache and loss of appetite. The breathing is wheezing and painful. The cough, which is at first dry, causes acute pain in the intercostal muscles and the diaphragm, and is often followed by the vomiting of mucus or of food.

On percussion, the resonance of the chest is normal; on auscultation, **snoring and sibilant rhonchi** are heard during inspiration and expiration on both sides of the chest (because bronchitis is always double), and are transmitted in different directions.

These initiatory symptoms last from three to five days, and coincide with the **immature** stage. When secretion from the bronchial mucosa (called the **mature** stage) replaces the dry swelling of the immature stage, the fever falls, the breathing becomes easier, the cough is loose, the sputum thick and yellowish-green, and the column of air, instead of being broken up against the rough, dry mucosa, meets the liquid bronchial secretion.



As a result, the rhonchi change to râles, which acquire the timbre of bubbles bursting in a liquid, and are known as **bubbling, mucous, or subcrepitant**. Mucous râles in a large bronchus may simulate the gurgling of a cavity; subcrepitant râles are finer, and have their origin in the smaller bronchi. Unlike rhonchi, the moist râles may disappear at certain spots after a fit of coughing has freed the bronchi from the obstructing mucus.

The second stage lasts about a week, during which improvement gradually supervenes, and resolution is complete in about a fortnight. The urine now becomes abundant, and contains much sediment. Slight diarrhœa often completes the **crisis**. The disease may become chronic, or change to capillary bronchitis. In old people who cannot cough up the secretion bronchitis may turn to suffocative catarrh.

**Ætiology.**—Acute bronchitis is a disease of cold and damp weather, and in certain predisposed persons it appears on the least chill, or at the approach of winter. It is one of the chief elements in measles and influenza. It is less frank in whooping-cough, asthma, and hay-fever, where the nervous element is so much in evidence that the inflammatory element is much less marked. The bronchitis present at the commencement of typhoid fever is rather hyperæmic than inflammatory, though it may turn to true catarrh, and become a troublesome complication.

Bronchitis in Bright's disease is not a genuine one, and its special characters depend on pulmonary œdema or uræmic dyspnoea. The same remark applies to pseudo-bronchitis, which is associated with heart disease, and is especially characterized by signs of congestion and stasis in the lungs. Dust and irritating vapours may produce an inflammation of the bronchi, usually associated with lesions in the lungs.

I make special mention of **syphilitic** bronchitis. The erythema and the catarrh, so frequent in the larynx during the first years of infection, also affect the trachea and the large bronchi. This subacute tracheo-bronchitis is liable to relapse from exposure to cold. Many syphilitics, formerly immune to chills, complain that they take cold easily, and suffer from laryngitis and bronchitis. They are always liable to erythema and specific catarrhs of the larynx, the trachea, and the bronchi—catarrhs which are readily excited by chills.

Bronchitis in **young children** deserves special mention. It seems sometimes to be associated with teething and digestive troubles, and in this connection the *Bacillus coli* has been said to play a prominent part. Even the most trifling forms of bronchitis in young children may be complicated by congestion of the lung. The temperature rises, and we hear at the bases of the lungs fine râles, which lead us to fear the onset of capillary bronchitis. All symptoms subside, however, after one or two days (Cadet de Gassicourt).

**Diagnosis.**—It is not enough to diagnose bronchitis, for that is quite simple; but it must be distinguished from conditions which may simulate it; above all, its cause must be made out, as the different forms require absolutely different treatment. Syphilitic bronchitis, to wit, demands treatment with mercury and iodides; bronchitis in Bright's disease only improves with milk diet; bronchitis in cardiac cases yields to the treatment suitable for heart diseases. Bronchitis in tuberculous patients cannot always be diagnosed by a study of the symptoms alone. Its tubercular origin is in some cases doubtful until bacilli are found in the sputum.

Speaking broadly, apparent bronchitis which does not begin in a regular manner must be suspected; a bronchitis which begins with fever is often spurious. On the other hand, we must be equally suspicious of so-called bronchitis in which cough is the chief symptom, to the exclusion of fever and of expectoration. Because an individual coughs much, we must not at once suppose that he is suffering from tracheo-bronchitis. Hysterical persons suffer from frequent and incessant cough without having bronchitis. We find people in whom the *tænia* and other worms provoke a cough that is quite wrongly styled bronchitis. After expulsion of the *tænia* the cough disappears.

**Bacteriology.**—Bacteriology cannot help us in classifying bronchitis. The microbes which inhabit the healthy air-passages, and include the *Staphylococcus albus* and *aureus*, the *Streptococcus pyogenes*, the pneumococcus and the pneumobacillus, may all be found in bronchitic sputa, and are in no way specific. Besides these microbes, the sputum in bronchitis often contains a bacillus identical with the *Bacillus coli*, a fungus of the genus *Oidium*, and microbes which give the sputum its yellowish or greenish colour.

**Treatment.**—In slight cases we should induce sweating by diaphoretic drinks. We must give soothing inhalations, and quiet the pain and the fits of cough by the following draught:

|                       |    |    |    |    |    |        |
|-----------------------|----|----|----|----|----|--------|
| R Orange-flower water | .. | .. | .. | .. | .. | iv.    |
| Syrup of chloral      | }  | .. | .. | .. | .. | āā 3v. |
| Syrup of morphia      |    |    |    |    |    |        |
| Cherry-laurel water   | .. | .. | .. | .. | .. | 3ii.   |

Dose: For an adult, a tablespoonful every two hours.

In the severe form we should employ stimulating applications (mustard-plasters and blisters) and dry-cupping to the chest. If the bronchi is much choked, especially in old people, emetics must be given.



## II. CAPILLARY BRONCHITIS—BRONCHO-PNEUMONIA—LOBULAR PNEUMONIA

**Discussion.**—Should the temperature rise, the respiration become panting, and auscultation show fine subcrepitant râles on both sides of the chest, on the sixth, seventh, or eighth day, in a child suffering from **measles** complicated by ordinary bronchitis, **capillary bronchitis** has set in. The thermometer soon registers  $104^{\circ}$  F., the dyspnœa becomes acute, and the respiration rate rises to 60, while dullness and tubular breathing are found over one lung or at both bases. **Broncho-pneumonia** has supervened.

Another child has reached the stationary stage of **whooping-cough** without accident. In spite of the convulsive cough, fever is slight or absent, and but few bronchitic râles are heard in the intervals of the fits. Fever then appears; the temperature rises; the character of the cough changes, being more continuous and less convulsive; the breathing becomes panting, and on auscultation fine scattered râles are audible on both sides of the chest. **Capillary bronchitis** has begun. The thermometer now registers  $104^{\circ}$  F., the cough becomes incessant, the dyspnœa is extreme, percussion and auscultation show one or several pneumonic patches. **Broncho-pneumonia** has developed.

In another child suffering from **croup**, who has or has not been operated upon, the situation appears quite favourable. The fever is very moderate, no toxic symptoms are present, and the prognosis is good; but suddenly the temperature rises, the dyspnœa grows worse, and is no longer of laryngeal but of bronchial origin. Auscultation, although difficult, because of the laryngeal sounds, reveals fine râles in the chest. This condition means **capillary bronchitis**. Then the thermometer rises, and the dyspnœa becomes excessive. The dreaded **broncho-pneumonia** of diphtheria has set in.

In an infant suffering from teething or from gastro-intestinal troubles a bronchitis, which may or may not be due to chill, begins. On auscultation, large bronchitic râles are heard, yet so far there is little fever or dyspnœa. The fever suddenly increases, the dyspnœa grows, the râles become fine and scattered. **Capillary bronchitis** is beginning. The temperature reaches  $104^{\circ}$  F.; the intensity of the dyspnœa becomes acute; auscultation shows tubular breathing at both bases. **Broncho-pneumonia** has supervened.

In an adult who has had an apparently normal **influenza**, with moderate fever, bronchitis, and catarrhal expectoration, the temperature rises, the dyspnœa grows worse, the expectoration becomes muco-purulent, and on auscultation râles of all kinds are audible. These symptoms indicate **influenzal capillary bronchitis**. A step further, and bronchial breathing is

perceptible in different spots—or at both bases. **Broncho-pneumonia** is present.

These different examples prove that we find not a “single broncho-pneumonia,” but “broncho-pneumonias,” which, strictly speaking, may all follow bronchitis from cold, though they most often supervene in the specific infections of measles, whooping-cough, diphtheria, influenza, tuberculosis, typhoid fever, erysipelas, etc.

In some cases the capillary bronchitis is the trouble, especially in adults and the lobular pneumonia is of less importance. In other cases, on the contrary, the capillary bronchitis is not important, but has joined hands with broncho-pneumonia, which is then the chief lesion.

Bacteriological research in broncho-pneumonia permits in some cases the isolation of such specific bacilli as those of diphtheria, typhoid fever, or tuberculosis, but these specific microbes alone are not sufficient to cause broncho-pneumonia. They are not always found in the foci of broncho-pneumonia, while other microbes normally present in the mouth or the air-passages are always found. These microbes, which are the true cause of broncho-pneumonia, are the *Staphylococcus albus* and *aureus*, the *Streptococcus*, the *Pneumococcus*, the *Pneumobacillus*, and sometimes the *Bacterium coli*. They increase the danger and make the prognosis worse.

We can now begin the detailed study of capillary bronchitis and of broncho-pneumonia.

**Definition—History.**—When inflammation affects the bronchioles, the bronchitis is said to be **capillary**. In ordinary bronchitis the larger bronchi are alone attacked, and therefore the air can circulate freely, in spite of the lesions present. In **capillary** bronchitis, on the contrary, where the small bronchi are inflamed, the narrowness of the tubes, the thickening of their mucosa, and the obstruction by morbid products prevent the free passage of air, and produce dyspnoea, which too often ends in asphyxia. This dyspnoea is such a marked symptom that it gained for the malady the name of **suffocative catarrh** at a time when neither the seat nor the nature of the lesion was known with certainty. Under the terms “suffocative catarrh,” “peripneumonia notha,” and “false inflammation of the chest,” Sydenham had confused bronchitis, asthma, and emphysema. His successors followed the same track, and Home, all through his work on membranous exudates, found the explanation of every kind of suffocative bronchitis in the false membranes. Laënnec put an end to the confusion, and showed that suffocative catarrh is a phlegmasia of the bronchi, but he attributed its extreme gravity only to the extent of the lesions. Andral substituted the more correct idea of the seat of the phlegmasia, and localized the lesions of capillary bronchitis in the bronchioles. The works of Gendrin, Rilliet and Bathez, Fauvel, Legendre, and Barrier then appeared, and it may be



said that capillary bronchitis, as now established, was the work of the French school.

Capillary bronchitis, however, rarely exists in a pure form. More often **in children—we might say always**—the pulmonary lobules participate in the inflammation, and the disease takes the name of **broncho-pneumonia** (Seiffert), or of lobular pneumonia. This disease often follows measles, whooping-cough, and diphtheria.

**Pathological Anatomy.**—In a child who has died of capillary bronchitis and broncho-pneumonia we see, after opening the thorax, that the lungs do not collapse. The edges and the apices are emphysematous. The posterior and lower part of the lungs is bluish or brownish, and in these regions, which are dark and violet-coloured, isolated or confluent nodules of lobular pneumonia are perceptible both to sight and to touch.

Whether capillary bronchitis exist alone, **which is rare**, or be associated with inflammation of the pulmonary lobules, which is the usual condition, the inflammation of the small bronchi determines two kinds of lesions. The former affects the **bronchi**, the latter the corresponding **pulmonary lobules**. The mucosa of the bronchioles is swollen and smooth, instead of being folded longitudinally, as in the normal state. The vessels are engorged with blood, and allow the white corpuscles to pass out. The epithelial cells lose their cilia, and become vesiculated; they desquamate or become segmented, and give birth to new elements. The glands increase in size, and allow epithelial cells and muco-pus to escape by their ducts. The sub-epithelial connective tissue is infiltrated with leucocytes, and in the bronchioles near the lobule the muscular fibres disappear, owing to the invasion of embryonic tissue.

The inflamed bronchioles are enlarged in calibre. These **acute dilata-tions** chiefly invade the smaller bronchi, and we see bronchioles which measure a centimetre instead of 1 to 2 millimetres in diameter. The dilata-tions are usually cylindrical, sometimes ampullary, and appear, when the lung is cut, as cavities which are filled with pus. They may be isolated or may communicate with one another. These dilatations are due partly to the mechanical action of secretions driven back by the inspired air, but chiefly to the diminished resistance of the inflamed tissue, which is deprived of its muscular elements. They exist in the broncho-pneumonia of young children, but may disappear with the cure of the disease. The inflammatory products, including cells, leucocytes, and fibrinous exudate, accumulate in the bronchi, which are “filled or almost obliterated from the branches of the second size to the smallest ramifications by a yellowish-white substance” (Hardy and Béhier).

This obliteration of the small bronchi induces changes in the **corre-sponding pulmonary lobules**. These changes have been explained as follows:

The inspired air cannot reach the lobules, because the small bronchi are choked with inflammatory products. During expiration, however, by the help of coughing, the air previously contained in the lobules can overcome the resistance of the plugs which act as valves, and close the lumen of the bronchi. As a result, the infundibula empty themselves of the contained air, and, as this air is not renewed, the lobules collapse, and cause the collapsed parts to look like the lung of a foetus which has never breathed, whence the name of **foetal state** applied to this lesion (Legendre). There is no proof of the absolute truth of this theory, and it may be asked if the air in the alveoli is not simply absorbed there. This fact would explain the production of the foetal state.

The foetal state, also called **atelectasis**, or **pulmonary collapse**, is chiefly met with at the edges and the base of the lungs in young subjects with broncho-pneumonia. The collapsed parts are bluish, brownish, or violet-coloured, and do not crepitate on pressure. The cut section is smooth and uniform. They do not float in water, and although they may have this character in common with lobar pneumonia, they differ totally in that they can be blown out. Under the microscope, the alveoli are seen to be intact, but the vessels are gorged with blood. This stasis causes oedema, and atelectasis is often the first stage of **splenization**. So far, having in view only the changes proper to capillary bronchitis, I have described two lesions: the first active—that of the bronchus; the second partly mechanical—the foetal state of the lung. Let us study the lesions of **broncho-pneumonia**.

The diversity of the lesions complicates the description. There is not one lesion, as in pneumonia, and the morbid process is not homogeneous, but comprises bronchitis, congestion, hepatization, splenization, atelectasis, emphysema, etc. Sometimes splenization, at other times hepatization, is the chief factor, and as these expressions will often recur, it will be well to explain their meaning. We already know the signification of **atelectasis**, or the foetal condition. **Hepatization**, so called because of its coarse resemblance to liver tissue, is the result of the inflammatory process which affects certain parts of the lung. **Splenization**, so called because of its coarse resemblance to spleen tissue, results from lesions which are rather congestive than inflammatory, and is a kind of epithelial pneumonia. **Carnification**, so called because of its rough likeness to muscular tissue, is a condition of the pulmonary lobule described under Chronic Broncho-Pneumonia.

More exact knowledge as to the structure of the lung has led us to abandon the idea that, as regards structure and circulation, the bronchiole was quite independent of the pulmonary lobule. Extension of inflammation from the one to the other, which was formerly supposed to be the exception, is really the rule, so that any lobular pneumonia may be preceded or accompanied by capillary bronchitis. The bronchus, so to say,



carries the inflammation to the lobule. Inflammation of the small bronchi sometimes precedes the outbreak of the lobular pneumonia; at other times their appearance is almost simultaneous. **Lobular pneumonia**, or broncho-pneumonia, was called **catarrhal** when it was supposed to comprise only surface lesions. This designation is insufficient, for the lesions of lobular pneumonia are in some cases deep and parenchymatous.

The lesions affect both lungs, and by choice the lower lobes and their posterior part. The islets of broncho-pneumonia may be **diffuse** or **confluent**. In the former event, they vary in size from a pea to a walnut, and contain one or several inflamed pulmonary lobules. They are scattered here and there in the middle of lung tissue, which may be healthy, congested, bluish, violet-coloured, collapsed, or emphysematous. This form is called **disseminated** lobular pneumonia. In the latter event, the lesion involves a large number of lobules, and may even affect an entire lobe. The disease then takes the name of **confluent**, or **pseudo-lobar**, pneumonia. Besides the principal lesion, some scattered islets are also found.

In the **disseminated** form the nodules, which are of variable size, and often lozenge-shaped, are prominent, hard, and red in the congestive period; they are greyish in the next stage, because of the pus cells in the alveoli, and finally become yellowish and caseous.

The pseudo-lobar form differs from the preceding in the confluence of the nodules, which present various changes. Near a nodule in which pus is forming nodules which are simply congested or splenized may be seen; consequently, the bulk of the lung presents diversity of colour and of consistence.

The inflamed lobules are often superficial, and infundibula, filled with beads of pus, which have been termed purulent graulations (Fauvel) and vacuoles (Barrier), are sometimes seen on their surface. These granulations are yellowish, rounded, as large as a pin's head, and most common on the surface of the lung. The vacuoles, which have the same origin, also contain pus, but are larger, because the alveolar septa are destroyed.

On section, the cut surface presents a less granular appearance than in lobar pneumonia. As regards the minute structure, if we choose a well-inflamed lobule, and make the section at right angles to the intralobular bronchus, we see under the microscope—

1. In the centre the bronchus is dilated and choked with cells and pus. The walls are in part infiltrated with embryonic cells, and have lost almost all their muscular tissue. These points are best seen in very young children, but the lesion is curable. The bronchial dilatation, however, does not always disappear, and broncho-pneumonia may lead to chronic bronchiectasis.

2. Around the intralobular bronchus and its artery a zone of inflamed alveoli and ducts is found. This is the zone of **hepatization**, also called the **peribronchial nodule** (Charcot).

In this nodule the phases of engorgement, and of red and grey hepatization, can be seen. The lesions of engorgement are as follows: Cut surface smooth, tissue spongy, red, and uniform; crepitation has disappeared; tissue sinks in water; under the microscope, the intralobular bronchus is seen surrounded by distended vessels, which are crammed with red corpuscles; the alveoli contain red corpuscles and fibrino-albuminous fluid. The lesions of red hepatization are as follows: On section, mottled surface; around the bronchus, granulations like those of lobar pneumonia; under the microscope, alveoli filled with leucocytes and fibrino-purulent exudate; walls of the alveoli infiltrated with leucocytes. The lesions of grey hepatization are: Nodule is less firm on section, dry, and mottled with red; fibrinous exudate is replaced by pus; intralobular connective tissue infiltrated with leucocytes; tissue of the bronchus and the peribronchial nodule often blended in a puriform sheet, in the midst of which the pulmonary artery appears as a landmark. The peribronchial abscess, which is never perilobular, is formed in this way.

3. Around the peribronchial nodule is the zone of **splenization**, which is smooth on section. Splenization is characterized by lesions which are less inflammatory than those of hepatization. The alveoli contain a fibrinous exudate, with leucocytes and epithelial cells, derived from the alveolar walls, which have proliferated. This is the zone of **epithelial pneumonia** (Charcot). The walls of the alveoli at first undergo no change of structure. The perilobular spaces are well marked by the inflammation, but the suppuration does not affect them.

I have just described splenization in its relations to hepatization of the pulmonary lobule, but **splenization** is sometimes the chief lesion in broncho-pneumonia, especially in the pseudo-lobar form, nodules of hepatization being absent. The splenized lung is red, œdematous, heavy, resistant, smooth on section, and filled with muco-pus, which comes from the bronchi. "This splenization, which is a kind of epithelial pneumonia, might well be consecutive to the obliteration of the bronchi" (Charcot). The epithelial cells of the alveoli swell and desquamate, the congestion is intense, albuminous exudate occurs both inside and outside the alveoli, and if the lesion persists for some time the alveolar epithelium tends to become cubical, and the peri-alveolar connective tissue is thickened. Certain parts of the lung are affected by **inflammation** and **congestion**, independent of any pneumonic process.

The peribronchial nodule, or zone of hepatization, is associated with phlegmonous inflammation of the intralobular bronchus, while the lesions



of splenization are associated with obliteration of the bronchi in the splenized areas.

Whatever be the form of the broncho-pneumonia, the congestion is generally intense in both sets of vessels, though it is most marked in the bronchial vessels. Hæmorrhage also occurs in the interior of the pneumonic nodules. The entire lymphatic system may be involved, the bronchial glands are enlarged and congested, the intralobular spaces are thickened, and the lymphatic spaces are filled with white corpuscles.

Visceral pleurisy is frequently met with over the superficial nodules of lobular pneumonia, and subpleural hæmorrhages have often been observed by Parrot.

Lastly, in those who have died from broncho-pneumonia **emphysema** is often seen, especially at the upper lobes and the anterior borders of the lungs. This lesion, joined with the congestion, explains why, post mortem, the lungs appear cramped in the thorax. Such, then, are the many lesions of broncho-pneumonia. Changes in the small bronchi, atelectasis, splenization, hepatization, hæmorrhage, emphysema, combine in different degrees to produce the various anatomical types which I have just described.

**Experimental Broncho-Pneumonia.**—While lobar pneumonia is difficult to reproduce in animals, broncho-pneumonia may be readily caused in them.

The introduction of ammonia, essence of turpentine, and perchloride of iron through an opening in the trachea produces capillary bronchitis and broncho-pneumonia in animals. The same result is obtained by section of the vagi in the dog, an operation which favours the entrance of foreign bodies into the bronchi (Traube), and also that of pathogenic microbes. When the experiment has been quite successful, the lungs of the animal present all the lesions previously described: obstruction of the inflamed bronchioles, atelectasis, splenization, nodules of lobular pneumonia, and emphysema.

**Bacteriology.**—Microbes are the active agents in broncho-pneumonia. It has been found possible to produce broncho-pneumonia in animals by injecting into the trachea cultures of different microbes. Several species of microbes may give rise to broncho-pneumonia in man. The following table gives their relative frequency in adults (Netter):

|                                      | Per Cent. |    |    |    |    |       |
|--------------------------------------|-----------|----|----|----|----|-------|
| Pneumococcus .. .. .                 | ..        | .. | .. | .. | .. | 38·47 |
| Streptococcus .. .. .                | ..        | .. | .. | .. | .. | 30·77 |
| Encapsuled bacillus .. ..            | ..        | .. | .. | .. | .. | 23·08 |
| <i>Staphylococcus pyogenes</i> .. .. | ..        | .. | .. | .. | .. | 7·68  |

These various microbes are often associated. The pneumococcus is most important in adults, the streptococcus in children.

Mosny believes that the anatomical varieties of broncho-pneumonia correspond to the particular microbe—streptococcus in the lobular, pneumococcus in the pseudo-lobar form. Netter does not hold this opinion.

These different microbes are often found in the mouths, the nasal fossæ, and the bronchi of healthy persons. Why do they become virulent at certain times? I do not know, and I refrain from theories.

In broncho-pneumonia, which supervenes in the course of diphtheria and of typhoid fever, the specific bacillus of these maladies may be found in the diseased lung, or may be absent, while the broncho-pneumonic focus always contains the other microbes previously quoted. The specific bacillus of diphtheria, of typhoid fever, and the as yet unknown microbes of other diseases, do not therefore appear to be capable *per se* of causing broncho-pneumonia. Their associates must be present, and these, the real cause of broncho-pneumonia, are the microbes of which I have spoken above. It has been asked if bacteriological examination of the sputum would not throw light upon the prognosis of broncho-pneumonia by establishing a scale of gravity according as the chief pathogenic agent be the pneumococcus, the pneumobacillus, or the streptococcus; but the researches undertaken on this subject have as yet given no positive result.

**Symptoms—(a) Capillary Bronchitis.**—The symptoms of invasion—*i.e.*, the fever and the elevation of temperature—are more marked than in simple bronchitis. The fits of coughing are more painful, and are often followed by vomiting of mucus or of food. **Dyspnœa**, which is rapid in its appearance, is the chief symptom; indeed, it is not seen with such severity in any other acute inflammation of the bronchi or of the lung. The dyspnœa is as severe as the suffocation produced by the false membrane of croup or by œdema of the glottis, and, indeed, the mechanism is much the same, for in both cases it is a question of foreign bodies which prevent the free passage of air: only in disease of the larynx “the obstacle occupies the common trunk of the air-passages; in capillary bronchitis it is diffused in the terminal ramifications of the tree” (Jaccoud). The dyspnœa is continuous and progressive. It is not interrupted by suffocative attacks and by remissions, as in the diseases of the larynx to which I have alluded, and soon reaches its acme, so that the respiration rate may be 50 in an adult and 80 in a child. The extreme distress, the small irregular pulse, the jerky voice, the working of all the inspiratory muscles, the violet tint of the lips, the pallor of the face, and the coldness of the extremities, bear witness to the increasing asphyxia.

During the asphyxial period the scene is heartrending. The patient sits up in bed; his face is pale and covered with cold sweat; his body is bent forward and propped up by his arms behind; he tries instinctively to help his respiratory muscles, but yet, in spite of all his efforts, hæmatisis is



imperfect, and the blood is loaded with carbonic acid (**anoxæmic poisoning**). This toxic stage is characterized by cardiac weakness. The pulse is small and intermittent, the face becomes livid, the urine is scanty, and delirium appears.

When capillary bronchitis ends favourably, recovery is announced by the ease in expectoration; the sputum becomes more abundant, yellowish, and viscid; the fever falls, and the dyspnoea gradually diminishes. Remissions, however, must be mistrusted, for they are sometimes temporary, and may be followed by fresh inflammation.

The physical signs are as follows:

**Percussion** of the chest yields normal resonance. **Auscultation**, both during inspiration and expiration, gives **fine subcrepitant râles** scattered in front and behind over both sides of the chest. The large râles of ordinary bronchitis are often heard. Récamier gave the name *bruit de tempête* to these sounds, which form a perfect tumult. The sputum is composed of the thick purulent exudate from the small bronchi, and of a frothy secretion from the larger bronchi. In children there is no sputum. In some cases the secretion is so abundant that the patient repeatedly fills his spittoon with pus, as though he vomited it.

It has been asked if capillary bronchitis can be quite independent of broncho-pneumonia. It may be, especially in an epidemic. Whether the cause be cold, influenza, or measles, capillary bronchitis may be unaccompanied by broncho-pneumonia. It is, however, always accompanied by emphysema of the antero-superior parts of the lung and by splenization of the postero-inferior parts. We may also find yellow granules, subpleural ecchymoses, and congestive foci which would have ended in broncho-pneumonia if death had not been so prompt.

(b) **Broncho-Pneumonia**.—This malady behaves so differently, according to age, that it must be studied separately in adults and in children. Broncho-pneumonia in the adult presents some of the symptoms which have just been mentioned under capillary bronchitis. To this description the following symptoms must be added: The temperature rises to 104° F., and the sputum may be streaked with blood, but is not rusty. If the inflammation only attack isolated lobules, the signs on auscultation are but little changed. If, however, the lesion occupy a large surface, and especially if it assume the form of **pseudo-lobar pneumonia**, dullness, tubular breathing, and bronchophony appear simultaneously at the seat of the lesions, although these signs are not so severe as in lobar pneumonia.

As the pneumonic lesions vary in intensity, rough bronchial breathing may be heard at one point and faint tubular breathing at another. These lesions are usually multiple, and invade in succession several regions in one, or both lungs, but especially the posterior and inferior parts. The sibilant

and the mucous râles are more or less fine, numerous, and scattered, according to the concomitant changes in the bronchi. In children broncho-pneumonia is frequent, and is usually a very serious disease. As we shall see under *Ætiology*, it is rarely primary, and usually supervenes in the course of simple bronchitis, or appears as a complication of measles, whooping-cough, diphtheria, or influenza. In infants it is very often tubercular.

As a rule, high temperature, quick pulse, and severe dyspnoea indicate the invasion of broncho-pneumonia. Dullness and more or less harsh, tubular breathing indicate the part of the lung invaded. If the lesion is inflammatory, it takes some days to run its course; if, however, it is congestive, it may disappear by the next day, and reappear at another point, which fact gives to broncho-pneumonia an appearance of mobility. Moreover, as the inflammatory and hyperæmic lesions of the lung are differently combined, numerous clinical varieties may be observed.

Broncho-pneumonia usually matures by successive outbreaks. Sometimes one part of the lung is attacked before another part has cleared up; at other times the new outbreak supervenes after two or three days of improvement, which made recovery appear probable. On auscultation, the tubular breathing is in part masked by subcrepitant râles. It is not rare to find several foci of broncho-pneumonia. They are usually present on both sides, and more often at the base or the middle of the lung. The existence of a rub or of ægophony indicates concomitant pleurisy. The little patient, who is breathless and excited, utters plaintive cries, and the continual working of the *alæ nasi* indicate the violence of the dyspnoea. The respiration rate may reach 50, 60, or even 80 a minute. The cough is constant and overpowering, the face is pale, the lips are blue. When broncho-pneumonia is of moderate intensity, it lasts from a fortnight to three weeks, and ends in recovery. We see, however, cases in which it carries children off in less than a week, and also fulminant forms which are fatal in two or three days.

In the aged, broncho-pneumonia affects different forms. In the acute form, which much resembles the variety in an adult, we find violent cough, progressive dyspnoea, muco-purulent or muco-sanguinolent expectoration, with multiple râles and tubular breathing. In the suffocating form (suffocative catarrh) the dyspnoea rapidly becomes alarming; the expectoration is scanty, viscid, and sometimes quite airless; the pulse is small and irregular; the vital forces quickly fail; the extremities become cold, a clammy sweat appears, and the patient dies in delirium or coma.

**Course—Duration—Prognosis.**—The course of capillary bronchitis and of broncho-pneumonia has been divided into two periods—the one dyspnoeic, the other asphyxial—while the two together may last from one to three weeks. This division, however, is artificial. The prognosis is most grave.



Death is common in children and old people, especially when the disease occurs in an epidemic form, and when it breaks out in a children's hospital as a sequel to whooping-cough, measles, and diphtheria. I have twice remarked that in children suffering from capillary bronchitis with measles the bronchitic symptoms improved on the appearance of pneumonia, as if under the influence of a natural revulsive. The fine scattered râles partly disappeared, as if the inflammatory process were about to concentrate itself at certain points. It seems at first sight as though the localized lesions could be easily controlled, but new foci of lobular pneumonia develop, and after several alternations in the course of events the termination is too often fatal.

In favourable cases the dyspnœa mends, the fine râles disappear, and improvement is gradually established. Sometimes broncho-pneumonia shows a **subacute** course, and may finally become **chronic**. This course, although it be rare, is especially seen in the pseudo-lobar form, where splenization is the chief lesion. I shall refer to it under Chronic Pneumonia.

**Diagnosis.**—The diagnosis between capillary bronchitis and acute tuberculosis in the adult is given later.\* Let us at present differentiate capillary bronchitis from simple bronchitis, pseudo-membranous bronchitis, pulmonary œdema, and congestion of the lung.

The sonorous rhonchi and the mucous râles of **simple bronchitis** show no resemblance to the **sharp, fine** râles of capillary bronchitis; the slight distress in the former is quite unlike the terrible dyspnœa of the latter. The appearance of these signs, however, in the course of a simple catarrh indicates the change from ordinary to capillary bronchitis. **Pseudo-membranous** bronchitis may occur without membranes in the larynx and the pharynx. The membranes which line the ramifications of the bronchi cause fits of dyspnœa, similar to those of capillary bronchitis; but as the patient brings up long, branched membranes, the diagnosis is obvious. **Œdema** of the lung alone, or associated with **passive congestion**, is seen in Bright's disease, in mitral and tricuspid lesions, and in all cases where the pulmonary circulation is impeded. Auscultation reveals fine subcrepitant râles, which are most abundant at both bases; fever is absent, the dyspnœa is intense, and the expectoration is frothy, albuminous, and rosy, when the case is one of superacute œdema of the lung.

The diagnosis of broncho-pneumonia from lobar pneumonia is easy. Lobar pneumonia, as a rule, appears suddenly in healthy persons, and begins with stitch in the side and shivering; while broncho-pneumonia most often develops in those who are already ill (measles, whooping-cough, diphtheria, influenza). Lobar pneumonia is nearly always unilateral and

\* *Vide* Chapter IV., Section 16.

characterized by crepitant râles and tubular breathing, while broncho-pneumonia affects both sides of the chest at multiple centres, which are more or less extensive, and characterized by blowing breathing and multiple râles. In lobar pneumonia the cough is dry, and the sputum in adults is rusty; in broncho-pneumonia it is loose, incessant, overpowering, and the sputum is muco-purulent. Genuine pneumonia usually ends from the fifth to the ninth day, and its termination is almost always favourable in children; broncho-pneumonia is much more formidable, and lasts weeks or even months.

If the reader will turn to the chapter on **Asthma**, he will see that certain febrile forms of asthma, especially in children, may at first sight simulate capillary bronchitis.

The diagnosis of broncho-pneumonia from acute tuberculosis is often very difficult. Both may supervene in the course of another disease (measles, whooping-cough); in both we find extreme dyspnœa, high temperature, early cyanosis of the face, with subcrepitant râles and blowing breathing. The predominance of the lesions at the apices and the presence of blood in the sputum are presumptive signs in favour of tuberculosis, and the presence of bacilli is a certain sign. Sero-diagnosis gives valuable information.

**Ætiology.**—Capillary bronchitis alone is hardly ever seen except in adults; broncho-pneumonia is especially a disease of infancy and old age. Measles, whooping-cough, influenza, diphtheria, typhoid fever, erysipelas, cholera, and tuberculosis in infancy, are the affections in which broncho-pneumonia usually appears. I refer to these different diseases for the study of the special characters which broncho-pneumonia assumes in each. It often occurs after tracheotomy. It is more frequent in cold weather, and sometimes assumes an epidemic form.

Suppurative and infectious lesions of the nasal fossæ and of the throat may cause broncho-pneumonia by descending infection. The pathogenic agents pass through the larynx and the bronchi to the lungs; in some cases the lung is affected, although the intermediary air-passages appear free.

Simple bronchitis may in some circumstances be followed by capillary bronchitis and broncho-pneumonia.

Epidemics of broncho-pneumonia have been noted in workmen employed in crushing slag during the manufacture of steel. The dust and the microbes which are present both take their share in the pathogenesis.

Let us also note the broncho-pulmonary lesions secondary to such conditions of the nervous system, as cerebral hæmorrhage, softening, general paralysis, mental affections, etc.

**Treatment.**—In capillary bronchitis and broncho-pneumonia the indications are identical. We must reduce the broncho-pulmonary inflamma-



tion and favour the expulsion of the secretions which block the bronchi. The first indication will be attained by means of revulsives, blood-letting, dry- and wet-cupping, and application of leeches. Kermes, repeated emetics, ipecacuanha alone, or with tartar emetic, fulfil the second indication.

For a child :

|                        |    |    |    |    |         |
|------------------------|----|----|----|----|---------|
| R Syrup of ipecacuanha | .. | .. | .. | .. | ̄ii.    |
| Powdered ipecacuanha   | .. | .. | .. | .. | gr. xv. |

Give a teaspoonful or a dessertspoonful every five minutes till vomiting occurs.

For an adult :

|               |    |    |    |    |         |
|---------------|----|----|----|----|---------|
| R Ipecacuanha | .. | .. | .. | .. | gr. xx. |
| Tartar emetic | .. | .. | .. | .. | gr. i.  |

Divide into two doses, give at five-minute intervals, and make the patient drink tepid water to assist vomiting.

For the fits of coughing and the pain the following draught will be useful :

For an adult :

|                       |    |    |    |    |       |
|-----------------------|----|----|----|----|-------|
| R Orange-flower water | .. | .. | .. | .. | ̄iii. |
| Syrup of chloral      | }  | .. | .. | .. | āā̄v. |
| Syrup of morphia      |    | .. | .. | .. |       |
| Cherry-laurel water   | .. | .. | .. | .. | ̄ii.  |

A tablespoonful to be taken every three hours.

Inhalations of oxygen are of some use in asphyxia. Lastly, the strength of the patient must be carefully supported by broths, tonics, and alcohol. A child two years old can take during the day a draught containing 25 grammes of sherry and 15 centigrammes of acetate of ammonia.

Baths, both cold and hot, have been extolled (Renaut). This treatment cannot, and ought not, to be systematic. In a case with marked fever, very high temperature, and an ataxic tendency, baths at 75° F. are to be used. In a case with great bronchial embarrassment, much dyspnœa, and an adynamic tendency, hot baths are preferable.

Injections of serum, in amount appropriate to the age of the patient, may be used with advantage (*vide* appendix on **Therapeutics**).

Prophylactic treatment must not be neglected, and children who are predisposed to colds and bronchitis must not be allowed to come near those suffering from broncho-pneumonia.

### III. CHRONIC BRONCHITIS.

**Ætiology—Description.**—Although chronic bronchitis may be primary, it is often associated with constitutional maladies. In lymphatic and scrofulous subjects, in gouty persons, and those affected with skin lesions,

and in old people, bronchitis may be chronic from the first, or follow upon more or less acute attacks. Chronic bronchitis is most prevalent during cold, damp weather, and chills provoke acute attacks, which have a very bad effect upon the disease.

Chronic bronchitis is characterized by long and painful fits of coughing, which are frequently repeated, especially in the morning and the evening. Animated conversation or a quick walk may bring on a fit which is often followed by abundant expectoration. The sputum is thick, greenish-yellow, and often mixed with froth. More rarely the expectoration is scanty, and the balls of sputum have the consistence of starch (dry catarrh). The breathing is wheezing and difficult. On auscultation, snoring and sibilant rhonchi and large bubbling râles are heard over both sides of the chest; they may be so abundant as to simulate the gurgling of a cavity. There is no fever, the appetite is scarcely affected, and the dyspnoea is slight, apart from the fits of coughing. **Chronic** bronchitis has no fixed course; it lasts several months, improves in fine weather, but reappears with the first cold.

In arthritics subject to skin lesions, such as eczema or lichen, it is not rare to see a certain alternation between the cutaneous and the bronchitic troubles.

Chronic bronchitis usually leaves behind it pulmonary emphysema, but the patient may live indefinitely till complications change the situation.

These **complications** are of various kinds. Some, sudden in their appearance, comprise pulmonary congestions, which cause attacks of dyspnoea and acute broncho-pulmonary inflammations, which create immediate danger; others, slow in their development, are **pulmonary emphysema** and lesions of the **right heart**, which give rise to increasing difficulty in breathing, cyanosis, and general œdema.

**Dilatation of the bronchi** is also one of the consequences of chronic bronchitis. In feeble subjects, especially elderly ones, who have no longer strength to expel the secretion in the bronchi, chronic catarrh may change into a suffocative one.

In the course of bronchitis the breath and the expectoration may sometimes become **foetid**. This foetor, which may last weeks and months, is due to decomposition of the bronchial secretions, to the presence of butyric acid (Laycock), or to gangrene of the mucosa, which, however, is not so serious as gangrene of the lung.

Certain persons suffering from chronic bronchitis may bring up for months and years branched, and often cylindrical, **membranes**. These may measure from 4 to 6 inches in length, are whitish, elastic, and chiefly composed of mucine. They must not be confused with diphtheria of the bronchi.



**Diagnosis.**—Chronic catarrh of the bronchi must not be confounded with spurious bronchial inflammation, which is only an episode in the course of some other disease. In people suffering from **cardiac** disease (mitral lesions), the broncho-pulmonary circulation is easily impeded, and congestion and œdema of the respiratory passage are the result. Multiple râles are heard on auscultation, and the distress sometimes takes the form of fits. If the cardiac lesion is not recognized, these different troubles are set down to chronic bronchitis until other symptoms call attention to the true cause.

The same remark applies to bronchitis in patients with **Bright's disease**. A patient complains of distress and suffocation, accompanied by cough and expectoration. On listening, we find mucous râles on both sides of the chest, usually most abundant at the bases. A superficial examination would readily lead to the idea of subacute or chronic catarrh, but a minute interrogation reveals other important signs. The patient has frequent micturition, his eyelids are puffy, the heart is hypertrophied, and we find a gallop rhythm (Potain); the urine is albuminous; and, in short, the so-called bronchitis is only an episode in Bright's disease.

We shall see later that chronic bronchitis is often associated with asthma, dilatation of the bronchi, and emphysema. It precedes or follows them, and may, especially in asthmatics, become the chief trouble, so as to mask the real nature of the disease.

**Pathological Anatomy.**—The diseased bronchi are thickened, and the connective tissue of the mucosa is invaded by fibrous tissue, which sometimes forms prominent vegetations. It is not rare to meet with superficial ulcerations at the glandular openings. Emphysema is often seen as a complication.

**Treatment.**—The end in view is to dry up the secretion and modify the secreting parts. Creosote (Bouchard), iodoform (Sée), and essence of turpentine give good results. These different medicaments may be administered by the respiratory passages as vapours (inhalation-rooms), and sprays (atomizers), or by the digestive tract.

The following formulæ relate to the administration of these remedies :

Capsules of creosote, each containing 1 minim. Dose, 4 to 8 with each meal.

Perles of essence of turpentine, each containing 4 minims. Dose, 4 to 6 with each meal.

Pills of iodoform, each containing 1 grain. One pill to be taken with each meal.

Capsules, containing 4 minims of copaiba and 4 minims of tar. Dose, 4 to 8 with each meal.

Terpene may be employed, giving daily 6 to 12 pills, each containing  $1\frac{1}{2}$  minims, amounting to from 10 to 15 minims in twenty-four hours.

Good results may be obtained with eucalyptol, given in doses of from 4 to 10 capsules daily.

Arsenic and sulphur should be used with these different remedies. The sulphur cures of Saint-Sauveur, Luchon, and Cauterets, and the arsenical ones of Mont-Doré and La Bourboule, will be of much service. I often prescribe Labassère water, taken in the morning, fasting. The dose is 4 table-spoonfuls in hot milk. In old people especially the blocking of the bronchi by secretion must be carefully watched, and combated by means of repeated emetics.

The patient who suffers from chronic bronchitis should avoid chills and pass the winter in a temperate climate.

#### IV. DILATATION OF THE BRONCHI—BRONCHIECTASIS.

**Pathological Anatomy.**—When we read the chapter which Laënnec devoted to dilatation of the bronchi, and consider that this subject was previously untouched, we are astonished at his great accuracy.

The lesions found post mortem are usually multiple, because dilatation of the bronchi is accompanied by chronic broncho-pneumonia, fibrosis of the lung, chronic pleurisy, with adhesions and pulmonary emphysema—lesions which are more or less marked according to the case. On opening the chest, the lungs do not collapse; in certain parts they are indurated, carnified, and hepatized, and their surface is mottled and cyanotic. In some cases a lobe is atrophied and cirrhotic. The lesions of recent acute broncho-pneumonia are not rare. In some cases the dilatations appear on the surface of the lungs; in others they are only seen on section in the depth of the organ. The large bronchi are rarely dilated; bronchiectasis occurs chiefly in the third and fourth order of bronchi, which are 2 to 3 millimetres in diameter.

The pathological anatomy of the dilated bronchi comprises their shape, structure, and situation.

The **shape** is variable. Uniform dilatation, the rarest form, affects the whole length of the bronchus, the calibre alone being altered. Moniliform dilatation (Cruveilhier), by its successive swellings, gives the bronchus the appearance of a string of beads. Ampullary dilatation is the commonest form, and is made up of cavities which may be isolated, like a cyst, or communicate with neighbouring dilatations. In the latter case, the invaded area resembles the auricle of the heart, or an alveolar mass in which the septa are formed by the atrophied and fibrous lung tissue.

Ampullary dilatation has different forms, according as the enlargement invades the lateral parts of the bronchus, or an entire segment. These enlargements, or **bronchial aneurysms**, have been compared to aortic



aneurysms—a comparison which is the more justified as it is the change in the bronchial wall which is chiefly responsible for the swelling. The dilatations vary in size from a pea to a pigeon's egg; the peripheral portion of the bronchus, which is usually atrophied, terminates in a cul-de-sac, but it may in exceptional cases be dilated into a kind of cyst on the surface of the lung (Gombault). The different forms which I have described may be found combined in the specimen.

The **structure** of the dilated bronchus varies according to the stage of the lesion. Where the lesion is but little advanced, the mucosa of the cavity is almost healthy, and is continuous with that of the bronchus; the cylindrical epithelium remains; the glands are atrophied; the submucous connective tissue has lost some of its elastic folds; and the muscular fibres are much separated, but rarely destroyed.

When the lesion is very advanced, the bronchi have quite lost their normal structure; the mucosa is no longer wrinkled, because the elastic fibres have disappeared; the cylindrical epithelium (propulsive) is replaced by pavement (protective) epithelium; the mucosa is covered with villi, which are composed of embryonic tissue and vessels. The muscular layer is converted into embryonic tissue with new vessels; the cartilages also return to the embryonic state, and are infiltrated with calcareous salts. The elastic fibres of the connective tissue are broken up, and no longer form a complete ring around the bronchus. The connective tissue forms, so to speak, the skeleton of the diseased bronchi. The capillary vessels are numberless, tortuous, and dilated; they present swellings of every shape, and project into the cavity of the bronchus. This capillary network is especially developed in the villi and in the layers adjoining the lumen of the bronchus. The vessels anastomose in every direction and form a cavernous tissue.

In the lesions of bronchiectasis there are, then, two kinds: the one—neo-formative—ends in the development of embryonic and vascular tissue; the other—destructive—causes a loss of normal tissue (Hanot).

In certain cases ulceration occurs, and the surface of the cavity may be attacked with “a superficial curable gangrene, which is analogous to the death of connective tissue in phlegmon.” The liquid in the dilated bronchi is composed of mucus and pus in variable proportions; it contains crystals of margarine and of cholesterine, and its mawkish odour becomes foetid in cases of gangrene. When a dilatation has lost its connections with the parent bronchus, it becomes filled with a caseous mass, and looks like an isolated cyst.

Bronchiectasis chiefly affects the apex of the lung when the dilatation is moniliform, while it more often occurs at the periphery when the swelling is ampullary. The dilatations are more frequent on the left side than on the right, and affect one lung only in the proportion of 26 to 4 (Barth). Bronchiectasis is always accompanied by chronic inflammation of the

tissues—*i.e.*, peribronchitis, fibrosis of the lung, and chronic pleurisy—but the origin of these tissues of new formation has not been completely elucidated. According to recent researches, the tissue of the chronic pneumonia which accompanies bronchiectasis is said to arise solely in the fibro-vascular framework of the lung, while the epithelium of the parenchyma takes no part, and the pulmonary tissue itself disappears by reason of the marked diapidesis which gives rise to the new fibrous tissue. “The parenchyma of the lung is transformed into granulation tissue, which finally forms adult connective tissue, more or less studded with vascular neoplasms, according to the age of the lesions. According to their more or less advanced organization into fibrous tissue, we may see the splenization, the carnification, and the areolar state of the parts of the lung which surround the dilated bronchus.” Emphysema often accompanies bronchiectasis. Hypertrophy of the bronchial glands is sometimes seen, and tuberculosis, which was regarded as rare (Barth), appears to be fairly often associated with bronchiectasis (Grancher).

**Ætiology.**—Bronchiectasis is a disease of middle and advanced age. It may result from acute bronchitis or broncho-pneumonia, but more often it follows chronic bronchitis, or broncho-pneumonia which has not cleared up, and is consecutive to measles, whooping-cough, influenza, or typhoid fever. In some cases the ætiology completely escapes our grasp.

The chief theories of the pathogenesis of bronchiectasis are :

Laënnec : Accumulation of secretion in the bronchi, and consequent enlargement of these channels.

Andral : Alteration in nutrition and diminished elasticity of the walls of the bronchi.

Corrigan : Fibrosis of the lung, the retractile tissue of which pulls upon the walls of the bronchi.

Stokes : Paralysis of the muscular fibres and diminution in the resistance of the bronchi.

Mendelssohn : Pressure exerted by the air upon the diseased bronchi during fits of coughing.

Gonbault admits the union of these different processes.

Barth adds chronic pleurisy, acting, like pulmonary fibrosis, by its retractile tissue.

Which of these theories are we to adopt ? Any morbid state capable of producing faulty nutrition of the bronchi (acute and chronic bronchitis), and any formation of extrabronchial fibrous tissue, might favour or produce bronchiectasis.\* I admit the fact, but we must assign the proper value to each of these factors.

\* Paludism appears to act in this way in developing interstitial pneumonia. The cases of Frerichs, Lancereaux, and Grasset, are quite conclusive (Thèse de Grasset, Montpellier, 1874).



It seems to me that we have exaggerated the rôle of chronic pneumonia and of pleurisy—in short, of fibrous tissues—when we look on them as retractile, and capable of producing bronchiectasis by mechanical action. There are cases, it is true, in which the action of the extrabronchial fibrous tissue appears evident; perhaps, too, the bronchial and extrabronchial lesions develop on parallel lines, but the present tendency is to admit that the pulmonary lesion which accompanies the bronchial dilatation is most often the consequence, and not the cause, of the lesions in the bronchus.

The phlegmasia begins in the bronchus; it then extends around the bronchus, reaches the neighbouring pulmonary tissue, and thus gives rise to a systematic fibrosis. The proof that events must follow this course is that chronic lobar pneumonia is not accompanied by bronchial dilatation, because the bronchus does not take part in the morbid process (Charcot), while bronchial dilatations are found in lobular or broncho-pneumonia, because the bronchus is always much altered. Therefore, whether there may or may not be pulmonary or pleural fibrosis, the lesion of the bronchus is the necessary fact that is indispensable to the production of bronchiectasis. By reason of the bronchial lesion the bronchus loses its normal elements, the elastic and muscular fibres give place to embryonic and vascular tissue which have no resisting power, and the bronchus dilates at its weak spot like an artery, the altered wall of which permits the formation of an aneurysm.

It has been asked if there might not be some special condition capable of preparing the way for enlargement of the diseased bronchus, and arterio-sclerosis of the bronchial arteries has been suggested. In a case recently reported by Hanot arterio-sclerosis of the bronchial arteries was very marked.

**Symptoms.**—Bronchiectasis shows itself by the following symptoms: The cough is frequent and spasmodic, as in chronic bronchitis, which often precedes dilatation. The expectoration becomes abundant, so that patients bring up daily 4 to 12 ounces of muco-purulent fluid, with a mawkish and sometimes foetid smell (**bronchorrhœa**). If the sputum is examined in the spittoon when its superficial layer alone is visible, it is frothy and aerated; but if it is put in a measure-glass, it will be seen to have an opaque lower layer, which contains pus corpuscles, epithelial cells, and crystals of cholesteroline or of margarine. The expectoration may be uniformly divided through the course of the day. Some patients, however, empty their dilated bronchi three or four times daily; they are seized by fits of coughing or straining, like a fit of vomiting, and bring up each time 2 or 3 ounces of muco-purulent liquid. This fluid, which smells like fresh plaster, may become foetid from decomposition of the fluid or from gangrene of the cavity. The breath then acquires such a stench that a whole ward may be tainted.

This foetor, which is very tenacious, may exist, with or without remissions, for months and years, and the unhappy sufferer avoids all company, and dare not show himself in public.

**Hæmoptysis** is frequent in bronchiectasis. In some cases it can be set down to tuberculosis, which may be associated with bronchiectasis, but in a large number of cases there is no question of tuberculosis. The hæmoptysis depends solely on the bronchiectasis, and the bleeding is due to rupture of the tortuous, dilated capillaries (angiomata) which are present in the newly-formed bronchial and extrabronchial tissue. These forms of hæmoptysis are a fresh source of difficulty in diagnosis. In some cases they have been the cause of death.

In most cases dilatation of the bronchi exists on one side only, and affects the apex of the lung as often as the base. We find, on examination of the chest, a depression, which corresponds to the site of the dilatation, and is especially marked when bronchiectasis is associated with chronic pleurisy. This depression is frequently found at the middle and back part of the thorax. Percussion shows dullness, corresponding to the diseased region. On auscultation the **signs of a cavity** are found, but the signs vary according to the size and number of the cavities, and according as they are empty or full. They are sometimes masked by pleurisy, or by chronic pneumonia. Thus, in some patients the breathing is blowing, cavernous, and even amphoric, and may or may not be accompanied by gurgling; while bronchophony or pectoriloquy may be heard.

**Course—Duration—Termination.**—When bronchiectasis is not associated with tuberculosis, the general symptoms are benign for a long while. There is no fever; many patients retain their appetite, and continue to attend to their business, and in those who are not attacked by foetid bronchitis life is fairly comfortable, save that expectoration is abundant and dyspnoea may cause an increasing distress. In short, we are surprised, says Trousseau—and this is also Laënnec's opinion—by the apparent innocence of the disease up to almost its final stage.

In some cases death is the result of such complications as acute pneumonia, acute broncho-pneumonia, gangrene of the lung, hæmoptysis, cerebral abscess, purulent pleurisy, pneumothorax, tuberculosis, or pyæmia with articular and visceral abscesses. In other cases death comes more slowly, from the progress of the disease and from concomitant pulmonary troubles, which, after many years, are complicated by lesions of the right heart, or by consumption. The absorption of microbic products from the surface of the bronchial cavities produces a true septicæmia. The wasting, the hectic fever, the diarrhoea, the cachectic oedema, and the clubbed fingers, recall the picture of pulmonary phthisis.

**Bacteriology.**—The fluid from the cavities contains numerous microbes,



including the *Streptococcus pyogenes*, the *Pneumococcus*, and the *Staphylococcus aureus*, as well as saprogenic microbes, such as the *Bacillus pyogenes fœtidus*.

These microbes perhaps play some part in the destruction of the bronchial tissues which favour dilatation, but they certainly play an important one in most of the complications. These complications are not due solely to the toxins elaborated by the microbes, but also to the direct passage of certain microbes into the blood and the organs. The acute septicæmia which sometimes supervenes in bronchiectasis, the suppurative arthritis, the infarcts, and the **cerebral abscesses** are due to different streptococci (*Streptococcus pyogenes*, *Streptococcus septicus liquefaciens*) which have their original focus in bronchiectasis.

Infective endocarditis with abscess in the liver and the kidney has been seen. It was consecutive to infection by the *Staphylococcus aureus*, from an infected bronchial dilatation.

**Diagnosis.**—Pulmonary phthisis, fœtid bronchitis, and pleural vomica have many signs in common with bronchiectasis. Let us first distinguish bronchial dilatation from **tuberculous cavities** in the lung, and let us take a well-marked case, in which bronchiectasis is not associated with tuberculosis. The site of the lesion furnishes no distinctive sign, since bronchiectasis is as often unilateral as bilateral (twenty-six times in forty-three cases), and as frequent at the apex as at the base (Barth). Hæmoptysis, cough, and clubbed fingers are also seen in both diseases. Lastly, cavernous breathing, gurgling, and pectoriloquy show in both cases the existence of cavities, but give no information as to their nature. Upon what points, then, can an affirmative diagnosis be based? The expectoration in bronchiectasis is, it is true, more diffuent, more aerated and abundant than in phthisis; but this distinction is not sufficient, and the true distinction lies in the **different course** of the two diseases. The patient affected only with bronchiectasis has for five, eight, or ten years suffered neither fever nor marked wasting, but has simply coughed up abundant and sometimes fœtid sputum, with or without dyspnœa. Such is not the course of tuberculosis. Fever, wasting, dyspepsia, loss of strength, sweats, diarrhœa, are symptoms which coincide with the formation of cavities, and gradually increase with the spread of the lesion. Lastly, all doubts can be removed by examination of the sputum; the presence of bacilli therein being a sure sign of tuberculosis.

In **fœtid bronchitis**, which is characterized by sloughing of the mucosa in the bronchioles (Lasèque), we find fœtor of the breath and of the sputum, which is profuse, as in bronchiectasis; but **the signs of a cavity are wanting**.

Between dilatation of the bronchi and **vomica\*** of the pleura diagnosis is difficult. I am not referring to a vomica of the great pleural cavity occurring in purulent pleurisy, the evolution of which we have followed out. It is

\* Vide Chapter V., Section 16.

evident that in such a case the abundance of fluid, its sudden appearance by way of the bronchi, and the signs of a large cavity replacing those of effusion, leave no doubt as to the diagnosis. I allude to those interlobar varieties (pleuro-pulmonary cysts) which differ from effusions of the great pleural cavity, in that the symptoms are obscure and the fluid is scanty. A patient coughs up for some time 10 to 15 ounces of foetid muco-purulent fluid daily. On inspection, the thorax is found to be flattened in the sub-scapular region. At the same level impaired resonance exists. On auscultation, the signs of the cavity may be recognized. The condition is just like dilatation of the bronchi. How, then, can we diagnose between this condition and a vomica? We must investigate the **course** of events. Peter and I followed this course in a case similar to one described by Trousseau.\* The signs in bronchiectasis appear gradually, while the course of events is more sudden in interlobar pleurisy followed by vomica. The pleuritic phase is succeeded quite suddenly by the vomica, and the evacuated fluid is at first abundant, but diminishes during the next few days.

**Treatment.**—The remedies employed to dry up the secretion from the dilated bronchi are practically those used in chronic bronchitis. Terpene may be prescribed, 6 to 10 pills daily. Good results may be obtained with eucalyptol, given in doses of from 4 to 10 capsules daily. Turpentine, iodoform, and creosote will also find their indications. Inhalations and sprays of sulphurous and arsenical waters sometimes give good results. Revulsives, blisters, and the cautery must not be neglected. For the foetor of the breath and of the sputum, Paquelin's thermo-cautery should be employed. I have several times caused foetor to disappear for several days by multiple applications over the diseased region.

As medical treatment too often fails, it is recognized that resort must be made to surgical measures. I am not speaking solely of antiseptic injections into the diseased bronchi. I allude to pneumotomy. I am well aware that the published results are not very encouraging, and the pulmonary hæmorrhage which supervenes during the operation is a source of danger. Roswell Park, in his statistics, had nine deaths in twenty-five cases. These figures are not as yet very favourable to surgical attempts, but yet, when we think of the almost fatal prognosis of bronchiectasis, we confess that the hope of rational treatment lies in surgery.

## V. PSEUDO-MEMBRANOUS BRONCHITIS.

This disease may have various origins. Sometimes the false membrane contains Löffler's bacillus, and increases the gravity of the prognosis in diphtheria of the larynx or pharynx; sometimes the false membrane is

\* "Dilatation de Bronches" ("Leçons de Clinique Médicale," tome i., p. 588).



consecutive to pneumonia, especially massive pneumonia (Grancher), and the cause is the pneumococcus. Finally, in much rarer cases, we sometimes see after tuberculosis, measles (Jaeger), heart lesions, and ordinary bronchitis, cases of pseudo-membranous bronchitis which are difficult to classify.

Pseudo-membranous bronchitis does not always show a well-defined course. The bronchitis is only an episode of the disease which it happens to complicate. It is not so in the case of chronic pseudo-membranous bronchitis, in which the course and the symptoms are quite characteristic (Paul-Lucas Championnière).

### Chronic Pseudo-Membranous Bronchitis.

**Ætiology.**—This form is chiefly seen in adult and elderly persons. It is more frequent in men than in women. Pulmonary affection and arthritism in the patient's ancestors appear to predispose to it. It may follow ordinary bronchitis, or be a complication of pulmonary tuberculosis. It is sometimes associated with the *Aspergillus fumigatus*.

**Pathological Anatomy.**—Post mortem the bronchial mucosa is seen to be slightly red and thickened. The false membranes are found in the trachea, the large bronchi, but especially in the smaller bronchi. The false membranes are branched like a tree or a piece of coral and form exact casts of the bronchi. These casts may be coughed up in small fragments, or in branching pieces 4 or 5 inches in length. The substance which forms them is white or rosy, and is often disposed in concentric laminæ. In the large bronchi the membranes are hollow, and show a central lumen; in those of small calibre the casts are solid. The structure of these membranes varies. They may be composed of firm mucus, coagulated albumin (Grancher), fibrin (Caussade), or fat (Model). Charcot-Leyden crystals and eosinophile cells are at times found in them. In a case published by Claisse membranous bronchitis was associated with the streptococcus. In Griffon's case the pneumococcus was the pathogenic agent.

**Symptoms.**—The affection generally begins as acute bronchitis, which may be accompanied by the expectoration of false membranes, and then gradually becomes chronic. The membranes may not appear till long after the disappearance of the acute bronchitis. In some subjects the affection is chronic from the start.

The course is neither continuous nor progressive; it is characterized by **attacks**. At more or less lengthy intervals the patients are seized with intense dyspnoea, retrosternal pain, and convulsive cough. At first they bring up abundant shreddy matter, and then false membranes, which may take the form of isolated fragments, or be rolled up in balls which unfold in water. In some cases an entire bronchial tree is brought up. Hæmoptysis is very rare. After the expulsion of the membranes the dyspnoea ceases. During

the attacks the vesicular murmur is often diminished. We sometimes find a focus of crepitant râles which may be long persistent (Hyde Salter), or a *bruit de drapeau*, as in croup. The attack is generally apyretic.

At the Necker Hospital I saw, in 1891, a man, fifty years of age, with pseudo-membranous bronchitis. For twelve months membranes were absent; then he coughed them up every week for a month. The attack was preceded by certain symptoms the day before. He used to feel depressed, and would next day cough up large casts. He never had hæmoptysis, and I found no signs on percussion and auscultation. He improved rapidly under iodide of potash, although he was not syphilitic.

The duration of the disease is unlimited. A case of twenty-five years' duration has been quoted (Kirsch). The prognosis, not unlike that of simple chronic bronchitis, is only grave in a tubercular subject.

**Diagnosis.**—The diagnosis rests entirely upon the examination of the sputum. When false membranes are discovered, histological and especially bacteriological examinations must be made, in order to ascertain if it be a case of diphtheria, pneumonia, or of chronic bronchitis, with or without tuberculosis.

Rational treatment consists in the exhibition of iodide of potash, mercury, terpene, and creosote.

## VI. TRACHEO-BRONCHIAL SYPHILIS.

As Mauriac justly remarks, the trachea and bronchi are simple conductors of air, and play a purely passive part, which cannot be compared with the many functions of the larynx. The symptoms of tracheo-bronchial syphilis are therefore respiratory, but are more serious than in the larynx, because tracheotomy is not applicable when the lesion is situated in the trachea or at its bifurcation.

### 1. Secondary Troubles.

**Description.**—Erythema, catarrh, and erosions exist as secondary manifestations in the trachea and the large bronchi; but while vocal troubles and laryngoscopy readily reveal these secondary troubles if they are seated in the larynx, they can only be suspected when located in the trachea or the large bronchi. In certain cases, however, laryngoscopy may reveal them, although they are in the trachea.

The symptoms are those of ordinary tracheo-bronchitis—viz., cough, slight dyspnoea, and expectoration. Their syphilitic origin may in many cases be affirmed, because they often coincide with mucous or cutaneous syphilides, and because they improve rapidly under treatment.

Acute or subacute syphilitic tracheo-bronchitis is more common than we think. A case of tracheo-bronchitis which has lasted a long time, has been rebellious to other treatment, or has been set down to tobacco, arthritism, or chills, yields rapidly to mercury and iodide of potash.



## 2. Tertiary Troubles.

**Pathological Anatomy.**—In the trachea and the large bronchi, as elsewhere, the syphiloma may be circumscribed or diffuse. The lesion spreads either in the thickness of the mucosa or below it. Its action is not limited to the soft parts, and it invades the cartilages, the fibrous tissue, and the muscle. The gumma may be of large size, and the thickenings are more or less extensive.

These tertiary growths usually end in circumscribed ulceration in the case of gumma, and serpiginous ulceration, which has a phagedænic tendency, in the diffuse syphiloma.

The ulceration may occupy a segment or the whole circumference of the trachea; the phagedæna may attack the perichondrium and the cartilages, and perforate the trachea or furrow it from above downwards.

In some cases the perichondritis and the chondritis are **primary**, the walls of the trachea being indurated and converted into a rigid narrow tube.

**Tracheo-stenosis** and **broncho-stenosis** are the result of these lesions. The narrowing of the trachea may take place slowly or rapidly. The gummata and the ulcerations, with the resulting œdema, narrow the trachea and the bronchi. The fibrous tissue forms longitudinal, oblique, transverse, or annular bands, and causes most marked constrictions. The fibrous scars produce two kinds of deformity in the trachea and bronchi, which diminish both in length and in breadth. The trachea, which is normally about  $\frac{3}{4}$  inch in diameter, is reduced to a third or a quarter of its size. The constriction is rarely circular, like a diaphragm, but takes the form of an irregular, anfractuous channel, with superposed stages. The length of the trachea diminishes in proportion to the number of rings destroyed. The firmness of its framework is destroyed by the substitution of fibrous tissue for cartilage, and the consequent flattening during inspiration may cause asphyxia.

The tracheo-bronchial syphiloma is nearly always accompanied or preceded by pharyngeal or laryngeal syphilis. In sixty-five cases collected by Mauriac, in six only was the pharynx or larynx unaffected. The **lower fourth** of the trachea, with or without the participation of one or both bronchi, is the most frequent seat of syphiloma. The tracheo-bronchial glands are almost always enlarged.

**Description.**—The cough and slight dyspnœa of the onset make us think at first of simple bronchitis, but other symptoms soon appear; obstinate spasmodic cough, the sensation of a foreign body, the feeling of constriction in the neck, pain behind the sternum, noisy breathing, stridor, continuous and paroxysmal dyspnœa, indicate stenosis of the trachea. Respiration is often quiet when the patient is at rest, but movement brings on acute

dyspnoea ; attacks of oppression, due to reflex spasm of the glottis, supervene night and day, and cause extreme distress.

These **attacks of oppression** are one of the most salient characteristics of tracheal syphilis (Mauriac). I verified the truth of this assertion in a patient whom I saw with Dr. Poyet.

The expectoration is frothy, but sometimes the patient brings up pieces of gummatous tissue, and even fragments of cartilage. The larynx, which normally rises during deglutition, is often fixed by scar-tissue in tracheal syphilis. Laryngoscopic examination may show the tracheal lesion even as far down as the bifurcation.

When cure has not been obtained, or treatment has not been carried out in time, slow asphyxia, syncope, sudden death, perforation of the gullet, of the vessels, or of the mediastinum, with consecutive abscess, bronchopneumonia and gangrene of the lung, are possible terminations. We must not mistake laryngo-bronchial syphilis for ordinary bronchitis, asthma, or tracheo-bronchial adenitis, lest we see the patient succumb for want of active treatment.

Retrosternal pain, feeling of strangulation behind the sternum, tracheal stridor, immobility or lowering of the trachea during deglutition and preservation of the voice, together with the dyspnoëic troubles I have described, show that the trachea is stenosed.

Several lesions in the larynx, as well as paralysis of the posterior crico-arytenoid muscles, may cause similar dyspnoea ; but the stridor is laryngeal, the voice is generally affected, and the laryngoscope removes all doubts.

The trachea may be compressed by an aortic aneurysm, in which case the signs of aneurysm are found.

The trachea is sometimes depressed by tumours of the mediastinum, but other symptoms, such as dysphagia, œdema of the face and neck, deformity of the sterno-clavicular region, dullness, according to the extent of the tumour, and swelling of the supraclavicular glands, are usually present, and indicate the nature of the lesion.

If syphilis of the trachea is recognized, or **even suspected**, mercury and iodide of potassium must be given in large doses. I prefer injections of biniodide of mercury. This treatment must be carried out thoroughly. **Most active treatment** may cause no improvement for a fortnight.

## VII. WHOOPING-COUGH.

**Description.**—Whooping-cough is a contagious and epidemic disease, which is probably microbic, attacks the young especially, and confers almost absolute immunity. It is composed of a double element : the one inflammatory, catarrh of the bronchi ; the other nervous, the fit of coughing.

It is customary to describe three stages in whooping-cough, but we may



add the period of incubation, which extends from the moment of contagion to the outburst of the attack, and varies, according to Roger, from seven to ten days.

In the **first stage** the catarrh resembles simple bronchitis. The child has fever and cough, as in ordinary catarrh; but yet certain fine distinctions already indicate the specific nature of the disease: the cough is more obstinate and frequent, and the fever is more stubborn than in a simple cold. This stage lasts from three days to a fortnight, and passes insensibly into the stationary stage.

In this stage—that of **convulsive cough**—the fever falls back, and the nervous element takes the lead. The cough is less incessant than formerly, but the expiratory jerks succeed each other so rapidly that the patient makes at first eight to ten, then fifteen to twenty, expirations without being able to take breath. The **fit** is now established, and the crisis shows the symptoms so graphically described by Trousseau that I cannot do better than reproduce his picture:

A child is at play. Some moments before the advent of the crisis he stops; his mirth gives place to sadness. If he should be in the company of his playmates, he stands aside and tries to avoid them. He meditates his crisis, feels it coming, and experiences that pricking and tickling in the larynx which announces it. At first he tries to avert the paroxysm. Instead of breathing naturally and expanding his lungs to the full, as he was doing just before, he holds his breath, for it seems to him that the full current of air, by entering his larynx, will produce the exhausting cough of which he has had a sad experience; but, I repeat, whatever he may do, he will not prevent, and at most he will only be able to delay, the fit. The fit takes place. You at once see the patient look around for a support to which he may cling. If he is a child at the breast, he throws himself into the arms of his mother or of his nurse. If he is older and standing up, you notice him stamping, in a state of complete distress. If he is lying down, he sits up quickly and clutches hold of the bed-curtains or of the rails. The attack leaves him with a swollen face, and this swelling, which at times lasts for three weeks, may in some cases be quite enough to cause a practised physician to suspect the existence of whooping-cough.

Let us return to the fit. The fit commences with a sudden, noisy expiration, followed by a series of short, aphonic, convulsive, and more or less hurried expirations. At this moment a pause, which may last ten to fifteen seconds, occurs, and the chest remains motionless in the position of full expiration. During this phase of the fit the air expelled from the chest is not renewed; consequently, the patient, whose eyes are injected and streaming, lips cyanosed and face bloated, is in a state of distress bordering upon asphyxia. Then comes a **long, singing, convulsive inspiration**, which ends the fit, and brings a short spell of rest.

A second fit, however, occurs at once, and is followed by several others, which decrease in severity; the inspiration, instead of being singing, becomes aphonic, showing that the spasm of the glottis is less. The attack may then be said to be at an end. The series of fits constitutes the attack.

During the fits the child brings up shreds of glairy mucus, which stick to the mouth. The end of the attack is often marked by the vomiting of glairy material or of food-stuffs.

These attacks, which vary in duration from some seconds to ten or twelve minutes, are more frequent at night than in the day, and occur as often as sixty times in twenty-four hours. When this number is exceeded, the child's life is in danger (Trousseau). Between the attacks the patient has no cough to speak of; the fit sums up the whole disease. During the catarrhal period, bronchitic râles are heard in the chest. As soon as the spasmodic period begins they disappear.

In the **third stage** all the symptoms improve. The fits are less frequent, the inspiratory stridor is less marked, and the glairy mucus is replaced, especially in adults, by the thick sputum of bronchial catarrh.

Herff examined the larynx both in himself and in others, after anæsthetizing the part with cocaine. He has shown that during the whole disease the mucosa, especially in the interarytenoid and arytenoid regions, is inflamed.

During the attack mucus accumulates in this region. If this mucus is removed by a probe, the attack can be stopped; but if the same region is tickled, the fits can be reproduced. Touching other parts of the larynx does not produce the same effect. The reflex which provokes the fit originates, therefore, in the interarytenoid region.

**Course—Duration.**—Whooping-cough usually shows three stages. The convulsive cough very rarely appears at first, but is preceded by the catarrhal period.

In some patients the fits are replaced by attacks of sneezing (Roger), and I have seen two examples of this in children of asthmatic parents. The fit, says Trousseau, sometimes ends by sneezing. Whooping-cough has an average duration of six to eight weeks. Some patients, however, have fits of spasmodic cough for months, as though the malady had passed into a chronic state.

The **diagnosis** of whooping-cough is indicated, even in the catarrhal stage, by the obstinacy of the cough and the tenacity of the fever. In the stationary stage the fit and the whoop are quite characteristic.

Sometimes certain tumours of the mediastinum provoke a cough, termed "pertussoid" (Guéneau de Mussy), which somewhat resembles the true fit of whooping-cough.

It is important in diagnosis to recognize the different forms of **defaced\*** whooping-cough. I have already said that the fit may be replaced by attacks of sneezing.

\* The term "fruste" (defaced) was applied by Trousseau to cases of scarlatina, in which certain symptoms were absent. In archæology it refers to an inscription, part of which is missing.



At Chantilly I saw an elderly lady (her daughter had whooping-cough) who was seized with spasms of the glottis and inspiratory stridor which lasted some seconds, and recurred repeatedly in the form of attacks, day and night. In this defaced form the fit of whooping-cough was reduced to an inspiratory spasm of the glottis, and the other elements were quite wanting. Similar cases have been published—two by Trousseau and one by Blache.

**Prognosis — Complications.**—Whooping-cough *per se* is not a serious malady, and the gravity comes from the complications. Thus, the catarrh of the early period, which usually improves at the beginning of the spasmodic period, may invade the small bronchi. Fever then lights up, the dyspnoea becomes intense, and the tubular breathing of broncho-pneumonia or the fine râles of capillary bronchitis are heard in the chest. This serious complication, which may arise in all the stages, is chiefly seen in the stationary stage. The appearance of the inflammatory process often causes the nervous element to disappear—*spasmos febris accedens solvit*. “When in a child with whooping-cough you find that the fits, which numbered fifty or sixty in the twenty-four hours, cease suddenly, although the disease may be still in the stationary stage, beware, for you will find yourself face to face with an inflammatory complication” (Trousseau). In an adult these inflammatory complications may take the form of pleurisy or lobar pneumonia.

Whooping-cough predisposes to tubercular disease of the bronchial glands and to tuberculosis. In some cases we find meningitis or miliary tuberculosis; in others, it runs the course of chronic pulmonary tuberculosis.

The nervous element is connected with complications of another kind. **Spasm** of the glottis is frequent and very serious in children under four years of age. **Convulsions** may come on during the attacks or in the interval, and are very serious in infants. The fits cause a vesicular emphysema which in exceptional cases has become interlobular by rupture of air-cells in the lung. The repeated congestions excited by the fits induce **epistaxis**, **hæmoptysis**, bleeding from the ear (rupture of the membrana tympani), subconjunctival ecchymosis, and cerebral congestion, with attacks of **eclampsia**.

Furthermore, the vomiting of food is due to the fits of coughing, and leads to malnutrition if often repeated. The exaggerated contraction of the expiratory muscles causes involuntary evacuations and **herniæ**, and the rubbing of the tongue upon the incisor teeth causes ulceration of the frænum (Bouchut).

**Ætiology.**—Whooping-cough is an epidemic and contagious disease which chiefly affects children between the ages of two and seven years. The contagion is so virulent that an infant may be infected after being a few minutes in the company of a patient suffering from whooping-cough. In

the sporadic state it follows the usual course which we have described, but it assumes different characters in the epidemic form. Thus, in the epidemic of Dillingen, in 1811, patients were carried off by eclampsia; in the Geneva epidemic, in 1850, capillary bronchitis was the chief complication (Rilliet); in other epidemics the catarrhal stage was very short, and the spasmodic one appeared almost from the start (Trousseau). Epidemics of whooping-cough and of measles sometimes follow each other so closely as to indicate a causal relation between them.

**Bacteriology.**—Afanassief thought that he had discovered the bacillus of whooping-cough. This microbe—*Bacillus tussis convulsivæ*—is said to be small, slender, and disposed in groups or colonies.

Quite recently Bordet and Gengou claim to have obtained the specific organism of whooping-cough in pure cultures. It is a small bacterium of ovoid form, and presents the same appearance both in the sputum and in cultures. The presence of hæmoglobin is necessary for its growth. It is specially abundant in the exudate during the first fits.

The broncho-pneumonia, however, which occurs as a complication is usually due to the streptococcus, the staphylococcus, the pneumococcus, etc.

**Treatment.**—The indications must be directed to the inflammatory and the spasmodic elements. The former is relieved by emetics; for the latter we employ opium, aconite, belladonna, and bromide of potash. The use of alkalies has been advised (Labadie-Lagrave). Revulsives and blisters should be reserved for the inflammatory complications. Anæsthesia of the interarytenoid region, induced by means of cocaine, has given good results. Fumigation with sulphur has been extolled. The patient enters the fumigating chamber, the windows having been opened after the fumigation.

H. de Rothschild has obtained good results with chloroform. It should not be given in severe cases, where the patient is weakened by vomiting and insomnia. Acute bronchitis and broncho-pneumonia are, of course, contraindications. It may be given till anæsthesia is complete, or may be inhaled during the fits alone. For inhalation 10 to 20 drops of the following mixture are sprinkled on lint:

|                       |    |    |    |    |    |      |
|-----------------------|----|----|----|----|----|------|
| Anæsthetic chloroform | .. | .. | .. | .. | .. | 3iv. |
| Essence of pine       | .. | .. | .. | .. | .. | 3ii. |
| Essence of eucalyptus | .. | .. | .. | .. | .. | 3ii. |

This measure has been curative in some cases and has caused great improvement in others.

Hygiene plays a large part in the treatment of whooping-cough. The rooms should be as large as possible, and the child should not spend day and night in the same apartment. At the beginning of defervescence change of air is most useful in shortening the duration of the disease.



Prophylactic treatment is essential. The patient should be isolated from other children. The things he has used and the room he has occupied should be thoroughly disinfected.

### VIII. ASTHMA.

The attacks of dyspnoea which constitute asthma are quite characteristic ; they appear suddenly, weeks, months, or even years apart. They may exist as a **pure neurosis**, without any inflammation of the bronchi, but in most cases they are complicated by a **catarrhal element** of variable intensity. Catarrh does not usually accompany first attacks of asthma, but is associated with them later. Sometimes it plays a minor part, and appears towards the end of the attack ; at other times it begins with the attack as a true febrile catarrh ; and lastly, in other cases, it is so marked that the nervous element is of secondary moment.

These two elements, of which the nervous is constant and the catarrhal is variable, may finally give rise to chronic bronchitis and emphysema, and form a morbid cycle, which may be complicated by lesions of the right heart.

**Description.**—The attack of asthma generally begins in the early hours of the night, without prodromata. A patient who has gone to bed quite well wakes up suddenly with acute distress. He feels agonizing constriction in the chest, and his breathing is painful and wheezing. He gets up and opens the window to get air, putting every respiratory muscle into play. Hoping to make his breathing easier, he sits on his bed with his body bent, kneels upon a chair, with his head bent forward, or props himself up by leaning with his elbows on a piece of furniture. He strives hard, but the distress increases in spite of his efforts. Inspiration is imperfect ; expiration is slow, wheezing, and convulsive. His distress is extreme, and seeing the patient with face bloated and covered with sweat, with starting eyes and cyanosed lips, we might think that asphyxia was imminent. We should be wrong, for after this struggle has lasted for one or more hours the breathing becomes easier, the air enters the chest better, the expiration is less convulsive and not so prolonged, and the crisis subsides.

The end of the attack is sometimes indicated by a belching, by catarrhal expectoration, or the emission of dry, crepitant, or gelatinous sputum, which takes the shape of vermicelli. The first urine voided is clear and abundant (nervous urine), and sleep returns. Next day the patient feels tired, and has a disagreeable sensation of thoracic constriction, with ballooning of the belly, and some tendency to breathlessness.

An attack of asthma is rarely single ; we usually see a series of attacks, which are repeated for several days or weeks ; they often return at the same hour—usually at night. This series of fits forms an **attack** of asthma.

The attacks do not always have the intermittent form just described. In some asthmatics the distress continues throughout the whole attack, and the paroxysms reappear day and night, or several times in the twenty-four hours, allowing no rest.

Analysis of the attack of asthma shows that the dyspnoea presents peculiar characteristics. A respiration rate of 40 or 50 is not seen in asthma, as in broncho-pulmonary inflammations; the number of respirations is, on the contrary, less than normal. The painful and wheezing inspiration only allows an insufficient quantity of air to enter the chest, and the expiration, which is still more painful, and three or four times as long as the inspiration, does not drive out the inspired air. Let me add that, even at the height of suffocation, we do not see sucking-in, as in croup, because in asthma the chest is always filled to its **maximum** with air.

During the attack snoring rhonchi and mucous râles, which depend on the severity of the catarrh, are heard in the chest, or we may find total absence of the respiratory murmur in places. The chest is bulging and rounded, the diaphragm is depressed, and percussion gives the exaggerated resonance of acute emphysema.

In the variety of asthma above described the nervous element is almost alone in play; but when the catarrhal element is associated with it, the clinical picture is more or less modified. Some asthmatics during or towards the end of the attack suffer from fatiguing spasms of coughing, and bring up catarrhal sputum, which is also present between the attacks. The expectoration is chiefly composed of gelatinous sputum, which is vermiform, and resembles fragments of vermicelli, or consists of rounded pellets, crepitant to the touch, and known as Laënnec's perles. The sputum often contains Curschmann's spirals, octohedral crystals (Charcot-Leyden) and eosinophile cells (Müller), which, however, are not characteristic of asthma. In some asthmatics the fever, the cough, and the nature of the sputum prove that bronchitis is present; but the dyspnoea preserves its characteristic type, and appears in the form of paroxysms. In some persons, however, the bronchitis becomes chronic, emphysema appears, and from that time it becomes more difficult to estimate the nervous element.

In some persons the attack of asthma is preceded or replaced by fits of **sneezing**. These spasmodic fits may occur thirty or forty times in a few minutes, and reappear night and morning for several consecutive days. During the attack of sneezing the eyes, which are injected and weeping, often itch acutely; the face is congested, and the nose runs freely. Everything then returns to its normal state. This description would do well for **hay-fever**, as the reader will no doubt notice. In those affected with these sneezing fits it has been possible to predict asthma several months or years in advance.



**Pathological Anatomy.**—The theories which hold that asthma is consecutive to emphysema (Louis and Rostan), catarrh of the small bronchi (Beau), catarrh (Laënnec), or pulmonary congestion (Bretonneau), are erroneous, as in the main they mistake the effect for the cause. The attack of asthma is certainly produced by spasm of the muscles of respiration. A difficulty begins when we try to decide which muscles are affected. Some authors (Biermer and Williams) would place the spasm in the bronchial muscles; the contraction of the bronchi is said to prevent the circulation of the air and the lowering of the diaphragm to be consecutive to the exaggerated fullness of the lung. According to others (Sée and Wintrick), the bronchi have nothing to do with asthma, and the spasm pervades in turn all the extrinsic muscles of inspiration, including the diaphragm, intercostals, scalenis, trapezius, etc., which remain convulsed through the attack, keep the lung in a position of constant dilatation, and only permit a scanty renewal of the air in the pulmonary vesicles. A third opinion (Trousseau and Jaccoud) combines the two preceding ones, and admits spasm both of the extrinsic and of the intrinsic muscles of respiration.

I share the last opinion, and believe that the number and the species of muscles involved depend on the severity of the attack. In violent attacks both extrinsic and intrinsic muscles are involved; in slight cases the spasm may well be limited to the muscles of the bronchi, or only encroach on the diaphragm. In all cases the sphere of nervous excitation does not remain localized to the muscles of inspiration; it also attacks the muscles of expiration, and the rhythmic spasm of the **expiratory** muscles explains the length and the intensity of each expiration, which can only overcome part of the resistance of the inspiratory muscles. The renewal of the air in the lungs, therefore, is very incomplete, and dyspnoea is the result.

When we see the swelling and secretion of the nasal and ocular mucous membrane in some asthmatics, we are tempted to admit that like swelling and secretion exist in the mucosa of the bronchi, and help to narrow their calibre. This hypothesis is the more probable in that Stärk has seen swelling of the tracheal mucosa with the laryngoscope.

The spasm of the respiratory muscles and the phenomena of vasodilatation and vaso-secretion are themselves due to a special condition of the nerves which govern these functions. This state of irritability is sometimes spontaneous, or at least apparently so; at other times it is due to a reflex act which starts in the terminals of the sensory nerves which supply the bronchi or the nasal passages. Many theories now in vogue would, indeed, assign the chief part to the nasal mucosa. I shall speak of this later, under *Ætiology*.

**Course—Duration—Termination.**—First attacks of asthma are generally benign, and leave no sequelæ; later, if catarrh also is present, and more

especially if it predominate, the patient, even in the intervals, has a morbid condition, which is analogous to chronic bronchitis, and is the more tenacious because emphysema may also be present. The lesions of chronic bronchitis and of emphysema are especially seen in people who have at first suffered from asthma alone. Dilatation of the right heart and tricuspid insufficiency may finally be met with. Fortunately, the disease does not always follow the same course ; it may improve, without causing such complications, or may be cured by well-directed treatment. As a matter of fact, persons may have several attacks of asthma, which finally disappear.

**Ætiology.**—Asthma presents all the caprices of a neurosis ; it may be provoked or reawakened by the most insignificant causes, such as a smell (Floyer), the fumes of a sulphur match, the dust of oats (Trousseau), or powdered ipecacuanha (Cullen). A man who suffers from asthma in the North remains well when he resides in the South. A patient has asthma in Paris, but not in Versailles. I know a man who is subject to severe attacks in Egypt ; he recovers as soon as he is at sea. Some asthmatics cannot enjoy a hearty laugh without being seized with a slight attack. A high altitude and life in the mountains are often prejudicial to asthmatics. Asthma is a neurosis, and, further, it is almost always a **diathetic and hereditary neurosis**, as Trousseau has clearly shown. “Skin diseases, rheumatism, gout, hæmorrhoids, gravel, and migraine are affections which asthma may replace, and which, in their turn, may replace asthma. They are different expressions of the same diathesis.” A gouty father begets children ; while young they have migraine or piles, and later gravel, asthma, or gout.

The alternation of asthma and urticaria is a well-known fact, and asthma has been called “bronchial urticaria.” The alternation of asthma with such neuroses as epilepsy, hysteria, hypochondria, and mania has also been noted. Asthmatics may have relapsing or cyclical attacks of mania.

Asthma is more frequent in men than in women. It is a disease of all ages, and has been seen even in children, especially in the latter part of childhood. In children, asthma may behave as in adults, but it fairly often assumes the features of capillary bronchitis, suffocative catarrh, or broncho-pneumonia. The disease presents a grave appearance, and correct diagnosis is most important, because proper treatment gives excellent results. Trousseau and Sée report striking examples of this infantile asthma, which must be recognized, so as to avoid serious mistakes.

**Diagnosis.**—Hay-fever closely resembles asthma, and belongs to the same family.

Mediastinal tumours, enlargement of the bronchial glands, and aortic aneurysm produce attacks of dyspnoea, which must not be confounded with asthma.



A form of pulmonary tuberculosis simulates asthma, and is known as **pseudo-asthmatic**. I am not speaking of acute miliary tuberculosis, which Andral compared to asthma because of the continuous or paroxysmal dyspnœa, but I allude to ordinary chronic tuberculosis. Certain tubercular patients at the outset, or during the course of their malady, suffer from attacks of asthma, and the tuberculosis may be overlooked if we forget this pseudo-asthmatic form.

Diseases of the heart, especially mitral lesions, often provoke dyspnœa. In addition to this dyspnœa, the patient sometimes experiences **attacks of oppression**, which are more frequent at night. The attacks are as severe as those of asthma, and the dyspnœa has therefore been called "**cardiac asthma**." The term is wrong. Mitral disease does not produce true asthma, and the paroxysmal dyspnœa of cardiac disease has not the characters of the dyspnœa in asthma. In a cardiac case the breathing is rapid and panting; both inspiration and expiration are short and jerky; palpitation is frequent; the pulse is small; the face is pale; and the lips are cyanosed. Asthma presents quite a different picture. The breathing is not quickened, inspiration is slow and difficult, and expiration, which is wheezing and spasmodic, is three or four times as long as inspiration; palpitation is absent, and the pulse remains regular. In some cardiac cases attacks of dyspnœa are the chief symptom, and mitral disease may show itself at first solely by attacks of cardiac dyspnœa, which are mistaken for asthma, just as in patients with Bright's disease the renal lesion shows itself by attacks of dyspnœa, wrongly called asthma. In any case, cardiac asthma and true asthma must not be confused.

Lesions of the aorta are also accompanied by attacks of oppression, which bear some slight resemblance to those of asthma. These attacks of **aortic dyspnœa**, however, are more or less painful, like angina pectoris. In nowise have they the character of the true asthmatic attack, and the aortic lesion quickly decides the diagnosis.

From want of care the dyspnœa of **Bright's disease** may be taken for asthma, and many patients with Bright's disease are treated for asthma. This dyspnœa presents forms which are slightly different. In the first variety the patient is "short of breath," especially if he walks upstairs too quickly. The shortness of breath may be mistaken for asthma, but examination reveals pulmonary œdema, albumin in the urine, and other symptoms of Bright's disease.

In the second variety the threatening asphyxia is due to superacute œdema of the lung. The œdema rapidly invades the lung, the dyspnœa becomes acute, fine râles are heard all over the chest, and the patient coughs up frothy, rose-coloured, albuminous sputum, which is characteristic of superacute œdema.

In a third variety the dyspnœa is uræmic and toxic ; it may show the character of Cheyne-Stokes breathing. This dyspnœa may appear in the form of attacks, which patients mistake for asthma. The attack comes on night and day, rapidly becomes acute, and reappears at the slightest movement. It may recur several times in the twenty-four hours, or disappear and return after more or less lengthy intervals.

Dyspnœa comes on at all periods of Bright's disease, but we must never forget that it may be the first symptom. In some cases it appears suddenly, like an attack of asthma ; in others it establishes itself gradually, like a chronic bronchitis. A knowledge of these facts will prevent our sending patients to Mont-Doré or to Cauterets for bronchial catarrh, asthma, or emphysema, when they are really suffering from Bright's disease.

Much attention has lately been paid to asthma following nasal lesions. Voltolini of Breslau in 1871 published cases of asthma in patients with nasal polypi. Removal of the polypi cured the attacks. Hack thinks that the pathological reflex has its origin in inflammation of the erectile tissue of the nose, and since then the nose has been accused (polypi, hypertrophy of the mucosa, deviation of the septum) of causing most cases of asthma, and also dyspnœa, migraine, cough, œdema, incontinence of urine, etc. The result was attempts to destroy the cause of so many ills by cutting and burning the nasal fossæ.

These statements are much exaggerated. It is quite true that the nasal mucosa plays a large part in asthma. Attacks sometimes appear after certain nasal stimuli. Sneezing, nasal secretion, and swelling of the mucosa, are present in true asthma, in hay-fever, and in asthma following nasal polypi. It is therefore necessary to pay attention to the undoubted part which the nasal mucosa plays in the production of asthmatic attacks. It may occasionally be necessary to deal with this mucosa, but this is only one side of the question, and if in some cases the nasal mucosa, by its special excitability, be the origin of the reflex which provokes the asthmatic attack, this special excitability is often present elsewhere (bronchi, lungs, or centres in the medulla).

**Treatment.**—We may first consider the treatment of the attack. The attack can be checked or much modified by prompt action. Inhalation of the fumes of *Datura stramonium*, nitrate paper, Espic cigarettes, hypodermic injections of morphia, inhalations of pyridine, and bromide of potash, are of service. Nitrite of amyl "is as dangerous as it is difficult to handle" (Sée).

I constantly prescribe stramonium, smoked in a pipe. The dry leaves should be finely cut up and some small pieces of nitrate paper added.

Pyridine may be used, either by inhalation of 10 or 12 drops on a handkerchief, or by allowing the drug to evaporate slowly near the patient.



These remedies may be repeated two or three times a day, and when employed for some time and between the attacks may cause marked improvement. Iodide of potash, however, is the best drug for asthma, both in the attack and in the intervals (Trousseau). The dose depends on the tolerance of the patient. We may begin with 10 grains daily, and increase to 30 grains, or even more (Sée, Jaccoud). The drug should be continued for a long while, care being taken to suspend its use at intervals. With iodide of potash Trousseau employed tincture of lobelia in small doses.

He also advises the use of belladonna and bromide of potassium, replaced, as the case may be, by preparations of arsenic.

I usually advise the following régime between the attacks. During the first and third week of each month I prescribe 10 to 15 grains of iodide of potash daily, with 10 drops of tincture of lobelia.

In the second and fourth weeks of the month I prescribe bromide of potash, 15 to 30 grains a day, and pills of extract of belladonna. This treatment should be continued for a very long while.

Between the attacks iodide of codeine has been extolled. It is given in syrup (Labadie-Lagrave and Rollin), and the dose is  $\frac{1}{6}$  grain.

The cure at Mont-Doré gives very good results (Tardieu).

The local treatment in the attack consists in painting the nasal mucosa with a 5 per cent. solution of cocaine. The nasal fossæ must always be carefully examined, in order to remove polypi, or cauterize the mucous membrane, if necessary.

## IX. SUMMER ASTHMA—HAY-FEVER.

This disease which in some respects resembles true asthma has been called **hay-fever**, summer asthma, or spasmodic rhino-bronchitis.

The term "hay-fever" is not good, for fever is usually absent, and the disease occurs when there is no hay. The term "summer asthma" is preferable; but as the disease is equally prevalent in the autumn, the name "annual asthma" is better still.

This disease, which is most common in the Anglo-Saxon race, is not rare, and I have seen a good many cases.

**Description.**—Annual asthma assumes two chief forms: the one is called **oculo-nasal**, the other **oculo-naso-thoracic**.

The disease appears usually about the 15th or 20th of May, and almost at a fixed date. A person who is not susceptible at other seasons is seized with a kind of "cold in the head." Sneezing, obstruction of the nose, and supra-orbital headache are present. The trouble is at first thought to be a cold.

The eyes, however, become the seat of pricking and intolerable itching, chiefly at the inner canthus. The patient rubs his eyelids vigorously. The

eyes are red, swollen, and weeping; the conjunctiva is cedematous; the tears are so abundant that they hamper vision, and flow over the cheeks; the photophobia is intense, so that the patient avoids daylight.

These troubles generally disappear or improve towards evening. In the **nose** we find the following symptoms: unbearable itching; violent sneezing, repeated ten, twenty, or thirty times in succession; profuse serous discharge from the nose, which runs "like a fountain."

The nasal, like the ocular, symptoms are brought on again or made worse by sunlight and heat; they become less severe under the influence of shade and cold.

Such is the **oculo-nasal** form of the disease. It persists for weeks, with alternating improvement and aggravation. The symptoms then improve, and recovery is complete till the following year. In the other variety, called **oculo-naso-thoracic**, in addition to the above symptoms, we find dyspnoea, like that of asthma. The dyspnoea begins about a fortnight after the ocular and nasal symptoms; it becomes gradually worse, is marked by the occurrence of fits, and is complicated by catarrh, in which the patient coughs and brings up bronchitic sputum. Improvement now comes on, and recovery is complete in six weeks, till the next year.

Patients have usually one annual attack. The prognosis is good, for this variety of asthma does not lead to chronic bronchitis or cardiac dilatation.

In my opinion, annual asthma should be included in the family of true asthma. It forms part of the gouty diathesis, and is often hereditary. It may recur annually for a great number of years.

This asthma is nothing but the spasmodic exaggeration of the defensive reflexes of the respiratory mucosa, which include sneezing, cough, and serous exudation from the mucous membrane. Its starting-point seems to be irritation of a submucous nerve filament belonging to the ethmoidal branch of the ophthalmic nerve, which is particularly sensitive in the angle formed by the nasal septum and the lateral wall at its upper part. Touching this point may provoke cough alone, sneezing and lachrymation, or the complete crisis. This point must be burnt with the galvano-cautery, without troubling about the malformations or the various lesions of the nasal fossæ. Partial or total relief is thus obtained. P. Bonnier, under the name of "**rhino-laryngitis sicca**," has described an inverse form of hay-fever, which appears under identical conditions, but is characterized by painful dryness of the respiratory mucous membrane, producing aphonia.

The various remedies employed are less efficacious in this variety than in true asthma. A cool, shady room is a good measure.



## CHAPTER IV

### DISEASES OF THE LUNG

#### I. GENERAL SURVEY OF THE ANATOMY OF THE LUNG

THE lung is made up of lobules of a polyhedral form, set one against the other, and separated by connective tissue. These lobules are well seen in the new-born, because at this age the network of connective tissue which surrounds them is much thickened. Later the delimitation is less apparent, because the connective envelope loses its thickness. The delimitation of the lobules, however, is readily seen on the surface of the organ, where the connective sheaths are infiltrated with dark material, and form a kind of mosaic.

The **pulmonary lobule** represents the structure of the whole lung. It is a small and spongy polyhedral or conical mass, of about 1 c.c. in size, and joined to the rest of the organ by a short **pedicle**. This pedicle contains a bronchiole, which is given off at right angles from a large bronchial twig, and a pulmonary arteriole and vein, with lymphatics and nerves, the whole being ensheathed by connective tissue. The bronchus and the artery penetrate the interior of the lobule at the hilum, but the vein ramifies over the lobule, following the perilobular connective sheath.

A transverse section of the lobule therefore presents two connective-tissue regions—a central one (the intralobular space, which contains the bronchus and the artery), and a peripheral one (the perilobular space), containing the vein.

When the bronchus enters the lobule, it takes the name of **intralobular**, and parts company with the vein, but is accompanied by the artery as far as its farthest ramifications. The intralobular bronchus traverses the lobule without diminution in size; it forms the axis of the lobule, and in its passage gives off alternate branches, and ends by dividing dichotomously. Each branch derived from the intralobular bronchus passes into an acinus, and takes the name of “**acinous bronchus**.” What is the acinus?

The lobule is formed by a number of acini, and each acinus, measuring 2 to 3 millimetres in each direction, forms within its bronchus a small system, which has the following arrangement:

On reaching the acinus, the branch, after a short course, spreads out like a funnel, and forms a kind of vestibule, whence arise three, four, or five **alveolar ducts**, which widen out to form **infundibula**. The infundibulum may be considered as the expanded end of the alveolar duct. All these parts, except the acinous bronchus, are lined by the alveoli.

The alveoli are air-cells in which hæmatisation goes on. They resemble shallow cups in the walls,\* and in the dried section of an acinus appear as oval or rounded cavities, separated by partitions, like a beehive. The interalveolar septa form the skeleton of the acinus, and, like it, are composed of a connective membrane and elastic fibres.

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\* Frey, “*Traité d’Histologie*,” p. 543. The opening of the alveolus into the infundibulum is narrower than the fundus of the alveolus.

This fibro-elastic skeleton permits the extension and the retraction of the alveoli, supports the capillary network, and "gives a foundation to the pavement epithelium of the lung" (Ranvier).

The capillary network of the lobule arises from the artery, and projects into the interior of the alveoli, when they are not too dilated.

Lymphatic vessels are everywhere present, and surround the alveoli, infundibula, acini, and lobules (Grancher).

Each disease of the lung alters the infundibulum, the acinus, and the lobule, in a particular way. In lobar pneumonia the lobule is filled with an exudate rich in fibrin, and the projection of the acini on the surface of the section explains the granular appearance of red hepatization. In disseminated or confluent broncho-pneumonia the inflammation spreads from lobule to lobule. The lobular bronchus is primarily invaded. Around the bronchus we find the peribronchial inflammatory nodule or zone of hepatization, and farther out the zone of splenization. The exudate is poor in fibrin, but rich in pus cells. In emphysema the lobule is distended to its maximum, and the alveolar septa are often atrophied or perforated. In pulmonary fibrosis the dense and fibroid interstitial tissue partly blocks the alveoli and the acini. In certain occupation pneumonias (miners, founders) the walls of the alveolus, the connective tissue, and the lymphatics are loaded with coloured particles (anthracosis). In tuberculosis the walls of the alveoli, of the small vessels, and of the bronchial ramifications contribute to the formation of the tubercles.

## II. PULMONARY CONGESTION.

**Congestion of the lungs** may be divided into two chief classes—**active**, produced by afflux of blood; **passive**, resulting from blood-stasis.

### 1. Active Congestion.

Active congestion of the lung is caused by inhalations of irritant vapours; by the sudden passage from a warm to a cold atmosphere, or *vice versa*; by pathological neoplasms, especially tubercle. It accompanies the eruptive fevers—principally typhoid fever—in which it often makes the prognosis worse. It may be due to malaria, gout, and rheumatism, and is provoked by the suppression of normal or accidental hæmorrhage (piles, menstruation). Some forms have a nervous origin (hysteria), arise from reflex action (large burns), and accompany cerebral lesions (hæmorrhage and softening).

The symptoms are in harmony with the severity and the extent of the hyperæmia. As a general rule, pulmonary congestion, if pushed to its extreme limit, may terminate in hæmorrhage, with or without hæmoptysis, and, indeed, cases of rapid death have been quoted (Devergie). Dyspnœa, cough, pain in the side, may be seen in the congestion of malarial, gouty, or rheumatic origin.

In articular rheumatism this complication arises suddenly: the expectoration is streaked with blood, the breathing is uneasy. On auscultation all the signs of broncho-pulmonary congestion, with œdema, are found, and death may occur in a few hours.



## 2. Passive Congestion.

Passive congestion is due to blood-stasis, caused by cardiac diseases (mitral and tricuspid lesions), degeneration of the cardiac muscle, and prolonged decubitus (hypostasis).

Passive congestion is slow in its course, and is always accompanied by œdema of the lung.

The capillaries allow the transudation of a highly coloured fluid, and pigmentation of the alveolar walls and of the epithelial cells is the result. Chronic hyperæmia often goes on to **splenization**—a condition in which the tissue of the lung is firm and reddish, like the pulp of the spleen. This morbid state has been called **hypostatic** pneumonia, although there is no pneumonia in the true sense of the word. The exudate is poor in fibrin and cellular elements. At the seat of the lesion percussion shows more or less complete dullness, and on auscultation the breathing is almost tubular.

**Treatment.**—Bleeding and cupping should be reserved for acute congestion. The treatment of passive congestion forms a part of the treatment of the cardiac disease. When the congestion is hypostatic, the position of the patient should frequently be changed.

## III. INFLAMMATION OF THE CHEST.

**Discussion.**—When I began to study medicine at Toulouse, my first teachers, former pupils of the Paris and Montpellier schools, and, consequently, rather eclectic than absolute, taught us that, besides pulmonary congestion and genuine inflammations of the broncho-pulmonary apparatus, there exist bastard phlegmasiæ, called **inflammations of the chest**, wherein hyperæmia and inflammation exist in irregular combinations.

The term “inflammation of the chest” has to-day almost ceased to exist. Treatises on pathology, the publications and the theses of our faculty, are for the most part silent on the subject. We speak of pulmonary congestion, pneumonia, and broncho-pneumonia, but *not* of “inflammation of the chest,” which seems to have lost all its claims as a distinct morbid condition.

I do not hold this view. I believe that inflammation of the chest should have a place in our nosology. Besides the genuine inflammations of the respiratory apparatus, such as pneumonia, broncho-pneumonia, pleurisy, and bronchitis, we find other bastard morbid states, in which the **hyperæmic and inflammatory elements are differently combined**, and which, by tacit accord, have been called “**fluxions**.”

It is, moreover, remarkable that, while the phlegmasiæ fix voluntarily upon certain parts of the respiratory apparatus so that they might be called “**systematic**,” an epithet employed in diseases of the spinal cord (Vulpian),

the fluxions, on the contrary, are naturally diffuse and multiple. Inflammation (fluxion) is rarely localized to the lung or to the pleura without affecting other parts of the respiratory apparatus. It may touch or strike the lung, the bronchi, the pleura, and the muscular layers of the thorax—in a word, **all the superimposed layers of the chest**, and hence custom termed it **“inflammation of the chest.”**

Cruveilhier was pertinent in describing pleurodynia, accompanied by fever and pleural friction sounds, and Peter, in his “Clinical Lectures” tells us: “Inflammation, if it be intense, may not remain limited to the muscles, but may invade **all the superposed planes of the thorax**, including even the pleura.” A step farther, and Peter would have come to inflammation of the chest.

**Description.**—Inflammation of the chest is a morbid state of variable **intensity and nature**. It is neither abortive pneumonia nor broncho-pneumonia, but another entity. Sometimes it spreads its action over all the parts of the respiratory apparatus; at other times it leaves some parts almost untouched, and concentrates its action on the lung, the bronchi, or the pleura.

As examples—

An individual is seized with a sharp pain in the side; he has slight rigors, cough, and fever. On examination, we find **pleurodynia**, with or without **cutaneous hyperæsthesia**. The pains affect not only the intercostals, but all the muscles of the part. The abdominal and lumbar muscles may also be involved. Friction sounds over the painful area, and bronchitic râles, scattered over both sides of the chest, are heard. In this case the skin, the muscles, the pleura, and the bronchi have been affected. The pleurisy is in the incomplete stage, and no effusion appears. The bronchitis is but slight, and the patient will be well in a few days.

In another person the inflammation involves the bronchi and lung to a slight degree, but concentrates its action on the pleura, and effusion is found. To this category belong certain bastard pleurisies, obscured by the inflammation of the lung and of the bronchi.

In another case the clinical picture presents some difference.

The illness has commenced with fever; the musculature of the thorax may or **may** not be painful. On auscultation we find fairly severe bronchitis and pleuritic rubs, and at one part of the chest we recognize dullness and blowing breathing, with bronchophony, which are evidence of marked pulmonary congestion. From the onset the sputum is streaked with blood, the dyspnoea is fairly acute, and the temperature 102° F. or more. What name is to be given to this disease? It is not pleurisy, for the pleural lesion is but incomplete. It is not bronchitis, although the bronchi have been attacked. It is not pneumonia in the true sense of the word. It is a case of inflammation of the chest which has concentrated its action upon the lung.

Lastly, the pulmonary localization is yet more marked or extensive in some cases. The temperature reaches 104° F., the cough is painful, and the dyspnoea acute. Percussion reveals dullness, which shows the extent of the lesion. We might be tempted to call the disease pneumonia, and yet analysis of the signs and the symptoms proves that it is **not a true case**. The râle is neither so fine nor so dry as in pneumonia; the



tubular breathing is **not** so intense ; the bronchophony is not so marked ; the sputum, instead of being rusty, is rather streaked with blood. Bronchitic râles or friction sounds are scattered through the chest ; defervescence is not sudden ; and, although the inflammation may reach its maximum and be in excess of the hyperæmia, still, the disease is not true pneumonia.

However, I must say that these forms are on the road to become lobar pneumonia. Pneumonia does not always present the classical type given for purposes of description. It often conforms to inflammation of the chest, and the barrier between the two is not insurmountable. Grasset regards inflammation of the chest as an **attenuated pneumococcal infection**.

The **prognosis** varies according to the variety and intensity of the disease. Its **ætiological** conditions are diverse. In some cases it forms the whole disease, and follows upon a chill (Woillez, Bourgeois) ; in others it is secondary to some general condition, such as **influenza** and catarrhal fever.

Revulsives (dry-cupping, blisters), local bleeding (leeches, wet-cupping), emetics, quinine, and alcoholic drinks, should be employed, according to the nature and the severity of the malady.

#### IV. ACUTE LOBAR OR FIBRINOUS PNEUMONIA—PNEUMOCOCCIC INFECTION—PNEUMOCOCCIA.

Pneumonia is called **lobar**, when it invades a lobe, or part of a lobe, without healthy tissue intervening, in contradistinction to **lobular** pneumonia, which causes isolated or confluent nodules. It merits the name **fibrinous** because, of all the inflammations of the lung, it is the richest in fibrin ; the fibrinous exudate fills both the alveoli and the bronchioles. The alveolar walls and the connective tissue are almost completely unaffected by the process, and the parenchyma of the lung completely recovers its integrity after pneumonia.

I shall first describe the disease as localized in the lung—that is, pneumonia proper—and then review its extra-pneumonic localizations, such as pleurisy, pericarditis, endocarditis, peritonitis, arthritis, gastritis, meningitis, nephritis, otitis, etc., finally discussing the different clinical types which the pneumonic infection may assume.

**Pathological Anatomy.**—It is customary to describe three stages in pneumonia : **engorgement**, **red** and **grey hepatization**. The third stage is somewhat rare.

**Engorgement** is characterized by intense congestion. The congested region, which is violet-coloured and increased in volume, crepitates a little, and pits on pressure ; it is oedematous and infiltrated with a reddish serum, which flows out on section. The capillaries are distended with blood, and allow plasma, red corpuscles, and leucocytes to pass out. The cells of the

pulmonary epithelium become vesicular; some fall into the interior of the alveolus, where they are enclosed by a fine network of fibrin. The stage of engorgement lasts from twenty-four to forty-eight hours.

In the second stage, called **red hepatization**,\* the lung is converted into a kind of solid block, which is red, mottled, and **homogeneous** throughout the whole extent of the lesion. The hepatized lung is heavy, and sinks in water; it has become friable, and is easily torn or crushed by the finger. The cut section is practically dry, and studded with granulations. These granulations are about 1 millimetre in size, and are due to the fact that the infundibula are distended and moulded by the coagulated fibrin. The diseased lung is larger and heavier than the healthy one; it may weigh as much as thrice its normal weight. The hepatized tissue, after washing, becomes of a yellowish-grey colour, from the dissolution of the red corpuscles. Under the microscope the vessels are dilated and engorged with blood; the alveoli are filled with a fibrinous network, which enmeshes epithelial cells and red and white corpuscles.† The alveolar arches and the epithelium are unaffected. Cells and fibrin are found in the small and sometimes in the large bronchi (fibrinous bronchitis). The pneumococcus is found in the granular cells and the fibrinous reticulum. After three to five days, the red hepatization gives place to **resolution**: the fibrin in the alveoli becomes granular, the pus cells grow large, and the liquefied and altered elements are reabsorbed *in situ* by the veins and lymphatics, or expelled in the sputum. In this way recovery results.

When, however, pneumonia, instead of terminating by resolution, goes on to grey hepatization, the lung takes a greyish tint. Its cut section is granular; the fluid which exudes is purulent; the tissue becomes very friable, and finger-pressure produces a tear filled with pus. Under the microscope the alveoli appear full of pus corpuscles. Microscopic abscesses are frequently met with. The parenchyma, however, is seldom destroyed, and the pus is not, as a rule, collected so as to form an abscess.‡

Grey hepatization is not always a sign of pus (Rindfleisch). The grey colour of the lung tissue may indicate ordinary resolution. In this case the colour is brownish or yellowish-grey, and the tissue is fairly firm and granular, like that of red hepatization. The grey appearance is due to the small number of red corpuscles, the disappearance of the hæmoglobin, and the abundance of migratory cells, which absorb the exudate.

\* This stage was called **red softening** by Andral.

† This exudate is very rich in granules of glycogen (Loeper, *Arch. de Méd. Expérimentale*, September, 1902).

‡ The formation of abscesses is a very rare termination in pneumonia. In thirty-six cases collected by Grisolles ("Traité de la Pneumonie"), the abscess was situated twelve times in the upper lobe, nine times in the lower one, twice in the middle, and four times in several lobes at once.



Pneumonia always induces **lymphangitis**. The lymphatic vessels of the inflamed region, like the pulmonary alveoli, contain fibrin, endothelial cells, and red and white corpuscles.\* The corresponding lymphatic glands also participate in the process. Lobar pneumonia is found more often on the right than on the left side, in the proportion of 3 to 2 (Lebert). It is unilateral, or double, in the ratio of 8 to 1 (Grisolle); it affects the lower much more frequently than the other lobes. The blood is very rich in fibrin,† and contains two or three times the normal amount.

**Bacteriology.**—Pneumonic infection is due to the **pneumococcus**. This organism was first isolated from the lung by Talamon (1883), and was thoroughly studied by Fraenkel. Netter has demonstrated its intervention in the complications of pneumonia. In health it is found in the saliva, where it was discovered by Pasteur; and where Netter has found it to be pathogenic in one-fifth of normal persons, and Bezançon and Griffon have shown that it exists as a constant saprophyte on the surface of the tonsils.

The pneumococcus resembles a candle-flame in shape. The organisms usually face each other by their tapering extremities, sometimes, however, by their other ends.

In the hepatized lung the pneumococcus is present in pairs—*i.e.*, as diplococci. In grey hepatization and in the pus from the complications it often forms short chains (Griffon).

The pneumococcus stains readily with the aniline dyes; the microbe stains by Gram's method. Staining with carbolic methylene blue shows a capsule which surrounds the elements. This microbe develops between 75° and 108°, but preferably at 98·5° F. On agar or on gelatinized serum it forms transparent colonies, like little drops of dew. The best culture medium is coagulated rabbit serum, and the best preservative medium is blood rendered uncoagulable, or blood-agar, in which the pneumococcus retains its vitality for several months (Bezançon and Griffon).

The pneumococcus causes pathological results which differ according to its virulence and to the species inoculated. In the mouse little reaction is seen at the point of inoculation, but generalized infection occurs. In the rabbit the lesions differ according to the virulence of the injection—local and fibrinous if it is attenuated, general and hæmorrhagic if it is virulent (Bezançon and Griffon, Fournier and Carnot). In the sheep and the dog, which are more refractory, the local reaction is very intense, and inoculation of the lung produces hepatization. The blood contains a few microbes.

\* "It is impossible to affirm that a vacuole filled with pneumonic exudate is a transverse section of a lymphatic canal or of an alveolus" (Cornil and Ranvier, *loc. cit.*, p. 696).

† This **hyperinosis** of the blood is explained by the fact that the blood is charged with fibrinogenous material from the inflamed organ.

In man the germ is found in all the pneumonic products: pneumonic secretion; hepatized lung tissue; fibrinous exudate in the bronchi; glands of the hilum; fibrinous inflammations of the pleura, the pericardium and the meninges; vegetations of the endocardium, kidney, joints, and parotid glands.

The pneumococcus may be obtained during the course of the affection by withdrawing from the lung a drop of exudate by means of aseptic puncture, or by isolating it from the sputum, or from the mucus in the throat.

The pneumococcus may be found in the blood-stream; but this infection of the blood does not imply a fatal ending, though it almost always coincides with grave pneumonia.

To discover the pneumococcus in the rusty sputum or in the saliva, examination of stained films is not trustworthy, unless the preparation literally swarms with encapsuled diplococci. Inoculation of mice is the best and most convenient method. These animals are so susceptible to the virus that death supervenes from general infection twenty-four to forty-eight hours after inoculation with the sputum, and post-mortem the pneumococcus is found in the blood and the organs.

The toxine secreted by the pneumococcus has been studied by Klemperer, and quite recently by Fournier and Carnot.

**Agglutinative Reaction.**—Pneumococcal infection causes an agglutinative reaction which is as follows:

A little serum is obtained from the blood of a healthy individual or from a person suffering from some other disease (rheumatism, typhoid fever, tuberculosis, etc.). This serum is inoculated with a minute quantity of a culture of pneumococcus, and placed in the oven at 98° F. Next day this culture is as clean as though the serum had not been inoculated. Neither dust nor dots are visible, and if the tube is well shaken, the culture remains quite limpid.

On the other hand, if a culture of pneumococcus is sown in the serum of patients suffering from pneumonic infection (pneumonia, pleurisy, peritonitis of pneumococcal origin, etc.), the culture, placed in the oven at 98° F., presents a characteristic appearance by the next day. Sometimes a dome-like false membrane is present at the bottom of the tube; at other times we perceive several pseudo-membranous fragments. The culture in other cases contains dots resembling a coarse dust, which, on shaking, make the liquid turbid and then fall to the bottom of the tube. Without the aid of the microscope, by the comparative study of tube cultures, we can say whether pneumococcal infection is present or not.

Under the microscope, if we examine a culture of pneumococci on healthy serum, or on serum from those with some other malady (rheumatism, tuberculosis, typhoid fever), we see that the pneumococci are isolated, and



remain so, without any tendency to form chains or groups. No agglutination occurs.

On the contrary, if a culture of pneumococci on serum from patients with pneumococcal infection is placed under the microscope, we see that the microbes unite in long wavy chains, circumscribing clear spaces, in which, as a rule, free pneumococci are not present, and we can no longer distinguish the capsules of the pneumococcus.

In some cases, especially in the experimental infection of the rabbit, the collections of pneumococci form masses.

In human infection the formation of a true mass is exceptional at the outset of pneumonia, and wavy chains, separated by empty spaces, are more commonly seen. The chain therefore appears to be the first degree of agglutination, which later produces massing of the elements. Towards the end of the second stage the reaction becomes more intense. The pneumococcal sero-reaction gives equally positive results in the primary extrapulmonary localizations of the pneumococcus.

**Description.**—In 25 per cent. of the cases lobar pneumonia is preceded by prodromata, which include lassitude, pains in the limbs, headache, epistaxis, tracheitis, insomnia, and fever; they last possibly one or two days. More often pneumonia begins suddenly with a single rigor, which is as prolonged as that of an intermittent fever, and is accompanied by a rise of temperature in the axilla to 103° F. This invasion is sometimes accompanied by vomiting.

By the end of the first day, or at the commencement of the second, **dyspnœa, cough, and pain** appear. The patient complains of a **pain in the side**, at the level of the nipple. This pain is increased by the respiratory movements and fits of coughing. In some cases the pain is abdominal, or supraspinous, or may even be found on the healthy side. Distress appears from the first. The jerky and painful **cough** is at first dry, but by the second day, or in the course of the third, the patient brings up sputum, which is tinged with blood, **amber-coloured or rusty, aerated, and viscid**. It sticks to the sputum-cup, and is pathognomonic of acute lobar pneumonia. The sputum, which is at first amber-coloured, like barley-sugar or apricot marmalade, and then rusty, may on the following days become of a brick-red colour. It represents the pneumonic exudate, which comprises white and red corpuscles, epithelial cells, surrounded by fibrin and some fibrinous filaments from the small bronchi. Pneumonic sputum is rich in mucus and chlorides. The mucus renders it transparent and gelatinous. The pneumococcus is found in abundance.

Pneumonia does not cause anæmia, and the red blood-corpuscles are little diminished in number. The blood-count shows a leucocytosis varying from 20,000 to 25,000, with considerable increase (85 per cent.) in the poly-

nuclear cells. Insufficiency, as well as excess of leucocytosis, may mean a bad prognosis.

Percussion of the invaded area sometimes yields dullness; at other times, according to Jaccoud, a transient tympanitic sound; and auscultation reveals the driest and finest of all râles—*i.e.*, the **crepitant râle**—which occurs in puffs towards the end of inspiration. It is not heard during expiration, and is often perceptible only after making the patient cough. The crepitant râle must at times be looked for in the axillary region.

During the following days (**red hepatization**) the functional symptoms increase, and though the pain in the side improves, the dyspnœa reaches such a degree that the respiration rate may rise to 40. The pulse, which is full, varies between 100 and 110 beats, and the temperature from 102° to 104° F., with a slight morning remission. At this time the aspect of the patient is characteristic—cheeks burning, face injected, eyes bright, tongue dry and coated, and voice short. The hurried working of the nostrils indicates the acuteness of the dyspnœa. The urine is scanty and high-coloured; it is rich in urea and uric acid, but very poor in chlorides, which appear to be absorbed for the benefit of the exudate. **Delirium**, which sometimes appears at this stage, is quiet, but may be violent in drunkards.

In the hepatized region the **dullness** is complete, and the vocal fremitus is increased, and, as the voice and the respiration are so well conducted by the solidified tissues, the voice is **loud, but not articulate** to the listener's ear. This condition is called **bronchophony**, and the breathing takes on the rough, blowing tone known as **tubular**. Further, as all the inflamed parts do not undergo their different changes at the same time, we may find tubular breathing and crepitations close together, just as later we notice **redux crepitation** and tubular breathing at the same time.

The second stage lasts three or four days, or sometimes longer, after which recovery usually commences. The situation may, however, become worse. When pneumonia ends in recovery, the fever falls very rapidly with symptoms of crisis; the wasting ceases, and the patient enters on convalescence. This condition coincides with the liquefaction of the exudate; the sputum becomes opaque and rich in fatty elements, the dullness disappears, and the tubular breathing gives place to a râle that is larger and more moist than the crepitation. This râle is the **redux crepitation**, which would better be called a subcrepitant râle. It is audible both during inspiration and expiration.

If pneumonia passes into the third stage (grey hepatization), the sputum assumes a greyish or prune-juice tint, the fever becomes adynamic in type, the pulse is small and irregular, the abdomen is distended, diarrhœa appears, clammy sweat covers the patient, and delirium is present at the end, which is almost always fatal.



### Extrapulmonary Localizations.

The infection does not always spend all its force on the lung. In many cases, even in frank pneumonia, but especially in epidemic pneumonia of a well-marked infectious type, the pneumococcus invades the pleura, pericardium, endocardium, meninges, stomach, peritoneum, joints, ear, etc. The invasion of these organs may be consecutive, parallel, or anterior to that of the lung; it may, indeed, be independent of the pulmonary invasion. Let us study these forms.

1. **Pneumococcal Pleurisy.**—The pleura is almost always affected in pneumonia. The pleurisy is frequently dry, and limited to the production of false membranes, which vary in thickness, and cover the pleura to a variable extent, especially at the interlobar fissures.

In other cases we find pleurisy, with sero-fibrinous or purulent effusion. The pleural inflammation sometimes develops as a contact lesion from the pneumonic focus; at other times it arises at a distance from the focus, the pneumococcus invading the pleura on its own account.

Pleurisies with effusion often supervene in the decline of pneumonia or in full convalescence. They have, therefore, received the name of **meta- or post-pneumonic**. As a rule, they are not sero-fibrinous, but suppurative. Suppuration may be caused by the pneumococcus, without the aid of the usual pyogenic organisms (streptococcus and staphylococcus); and when these organisms are present, the infection is secondary. Meta-pneumonic pleurisy may invade the general cavity of the pleura, or else be encysted, interlobar, diaphragmatic, or mediastinal (see Chapter V.). These forms may arise without pain, the fever may or may not return, and after three to six weeks they often end by **vomica**. Some eventually open in an intercostal space; others, especially in children, terminate in absorption. The prognosis of these pleurisies is, as a rule, not grave. Those which occupy the general cavity may exceptionally yield to thoracentesis, but in most cases operation for empyema is necessary.

We also find early pleurisies in which the effusion occurs at the same time as pneumonia. Lemoine has given them the name of **para-pneumonic**.

We must not forget that aseptic puriform effusions may also exist in pneumonia; they must not be confused with septic purulent pleurisy.\*

2. **Pneumococcal Endocarditis.**—Endocarditis is a fairly frequent complication of pneumonia. It appears during the course of this affection (para-pneumonic), but most often during convalescence (meta-pneumonic). The pneumococcus itself is the cause (Netter), and is rarely found in association with other microbes, such as streptococci (Weichselbaum), or special bacilli (Lion). In exceptional cases endocarditis consecutive to pneumonia is the result of the streptococcus alone (Jaccoud), and the infection

\* *Vide* Chapter V., Section 6.

is secondary. Endocarditis is more frequent in certain epidemics of pneumonia, in cases associated with influenza, and in the course of pregnancy. It is in general accompanied by other extrapulmonary infections due to the pneumococcus, such as purulent pleurisy, pericarditis, arthritis, etc. Suppurative meningitis in particular is frequently associated with it. The invasion of the endocardium by the pneumococcus may be independent of pneumonia, and show itself in the course of certain affections caused likewise by the pneumococcus—viz., broncho-pneumonia, cerebro-spinal meningitis, inflammation of the great serous membranes, etc. Endocarditis may develop in subjects who have no cardiac lesion, but previous valvular disease is singularly favourable to its appearance.

Pneumococcal endocarditis usually attacks the left heart, and is found at the aortic more often than at the mitral orifice, in the proportion of 3 to 2 (Netter). Though it more rarely affects the right heart (one-seventh of the cases), it is, nevertheless, more frequent there than in endocarditis due to other microbes. Pneumococcal endocarditis causes vegetations rather than ulcerations. The vegetations are rounded, with a regular surface; the largest are sessile, with a broad base, and very adherent. They are only detached in exceptional cases. Embolism is rare, and septic capillary emboli are not often seen. It is, therefore, exceptional to find emboli of the spleen, kidneys, etc., contrary to what is seen in streptococcal or in staphylococcal endocarditis, where the loosely attached vegetations often give rise to these accidents. Destructive lesions may be seen—viz., little ulcers of the endocardium, tiny abscesses in the myocardium, followed by the production of valvular aneurysms. The virulent pneumococcus is found in the deep parts of the vegetations, just as in the blood. Pneumococcal endocarditis has been caused experimentally in the rabbit after previous injury to the valves, and even without traumatism.

As a rule, endocarditis is silent when it develops at the same time as pneumonia, and passes unnoticed if the heart is not examined daily. Metapneumonic endocarditis, which supervenes some weeks after the onset of pneumonia, and often after a more or less complete period of apyrexia, may commence with a rigor and rapid rise of temperature. It commonly assumes the typhoid form of malignant endocarditis, with fever and grave adynamia. Auscultation may reveal blowing murmurs, variable in intensity and position at the different orifices. In some cases the symptoms of concomitant meningitis are the chief feature (Osler). The usual termination is death, which occurs after a very variable period. Exceptional cases, followed by recovery (Traube, Lion), with or without persistence of the valvular lesions, are, however, recorded.

**3. Pneumococcal Pericarditis.**—This is another insidious manifestation, only discovered by daily auscultation. This pericarditis, which is



almost always accompanied by pleurisy, rarely begins before the fifth day of the pneumonia. Its frequency is variable, according to the epidemic. Abundance of fibrin is present, and the effusion is usually purulent.

**4. Pneumococcal Meningitis.**—Meningitis may supervene during pneumonia, or, later, during convalescence. In the former event it often passes unnoticed, the excitement and delirium being put down to fever or alcoholism. This is a common error. In the latter form the symptoms comprise fever, headache, quiet or violent delirium, pain in the nape of the neck, muscular rigidity, squint, contraction of the jaws, inequality of the pulse, Cheyne Stokes breathing, and coma.

Post mortem the pia mater is infiltrated with greenish-yellow exudate, and the dura mater is converted into a thick cap. Bulbar and spinal meningitis are frequently met with. In some cases epidemic cerebro-spinal meningitis, due to the pneumococcus, has been observed.

**5. Pneumococcal Nephritis.**—The urinary troubles are various. Albuminuria, hæmaturia, and anuria, have been noted. True nephritis, with uræmic symptoms, may occur.

The renal changes have been minutely studied by Caussade. The kidney is large and ecchymotic. The nephritis is almost always hæmaturic. It may be caused by the pneumonic infection, for the pneumococcus has been found in the kidney or may be grafted upon existing lesions.

**6. Pneumococcal Gastritis.**—The stomach, like other organs, may be infected by the pneumococcus. I have quite recently seen this gastric infection in two patients suffering from pneumonia with generalized infection—viz., peritonitis, pericarditis, endocarditis, meningitis, and arthritis.\* These patients presented gastric symptoms of pain, nausea, vomiting, and abundant hæmatemesis. Post mortem, the mucosa was studded with hæmorrhagic erosions, due to acute necrobiosis. In one case pneumococci were swarming in the erosions. This form of gastritis is described in detail under Ulcers of the Stomach.

**7. Pneumococcal Peritonitis.**—I do not allude here to the primary form,† which will be described later. I am at present concerned with peritonitis which arises as a secondary condition in the course of pneumonia. Secondary peritonitis is rarer than the primary form. The symptoms are abdominal pain, tympanites, nausea, vomiting, and diarrhoea. This secondary peritonitis is not, in my opinion, as serious as the primary form. It was on the road to recovery in the two cases described.‡

\* Dieulafoy, "Gastrite ulcéreuse pneumococcique" (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 11<sup>me</sup> leçon, p. 219).

† Dieulafoy, "Péritonite primitive à Pneumocoques" (*Clinique Médicale de l'Hôtel-Dieu*, 1896, 18<sup>me</sup> leçon, p. 396).

‡ Vide "Ulcerative Gastritis."

**8. Pneumococcal Arthritis.**—Arthritis and synovitis may supervene during the course or the decline of pneumonia. In exceptional cases they may precede it, and, indeed, we see primary arthritis without pneumonia. It usually presents the same clinical aspect. The affected joints are the seat of very acute pain, which is soon followed by swelling, œdema, and redness. It might almost be called a blennorrhagic arthritis, for the peri-articular synovial sheaths often participate in the process. Movement is very difficult, pressure is very painful, the temperature is raised, and the tongue dry, while the patient is prostrated and shows the signs of grave infection.

The course varies somewhat in different cases. Sometimes it is rapid, and pus forms in a few days, but at other times its progress is slower. The prognosis of these joint lesions is usually grave not from the arthritis, but from the more or less general infection. The gravity of the prognosis cannot be based on the degree of virulence of the pneumococcus, for in a case ending favourably the pneumococcus was very virulent, while in one ending fatally its virulence was low. However this may be, the therapeutic indication is to let out the purulent fluid.

Previous lesions (rheumatism, trauma) may favour articular infection by the pneumococcus. Experiments have given similar, but not constant, results.

The lesions vary, according to the intensity and the duration of the infection. We may find serous or purulent effusion, thickening of the synovial membrane, and destruction of the articular cartilages and the ends of the bones. In one of my patients suffering from arthritis of the wrist, the joints contained  $\frac{1}{2}$  ounce of pus, rich in pneumococci; the articular surface was rough, and in places the cartilage had completely disappeared.

**9. Pneumococcal Otitis.**—Otitis is a fairly frequent complication of pneumonia. The disease has an acute course, and usually ends in recovery. Nevertheless, it may give rise to cerebral or cerebro-spinal meningitis, sinus phlebitis, or abscess of the brain and of the cerebellum.

The enumeration of the complications of pneumonia, which include pleurisy, meningitis, parotiditis, peritonitis, otitis, and suppurative arthritis, shows that **suppuration** plays a great part. The pneumococcus alone can cause suppuration, and the pus in such a case has special characters. It is viscid, rich in cellular elements, and of a greenish colour, like laudable pus; the serum does not separate out. In other cases, however, the usual organisms of suppuration—*i.e.*, streptococci and staphylococci—cause secondary infection and suppuration.

**Varieties.**—Pneumonia does not always show the same characters, but assumes somewhat different forms, according to the age of the patient (old age), to the previous condition of health (want, alcoholism, pregnancy), to the numerous **extra-pulmonary** localizations, to the seasons of the



particular year, or to other causes which are still imperfectly known (**epidemic, or influenzal pneumonia**) ; and, as Peter says, pneumonic conditions exist besides pneumonia ; in other words, there is a clinical as well as a pathological aspect.

### Varieties in Situation.

**Central Pneumonia.**—Pneumonia sometimes remains localized in the deep tissue of a lobe, and though the symptoms—*i.e.*, rigor, high temperature, dyspnœa, cough, and rusty sputum—point to pneumonia, yet the physical signs are wanting ; dullness, râles, and tubular breathing are absent, until the inflammation extends and yields the usual signs.

**Double Pneumonia.**—A second attack of pneumonia may arise during the course of the first. This second attack shows itself from the sixth to the eighth day. “Pneumonia is never double from the first” (Grisolle). As regards site, every variety is possible, but most often the corresponding lobe of the opposite side is affected. The second pneumonia is generally less extensive than the first, and the inflammation is less severe. It does not begin with a fresh chill or pain in the side, and the appearance of the sputum is scarcely altered. Percussion and auscultation reveal the fresh focus which the symptoms had not announced. The dyspnœa, however, is more acute at the time of the fresh invasion, and the temperature in the axilla affords valuable help. In unilateral pneumonia the axillary temperature is higher by some tenths on the affected side ; in double pneumonia the temperature is the same on both sides (Landrieux).

**Apical Pneumonia.**—This form is justly regarded as very grave, and is often accompanied by collapse, adynamia, and a typhoid state. Suppuration is prone to occur, and delirium and jaundice may appear, while the usual signs of pneumonia—*i.e.*, pain in the side, cough, and expectoration—are less marked. Apical pneumonia is a serious disease—first, because it is more frequent in old age,\* and also because alcoholism and general ill-health favour apical pneumonia. The reasons for this predilection are well discussed by Peter, who regards the upper lobes of the lung as endowed with less vitality, and as “auxiliary and additional.”

Apical pneumonia is more frequent on the right side, and when the signs cannot be found in the clavicular region or in the supraspinal fossa, they must be looked for in the axilla. The situation of this variety might lead us to take it for a tubercular lesion. This error in diagnosis must be avoided.

**Massive Pneumonia.**—In this form of pneumonia (Grancher) the fibrinous coagulation extends throughout the whole bronchial network of

\* We see in the statistics of Durand-Fardel that in thirty cases of fatal pneumonia the apices were affected eighteen times (“*Traité des Maladies des Vieillards*,” p. 460).

the invaded territory, and reaches even the large bronchi. The air, therefore, cannot enter the bronchial tubes, and most of the stethoscopic signs of pneumonia are wanting. Neither tubular breathing nor râles are heard; expectoration is almost absent. It is therefore evident how difficult it may be to distinguish between pleuritic effusion and massive pneumonia. However, certain signs are of value in diagnosis. The dullness of pleural effusion is more complete and more fluid than that of pneumonia. Further, the displacement of organs (notably of the heart), which is absent in the case of pneumonia, is more or less marked in the case of effusion.

### Varieties of Pneumonia, according to Age.

1. **Children** are more subject to lobular than to lobar pneumonia; still, lobar pneumonia is often seen, even as early as the first year. In young children the onset is often accompanied by convulsions, vomiting, and erythematous eruptions. The respiration is panting, and the pulse-rate may exceed 140. About the age of five years the dry, crepitant râle is heard; below this age we find rather the subcrepitant râle. The other signs closely resemble those of pneumonia in the adult, but the child does not expectorate, and hence the absence of rusty sputum, which, however, is sometimes seen in four- to five-year-old children. Under two years of age the cyclical course of the fever is really the only differential sign between lobar and lobular broncho-pneumonia. The prognosis is grave only in the very young; a little later the disease is relatively benign.

2. **In the aged**, in whom there is little organic reaction, pneumonia is insidious; the rigor is insignificant, and the pain in the side may escape notice. The colour of the face and the dryness of the tongue are sometimes the only signs. We wait in vain for rusty sputum, which does not appear. The crepitant râle is coarser than in the adult—in a word, pneumonia is altered by the age of the individual. At the Salpêtrière ambulatory pneumonia is sometimes seen in old women, who continue to eat and to attend to their occupations. They die almost suddenly, and suppurative pneumonia is found post mortem.

### Clinical Varieties of Pneumonia.

At the beginning of this article I described the ordinary form of lobar pneumonia, but it is clear that the extrapulmonary localizations in the pleura, heart, meninges, and kidneys give a special aspect to pneumonia. On the one hand, some clinical varieties depend upon the predominance of the lesions in a certain organ, while in other cases the varieties depend on the general appearance of the morbid complex, and the disease assumes special features.

1. In the so-called **inflammatory** or **sthenic** form, often described by



old writers, and frequent in the country, the chief characters are as follows : Redness of the face, epistaxis, violent headache, restlessness, severe inflammation of the lung, sputum which is sometimes bloody, speedy hepatization, rapid and quivering pulse.

2. The **asthenic** form is characterized by the following symptoms : Insidious onset, lassitude, early delirium, subsultus tendinum, prostration, stupor, soft and unequal pulse, tendency to adynamia, and collapse.

3. The **nervous** forms show several varieties. Delirium is common, especially in drunkards. It may not be associated with cerebral lesions, while in other cases delirium is caused by cerebral or cerebro-spinal meningitis, due to the local action of the pneumococcus.

The **paralyses** consecutive to pneumonia show different forms. In the acute phase of pneumonia they nearly always affect the hemiplegic type, with or without aphasia, and with or without apoplexy. These paralyses, which are curable in the adult, but fatal in the elderly, are due to lesions of the cerebral vessels (Lépine).

The paralyses which supervene during convalescence affect the paralytic type, or the type of isolated paralyses, with or without muscular atrophy. These paralyses are in some cases due to meningo-spinal changes, in others to peripheral neuritis of toxic origin, like the palsies of diphtheria.

4. **Biliary** pneumonia comprises two very distinct forms. In the one there is pneumonia with jaundice, which is consecutive to catarrhal inflammation of the bile-ducts, or to perihepatitis produced by right basal pneumonia. These lesions are then purely **local**. In the other there is pneumonia with a biliary condition, which depends on a **general** morbid state. This condition is characterized by a subicteric tint, with absence of active reaction, soft pulse, headache, foul tongue, vomiting, and diarrhoea. The biliary condition accompanies the grave forms (apical pneumonia, alcoholism). It is connected with "the medical constitution of the season of the year." It forms part of what was called peripneumonic biliary fever, and explains the epidemics of biliary pneumonia. In some exceptional cases pneumonia is connected with a diffuse hepatitis, a variety of **icterus gravis**.

5. **Malignant and Epidemic Pneumonia**.—Every pneumonia, whether benign or malignant, is an infective disease, but clinically it is customary to reserve the epithets "infective" and "malignant" for the grave and abnormal forms. There is, first of all, the infectious pneumonia which has at different times appeared in epidemic form during epidemics of malaria, scurvy, and typhoid fever. Next, there is secondary pneumonia, supervening in the course of some general disease, and deriving its character and gravity from the surroundings in which it develops.

In many cases, however, pneumonia develops on its own account—sometimes in an isolated form, but more often in the form of more or less circumscribed epidemics, which may be associated with those of **influenza**. It is accompanied by all the features of an infectious malady.

The **contagion** is quite proved. The epidemic breaks out in a prison, in barracks, or in a small area, or confines itself to a house or to a family, three or four members being affected simultaneously or successively.

In some cases this epidemic pneumonia differs but little from genuine pneumonia. As a rule, it presents special features, which have earned for it the names “typhoid,” “asthenic,” “adynamic,” etc. Hepatization sometimes occurs at several foci. The sputum is bloody rather than rusty, and the disease is accompanied by swelling of the spleen, diarrhoea, albuminuria, jaundice, pleurisy, pericarditis, vegetative endocarditis, meningitis, parotiditis, precordial distress, and prostration. The fever-chart does not resemble that of frank pneumonia. The histological lesions are somewhat special. The prognosis is not absolutely bad. The disease is benign in one epidemic, malignant in another. The prognosis, like the clinical picture, comprises **every intermediate form**.

**6. Pregnancy and Pneumonia.**—Grisolle maintained that lobar pneumonia is formidable in pregnant women. At the present day the opposite opinion holds good. Many cases of pneumonia occurring at different stages of pregnancy have been published. The disease was relatively benign for mother and child. Last year I saw three cases of pneumonia during pregnancy, one at a very late stage. The women recovered, and delivery took place without mishap. The transmission of pneumonia to the foetus is an established fact. In the published cases the child has succumbed a few days after delivery. The infant may succumb without showing any lesions in the lung. Post mortem we find lesions in the liver and the spleen, due to toxines which have traversed the placenta.

Pneumonia in a nursing woman diminishes or suppresses the secretion of milk. As the pneumococcus can be transmitted by suckling, the mother should not continue to suckle her child.

**Ætiology.**—Lobar pneumonia is a disease of adults. In the aged it assumes special features, and the child, though more subject to the lobular form, may contract the lobar variety. Changes of season—as in November, March, and April—appear favourable to its development. Previous debility is not necessary as a predisposing cause, for pneumonia frequently attacks people in perfect health. Some persons have a special predisposition to **recurrences** and contract several attacks. Probably the pneumococcus is always present, awaiting a favourable opportunity for development.

I have previously discussed **epidemicity**. Epidemics of pneumonia



may be limited to a **locality**, or spread through a town, province, or country. They often coincide with influenza, and are due to unknown atmospheric or climatic causes, which increase the virulence of the microbe.

The question of **contagion** demands notice. Pneumonia is contagious, and the sputum most often spreads contagion, which may be active long after recovery. The foetus may be infected by its mother, and may itself show the pulmonary and extrapulmonary lesions of the infection.

The **nature** of lobar pneumonia has been interpreted in various ways, and till recently two chief theories were in evidence. One—the Hippocratic doctrine, upheld and defended by the Montpellier school—regarded the lesion of the lung as the local and secondary expression of a general state, called **pneumonic fever**. Pneumonia—that is to say, the lesion—would then be the result of pneumonic fever, which is the disease. The anatomo-pathological school of Paris has brought forward quite an opposite theory: The lesion in the lung represents the whole disease, and the local lesion is the cause both of the fever and of the general symptoms.

Between these extremes an intermediate opinion has found a place. It is generally admitted that a chill is perhaps the commonest cause of pneumonia; for effective action, cold, or any other provoking cause, must find the system in a **favourable condition of receptivity**.

Firstly, is it true that cold plays such a large part in the development of pneumonia? According to some statistics, cold is said to act as a provoking agent in only a half or a third of the cases. The cause of lobar pneumonia resides in the existence of an infectious organism—the pneumococcus—but the chill is the chief provoking agent.

The pneumococcus being the cause of pneumonia, how are we to admit that its entrance into the lung may be followed, after such short delay, by the symptoms of acute pneumonia? In answer to this objection, Jaccoud admits, with good reason, the possibility of auto-infection. “The human organism constantly lodges various microbes in large numbers. As long as its functions are normal, it is a hostile medium, which wards off their noxious effects. However, let disturbance arise and alter the physiological functions, the hostile medium becomes friendly, and the affected organism is deprived of effective resistance against the very microbes which it but lately tolerated without being affected.” The pneumococcus exists normally in the mouth (Pasteur), in the pharynx, and in the bronchi (Netter). Should it enter the lung in those who are not in a state of **receptivity**, its pathogenic influence is annihilated by the activity of the phagocytes. In the opposite condition pneumonia declares itself.

The dissemination of the pneumococcus produces the extrapulmonary localizations above described.

In opposition to the **primary** form just described, we see secondary

infections which are less frank in evolution, and supervene in other diseases (diabetes, cachexias, gout, eruptive fevers, etc.). This variety of lobar pneumonia is somewhat rare, for the secondary form chiefly devolves upon lobular pneumonia.

**Course—Duration—Termination.**—Lobar pneumonia has a mean duration of five to ten days. The period during which the temperature rises is short and rapid. By the second day it reaches its maximum ( $104^{\circ}$  to  $106^{\circ}$  F.), remains stationary for some days, with a morning remission of  $\frac{1}{2}^{\circ}$  to  $1^{\circ}$ , and in most cases defervescence is sudden and complete in twenty-four hours. This defervescence, which usually occurs between the fifth and the seventh days, is often accompanied by symptoms of **crisis**—viz., sweating, epistaxis, diarrhœa, and profuse secretion of urine containing albumin and excess of chlorides.\* This increase varies in proportion to the retention of chlorides in the tissues during the illness. It is remarkable that the defervescence is sometimes preceded by transient aggravation of the disease. This has been called the **precritical** phase. Labial herpes cannot be considered as a symptom of crisis, for it often appears at the onset of pneumonia.

Suppuration in the lung is a frequent cause of death, which, however, may occur before the appearance of grey hepatization. Some patients are struck down by adynamia and fatal collapse during the stage of red hepatization, while others succumb through the extensive nature of the lesion, which invades several lobes, narrows the field of hæmatisation, and induces asphyxia and cardiac paralysis. In some well-proved cases suppurative pneumonia has caused death by purulent infection, and the affected tissue may suppurate as a result of the pneumococcus alone.

**Diagnosis—Prognosis.**—Let us first differentiate between lobar pneumonia and other inflammations of the lung.

1. **Lobar pneumonia** is nearly always primary. It invades one or several lobes, remains confined to a definite area, and spares the rest of the organ. Onset, course, and termination are clearly marked: the crepitant râle, the tubular breathing, and the rusty sputum scarcely permit confusion; suppuration is exceptional, and recovery is the usual termination when the infection does not involve other organs. These characters are distorted when the pneumonia is secondary, takes on an epidemic form, or develops in the aged.

I would add, too, that even in its frank forms lobar pneumonia does not always present the complete picture sketched in this chapter. It has been my lot to see cases of lobar pneumonia which differ from the classical type. Many so-called lobar pneumoniæ border on **inflammation of the chest**,

\* Up to defervescence the urine was scanty, dark, and in twenty-four hours contained 35 to 50 grammes of urea, instead of the normal quantity of 28 to 30 grammes.



and are the intermediary forms which unite **clinically** the various inflammations of the respiratory passages.

2. **Lobular or broncho-pneumonia** is especially frequent in children, and is often consecutive to some other malady (measles, diphtheria, whooping-cough, influenza, tuberculosis, etc.). It deserves the name of "fibrinous" less than the preceding form. It is lobular—that is to say, it spreads by centres which are scattered through both lungs, and may be isolated or confluent. The disease does not exhaust all its action on the inflamed centres. It progresses by successive outbursts; its lesions and its course are irregular, and its description differs notably from that of lobar pneumonia.

3. **Inflammation of the chest** is not pneumonia, and, on the other hand, differs from simple congestion. It is a morbid condition in which the **hyperæmic and inflammatory elements are differently combined**. The inflammation affects one or several lobes, and does not spare the other parts of the respiratory system. The bronchi, the pleura, and the muscles of the thorax—in fact, all the superposed layers of the chest—may be affected in different degrees.

4. **Spleno-pneumonia** (Grancher) is thus defined by this author: "Between pulmonary congestion and lobar pneumonia, and side by side with broncho-pneumonia, there exists a morbid condition of the lung—a kind of subacute pneumonia, which simulates pleurisy with moderate effusion, and deserves its own description and denomination." Potain has described a variety of pneumonia which he has named congestive, and to which he says spleno-pneumonia may be likened.

5. **Hypostatic pneumonia** has not the anatomical attributes of a true pneumonia. It is a mixed condition, in which passive congestion and cedema play the chief part, and are accompanied by slight transudation of fibrin, and sometimes by hæmorrhage. This morbid condition, consecutive to heart disease, hypostasis, and prolonged decubities, chiefly involves the posterior and lower parts of the lungs.

6. **Pleurisy** at first presents numerous analogies with pneumonia. In pleurisy, however, the chill is less violent, the initial temperature is not so high, the pain in the side is often more acute, expectoration is absent, and the friction sound is more moist and diffuse than the dry, crepitant, and clearly localized râle of pneumonia.

It is not sufficient to diagnose pneumonia: we must also know whether it is inflammatory, biliary, or adynamic; whether it is or is not complicated by pleurisy, endocarditis, pericarditis, meningitis, or otitis; whether it is primary or secondary; whether it is accompanied by alcoholism; whether it is the first act in commencing typhoid fever; whether it has developed in a diabetic or in a tubercular subject. Each of these points affects the prognosis and the treatment as much as the diagnosis.

**Treatment.**—The treatment of pneumonia should be especially directed to the general condition. When pneumonia is regular and benign, we should be content with watchful expectancy, ordering acid drinks, laxatives, broths, and wine diluted with water. In the contrary case the indications must be acted on. Blisters are more harmful than useful in my opinion.

Pain in the side may be relieved by leeches, antipyrin, injections of morphia, or by an ointment of vaseline 10 parts, methyl-salicylate 1 part.

In sthenic pneumonia antiphlogistic treatment should be employed—*i.e.*, blood-letting, cupping, leeches, and tartarate of antimony, or, better still, kermes, administered to Trousseau's prescription :

|                      |    |    |    |    |    |          |
|----------------------|----|----|----|----|----|----------|
| Kermes               | .. | .. | .. | .. | .. | gr. xxx. |
| Extract of digitalis | .. | .. | .. | .. | .. | gr. iii. |
| Medicinal soap       | .. | .. | .. | .. | .. | q.s.     |

For 20 pills.

Ten to fifteen pills are given in the twenty-four hours, and if vomiting or diarrhoea supervene, 1 drop of Sydenham's laudanum is given with each pill.

Good results are also obtained by infusion of digitalis, given at intervals in the twenty-four hours (Hirtz); but I have never tried this remedy.

**Biliary** pneumonia may be cut short by emetics, and especially by ipecacuanha. Adynamic pneumonia should be treated with tonics and stimulants, such as quinine, tincture of coca, wine, or brandy. If the fever is severe, sulphate of quinine and antipyrin may be given.

When pneumonia takes the **ataxic** form, with delirium, restlessness, and high fever, the following draught, in tablespoonfuls every hour, may be given with advantage :

|                     |    |    |    |    |       |
|---------------------|----|----|----|----|-------|
| Orange-flower water | .. | .. | .. | .. | 3iii. |
| Cherry-laurel water | .. | .. | .. | .. | 3iii. |
| Syrup of ether      | .. | .. | .. | .. | 3x.   |
| Bromide of potash   | .. | .. | .. | .. | 3ss.  |

Cold baths have been extolled in the ataxic and hyperthermic forms of pneumonia. I have used this treatment several times with success. I would advise giving the baths just as in typhoid fever.

If pneumonia is accompanied by irregular pulse, feeble heart-action, and a tendency to syncope, subcutaneous injections of the following solution of caffein should be given :

|                      |    |    |    |    |      |
|----------------------|----|----|----|----|------|
| Distilled water      | .. | .. | .. | .. | 3ii. |
| Benzoate of soda     | .. | .. | .. | .. | 3ss. |
| Benzoate of caffeine | .. | .. | .. | .. | 3ss. |

I would also advise in such a case injections of serum (8 to 16 ounces or more daily). For further details, see chapter on Therapeutics.

The patient should drink plenty of milk, fresh water, and tisanes, with



or without lactose, for it is essential to promote urinary secretion. Milk has the further advantage of protecting the kidney—a fact not to be despised, especially as pneumococcal nephritis is now well recognized.

Lastly, I must say a few words about the treatment of pneumonia by subcutaneous injections of essence of terebinth.

Fochier (of Lyons) observed that in certain cases of puerperal infection, when no important lesion can be found, sudden improvement may coincide with the appearance of pus in the iliac fossa, the breast, a joint, or elsewhere. The appearance of a local abscess or cellulitis seemed to have a curative action. Fochier gave it the name of **abscess of fixation**. The question was then asked whether analogous abscesses could not be produced therapeutically. In women whose condition appeared desperate recovery was induced by subcutaneous injections of essence of terebinth.

As Lépine had been successful in a case of pneumonia, I applied the treatment to two women suffering from severe pneumonia which was probably on the point of suppuration. Both patients recovered. The treatment is as follows: An injection of 15 minims of essence of terebinth—*i.e.*, 60 minims for the four injections—is given by means of a sterilized syringe, in the subcutaneous tissue of the outer surface of each thigh and of the deltoid region of each arm. These injections produce extremely acute pain, lasting about two hours. The next day the injected regions present an œdematous, whitish, and diffuse thickening. The abscess which forms is opened, and the pus in my two cases was **amicrobic**.

Whether these phlegmons be called **abscesses of fixation** or **abscesses of derivation** matters little. What does matter is the therapeutical result. It deserves, I think, to be taken into serious consideration. This treatment, in my opinion, should be reserved for patients who are suffering from grave pneumonia in which grey hepatization is imminent.

**Collargol** has sometimes given good results (Netter). An ointment containing 13 per cent. of collargol is employed, and a piece about the size of a nut is daily rubbed into the skin. It is better to use intravenous injection of 15 to 30 grains of collargol.

The **prophylactic** treatment of pneumonia should not be neglected. It must not be forgotten that, as pneumonia is contagious, proper precautions must be taken to isolate the sick, and to disinfect the sputum and articles of bedding or clothing used by the patient.

## V. CHRONIC PNEUMONIA—FIBROSIS OF THE LUNG.

Chronic inflammation of the lung may affect the parenchyma and the connective tissue. These lesions give rise to the varieties of chronic pneumonia known as lobar, lobular, and cortical.

The **chronic pneumonia** called **caseous** is really tubercular, and will be studied later, under Phthisis.

**Chronic Lobar Pneumonia.**—The **lobar** form is much rarer than the lobular one. It may be primary, or may follow acute pneumonia. Malaria appears to play some part in its development.

In our study of acute pneumonia we have seen that residual inflammation may still remain in the alveoli, and may take several weeks to absorb (Andral). This process, though it may be slow, rarely ends in chronic pneumonia, because the parenchyma of the lung is unaffected; and if it do so end, it is because the parenchyma is affected by fresh inflammation.

Two stages are described in chronic pneumonia—viz., red and grey induration. The lung tissue affected with **red induration** is firm and increased in size; the cut section is less granular, and the tissues are less friable than in the red hepatization of acute pneumonia. The indurated part does not crepitate, and sinks in water. The walls of the alveoli and the perilobular connective tissues are invaded by fibrous tissue. The alveolar cavities are narrowed, and at times invaded by nodules of fibrous tissue which has replaced the wall of the alveolus.

The fibrosis is intra- and extra-lobular. After several months red induration gives place to **grey induration**. The lung tissue, which has become impermeable, retracts and diminishes in volume. It is hard, creaks under the knife, and has all the attributes of fibrous tissue (Cruveilhier's fibrous metamorphosis). In this fibrous tissue excavations (ulcers of the lung) are sometimes met with, but no bronchiectasis is found, contrary to what is seen in chronic broncho-pneumonia.

This chronic pneumonia extends in an uniform way through a whole lobe or a part of the lung. It is more common at the base than at the apex.

Dullness, deformity of the chest, muco-purulent sputum, tubular breathing, râles, and gurgling, are the signs of chronic pneumonia; and if we add frequent hæmoptysis and cachexia, with fever, sweats, and wasting, it will be admitted that the diagnosis from phthisis would be very difficult if the existence of the latter were not confirmed by the presence of **bacilli** in the sputum.

**Chronic Broncho-Pneumonia.**—Chronic broncho-pneumonia is more frequent than the preceding form, and usually follows acute or subacute broncho-pneumonia. It is most common in early life, and is caused by measles, diphtheria, whooping-cough, influenza, typhoid fever, and syphilis.

In the chronic, as in the acute, form the lesions affect both the bronchus and the lobule. In a vertical section of the lobule, during the **subacute** phase the bronchus is dilated, and its normal elements are converted into embryonic tissue. The region of the alveoli which surrounds the bronchus



is affected by hepatization; the walls of the alveoli show embryonic infiltration, and the alveolar cavities contain exudate, with epithelial cells and leucocytes. Around the hepatized area we find the splenized zone, which is the seat of congestion; the epithelial cells desquamate and fall into the alveolar cavity.

As the lesion becomes **chronic** the intra-alveolar elements undergo granulo-fatty degeneration, and the embryonic cells, which infiltrate the parenchyma, change into fibrous tissue. The fibrosis affects the peribronchial and perilobular connective tissue and the parenchyma of the lung atrophies.

Chronic broncho-pneumonia chiefly affects the lower lobes and the posterior part of the upper ones. The lung tissue is violet-coloured, dense, and dry; the cut section is smooth, without granulations, and the divisions between the lobules are still perceptible under the low power. These lesions were called **carnification**, by comparison with muscular tissue. In some cases the lung is fibrous and atrophied, and dilatations of the bronchi are found. They are due to changes in the bronchial walls, and as they are found before the lung atrophies (Charcot), it is hardly probable that they are consequent on the pulmonary fibrosis, as Corrigan supposed.

The **progress** of chronic broncho-pneumonia is very slow, and the disease passes through a subacute stage, subject to periods of arrest. Dullness, râles, tubular breathing, and sometimes gurgling, are the most usual signs. Fever is common, the expectoration is muco-purulent, or at times blood-stained, and the disease usually ends in hectic fever.

**Chronic Cortical Pneumonia.**—In some cases of pleurisy, when the absorption of fluid is very slow, the pleura becomes thickened, and forms a fibrous shell over the lung. The lobes are adherent to one another, and the parietal pleura is likewise adherent to the chest-wall. This fibro-plastic process does not always remain limited to the pleura, but reaches the lung probably along the lymphatics. The connective spaces between the lobules are transformed into fibrous arches, which surround the lobules, and finally involve the alveoli themselves.

**Pleuro-pulmonary fibrosis** is thus brought about. These cases are somewhat rare (Brouardel, Tapret). I have seen one case in which bronchiectasis was also present.

**Pulmonary fibrosis** as a secondary lesion is associated with diverse changes in the lung. It accompanies emphysema, tuberculous lesions, tumours, hydatid cysts, pneumokoniosis, etc. It is fairly common in elderly people. The fibroid regions are indurated and pigmented, the fibrous framework of the lung is thickened, the walls of the vessels take part in the change, and the alveolar cavities are atrophied by the new tissue. **Syphilitic** fibrosis of the lung exists, and will be studied under Syphilitic Lesions of

the Lung. The lesion called **slaty induration** of the apices, and frequent in the lungs of elderly people, is nothing but a highly pigmented fibrous tissue. This tissue bounds the alveoli, which are atrophied in some parts, emphysematous in others, and often contains small cysts of long standing, transformed into caseous or chalky material.

## VI. OCCUPATION PNEUMONIAS—PNEUMOKONIOSIS.

The dust from coal, iron, steel, copper, and silicon, gives rise to chronic pneumonia, which we shall study under the names of anthracosis, siderosis, and chalicosis.

**Anthracosis.**—Anthracosis may be physiological, most human lungs being normally mottled with black. When, however, the infiltration of carbon becomes excessive, the lesions cause special symptoms. Anthracosis occurs chiefly among miners, charcoal-burners, and moulders in copper, brass, or bronze, who use carbon dust in their work.

The lesions seen are: at first emphysema, then a black coloration of the lung, which no longer crepitates, creaks under the knife, and sinks in water. On crushing the lung tissue, the fingers are coloured black, and also the water allowed to flow over its surface. The cut section may be mottled with black or be of an uniform black colour. The lung tissue is divided by large bands of connective tissue, which contain particles of carbon, heaped up at certain spots to form nodules. Histologically, we see fibrosis, which affects the connective tissue surrounding the lobule and its central bronchus. A fibrous mass, crammed with black grains, is found in these regions. Bronchiectasis is hardly ever seen; on the other hand, obliteration of the bronchial arterioles is frequent, and hollow, irregular ulcerations, containing black, putrid material, are the result. The bronchial walls are intact. The pleura is adherent and thickened; the glands are hard and black. Lesions of the right heart are sometimes seen.

Does anthracosis predispose to tuberculosis? Authorities are not in accord on this point. Oberthür says positively that tuberculosis is rare in coal-miners. It may be admitted (Boulland) that if the damage to the lung favour the growth of the bacillus, this growth is arrested by the fibrosis, which isolates the foci and prevents their development. The symptoms display three periods (Tardieu). In the first period we see malaise, with loss of appetite, wasting, and fits of coughing, which are followed by blackened expectoration. On auscultation, the vesicular murmur is feeble, the voice-sounds are exaggerated and sibilant and snoring rhonchi are sometimes audible. In the second period the symptoms grow worse. Vomiting appears, the distress increases, and the induration of the lung is complete. Mucopus is often found in the sputum, and at times a little blood is present. The third



period is characterized by the progress of the anæmia and by decline. The lung is hollowed out by cavities, and death supervenes, either by cachexia or by asystole. The duration may be several years.

**Chalicosis.**—Infiltration of the lung by the dust of silica (chalicosis) is seen among stone-cutters (quarrymen, millstone sharpeners, flint-cutters, road labourers), needle sharpeners, glass, china, and earthenware workers, potters, and flax-combers (Greenhow).

Post mortem the lungs are crammed with nodules, which are very hard and blackish, or at times grey, white, or yellowish. Histologically, we find lobular fibrosis, with narrowing of the alveoli and small crystalloid granules, which are strongly refractive to light, and composed of silica. Cavities, surrounded by grains of silica, may exist. The glands are hard and of a blackish-grey colour. Lesions of the right heart are common. The symptoms, as in anthracosis, may present three periods. The sputum appears more abundant, and hæmoptysis is more frequent. The disease lasts three or four years. The phthisis of earthenware-makers (Porté) shows three forms—pneumonic, emphysematous, and suffocating.

**Siderosis.**—Up to the present twenty-one cases of infiltration of the lung by particles of iron have been collected (Zencker and Merckel); they have been seen in workmen using red oxide of iron (looking-glass-makers, gold-beaters, and mirror-polishers). In one autopsy (Zencker) the surface of the lung was of an intense and uniform brick-red colour, streaked with darker lines, corresponding to the interlobular spaces. The pleura was covered with red patches. There were several cavities in the lung, without a trace of tubercles. The microscope showed fibrosis of the lung, with granules of iron, which, on chemical examination, gave its special reactions.

The physical signs resemble those of anthracosis, and the red sputum is characteristic.

The **diagnosis** is chiefly based on the characters of the sputum, which is black in anthracosis, red in siderosis, and without objective characters in chalicosis. The diagnosis from pulmonary tuberculosis and from latent cancer of the stomach is most puzzling. In the first case inquiry as to the patient's profession, and examination of the sputum from the chemical and bacillary standpoint will be the chief points. In the case of latent cancer of the stomach, where confusion is possible (Letulle), error can only be avoided by careful study of the course of the disease.

**Treatment** is, in the first place, prophylactic. Workrooms must be freely ventilated, attempts at preventing the propagation of dust must be made, and masks used. When pneumokoniosis is present, change of profession should be advised. This radical measure often arrests the disease. Revulsives, balsams, arsenic, and iodide of potash are employed for the fibrosis.

## VII. THROMBOSIS AND EMBOLISM OF THE PULMONARY ARTERY.

**Thrombosis** of the pulmonary artery is the obliteration of the vessel by a clot of blood formed during life. It has many causes, such as cachexia (tuberculosis, athrepsia, malaria), compression of the artery by a mass of glands, or by a mediastinal tumour. Thrombosis is sometimes consecutive to extensive pneumonia, gangrene of the lung, and pleurisy (Vergely). Atheroma and fatty changes in the vessel are exceptional causes.

**Embolism** is the sudden obliteration of the vessel by a body circulating in the blood: This body, or **embolus**, often arises from a thrombus. The results of embolism and the lesions to which it gives rise vary with the size of the artery obliterated, and also with the nature of the embolus. It is therefore customary to study embolisms of the large, medium, and small branches of the pulmonary artery. To this last category belong the capillary embolisms; to the two first, the lobular and lobar ones.

The large embolus, which is arrested in an artery of large or of medium size, is generally caused by an inert body; the embolism is then called mechanical. Capillary embolism may also be mechanical, but is more often infective and microbic.

### Mechanical Embolisms of the Pulmonary Artery.

**Pathogenesis.**—The thrombus may break up and give rise to emboli, which, however, are much more rare than those which have their origin in the heart or in the great veins.

1. Embolisms of **cardiac** origin are chiefly seen in mitral lesions, and in mitral stenosis in particular (Duguet). Aortic affections, on the contrary, rarely give rise to them. Bucquoy has laid stress on pulmonary embolisms consecutive to arterio-sclerosis; but as the affection is often accompanied by chronic myocarditis, this is, without doubt, a cause of embolism. In all these cases the process is as follows: On the walls of the right heart, and especially of the right auricle, masses of fibrin are deposited, and become interwoven with the muscular bundles which project into the cavity of the auricle. The slowing of the stream and the change in the blood cause coagulation. The clot breaks down gradually, and its fragments escape to form embolisms in the branches of the pulmonary artery. Sometimes the embolus consists of the débris of the valves and chordæ.

2. **Phlebitis** is a very frequent cause of embolism, especially in acute infectious diseases (typhoid fever, erysipelas, diphtheria, influenza, variola, etc.), because the clot develops rapidly, and its adhesion to the walls of the veins is very slight. A separate place must be given to pulmonary embolism,



following puerperal phlegmasia. This accident usually appears within three weeks of the confinement; beyond the fifth week puerperal embolism is exceptional.

Phlebitis in chronic infectious maladies (tuberculosis, cancer) and cachexia (malaria, diabetes, gout, etc.) is more rarely followed by pulmonary embolism.

Phlebitis consecutive to varices, fractures, or compression by tumour, may also give rise to embolism, especially if the lesion affect a vein in the lower limbs. In some cases phlebitis has a **deep origin**, as in phlebitis of the uterine and utero-ovarian veins (cancer of the uterus, uterine fibroma, cysts of the ovary, renal tumours, etc.), and yet in these different diseases embolism is to be feared.

**Pathological Anatomy.**—In order to understand the lesions which result from pulmonary embolism, we must bear in mind that the lungs receive two kinds of arterial vessels—the bronchial arteries, charged with the nutrition of the organ, and the branches of the pulmonary artery, charged with assuring hæmatisation. These two systems remain independent. The branches of the pulmonary artery, like those of the spleen and kidney, are **terminal** (Cohnheim)—that is, each vessel occupies its own proper area, and does not anastomose with its neighbours. The result is a complete independence in their function and their diseases. In the case of thrombosis there is, then, no reason to count upon the collateral circulation to remedy the effects of obstruction in the vessel.

When the obstruction affects a **lobar** artery or the trunk of the pulmonary artery, **anæmia** is seen, or in case of sudden death, **atelectasis** of the territory that is no longer irrigated is found post mortem. If, on the other hand, the patient has survived some hours, we find congestion, œdema, or perhaps an **infarct**, which may occupy nearly a whole lobe.

As a rule, the obliterated vessel is much smaller—in most cases a **lobular** artery. The obstruction then shows itself by the formation of an infarct, which is called hæmoptoic (Laënnec). These infarcts may occur all over the lung, but are more frequent on the right side than on the left, and usually occur at the base, the posterior surface, and the edges of the lungs. They are sometimes single, at other times multiple, when their number may be unlimited, just like the number of embolisms which give rise to them. They have a blackish, truffled colour, and a firm consistency, which permits their recognition by simple pressure, when they are situated deep in the parenchyma of the lung. Their cut surface is shining, dry, and smooth, or granular if the blood distends the alveoli. Around the infarct the lung tissue is bright red, passing to yellow as the distance from the infarct increases. Œdema of the lung and pleurisy, which is generally quite limited, are frequently found.

Under the microscope the alveoli are filled with red corpuscles, which are crowded together, and more or less deformed, according to the age of the infarct. The interalveolar spaces and the septa are packed with red corpuscles. In an old infarct the red corpuscles are no longer recognizable, and crystals of hæmatoidin and of hæmatin, pigmentary granules infiltrating the alveolar walls, and fatty granulations predominate. The connective network is always thickened. This thickening is sometimes but slightly marked, and as the arteriole again becomes permeable, *restitutio ad integrum* is favoured. Sometimes, on the other hand, fibrosis predominates and causes a fibrous cicatrix, which is retractile and prone to infiltration with calcareous salts (Pitres).

The infarct may be invaded by various micro-organisms, whence the possible coexistence of suppurative or gangrenous centres. In other patients the embolus (phlebitis in puerperal women) contains the micro-organisms of suppuration or of gangrene.

The chief factor in the production of infarcts is still surrounded by obscurity, in spite of experimental researches. Ranvier and Duguet have shown that some time elapses between the obliteration of the artery and the formation of the infarct. The interval between these accidents is sometimes as long as two or three days, and it is then supposed that the walls of the artery become inflamed on the proximal side of the obliteration, break down, and finally burst. The infarct is produced at this moment. This interpretation is to-day more generally admitted than that of collateral congestion (Virchow, Rindfleisch).

When the capillary embolisms (non-infecting) are few in number, they are not, as a rule, accompanied by any change in the lung tissue, because the circulation is re-established by the anastomoses of the capillary network. If their number is considerable, they may give rise to grave results from the many capillaries obliterated. Experimentally, capillary embolisms are easily caused by the injection of finely-divided substances into the jugular vein. As these substances are irritant to the lung tissue, they determine **pseudo-tuberculous granulations** at the point where they are arrested.

**Symptoms—Diagnosis.**—The large embolisms give rise to very dissimilar results. The patient may be struck down by syncope, and **die suddenly**; he may survive some minutes or hours. Intense dyspnœa, rapid cyanosis, and cardiac troubles (angina and palpitations) are seen, though examination of the heart and of the lungs reveals no lesion. Lastly, in some cases the patient is seized with intense pain in the side, severe dyspnœa, and perhaps a rigor. Improvement soon occurs, and he subsequently coughs up more or less profuse blood-stained sputum, proving an infarct. In this form of embolism recovery may take place, but the patient often succumbs in a few days, after several attacks, with signs of acute asystole.



The ætiological conditions have a capital importance in diagnosis. The large embolisms are due to phlebitis, while the medium ones usually arise in cardiac lesions. These same ætiological indications will, in the case of sudden death, and in the absence of an autopsy, justify the diagnosis of pulmonary embolism, and not that of **angina pectoris** due to aortic disease. Likewise the **asthmatic** attack, the sudden suffocations of uræmia, which arise under very different conditions, will hardly give rise to confusion. Embolisms of moderate size—that is to say, those which always end in the formation of **infarcts**—have a more clear clinical history than the preceding variety.

We can diagnose an infarct in a cardiac patient whose heart begins to fail if we find sudden dyspnœa and intense pain in the side, followed, some hours later, by the rejection of bloody sputum. The patient coughs up brownish or blackish viscid sputum, and not frothy blood, as in hæmoptysis. Auscultation, which is negative when the infarct is deep, reveals, in the case of a superficial infarct, a silent or tubular area, around which subcrepitant râles are heard. Friction sounds, due to dry pleurisy or to slight effusion, may be heard. The early râles sometimes become moister and larger, while the vesicular murmur reappears; sometimes cavernous breathing is heard when the infarct has been emptied through the bronchi, leaving a cavity, which will finally be filled up. In cases where some secondary infection is grafted on the infarct, so as to produce suppuration or gangrene, fever appears, and the expectoration takes special characters as regards appearance and odour.

In elderly people the discovery of sudden effusion, which is preceded by pain in the side, often finds its explanation in a **latent** infarct (Vulpian).

The repetition of complications and the coexistence of progressive cardiac weakness govern the prognosis.

We have already shown the means of distinguishing between hæmoptysis and the bloody expectoration due to infarct; in some cases, however, the infarct may give rise to true hæmoptysis. The rusty sputum of pneumonia is more viscid and aerated than that of pulmonary embolism, and again the clinical picture is quite different.

**Treatment.**—To prevent, as far as possible, the formation of emboli every patient suffering from phlebitis should be kept in bed four or five weeks. When embolism occurs, we must treat symptoms: cupping, inhalations of oxygen, and sinapisms, quiet the dyspnœa. The quantity of blood brought up is rarely so abundant as to be formidable. The means recommended for hastening the absorption of an infarct are practically futile. It is important to watch the condition of the heart, and to restore its tone by digitalis and caffeine.

### Special and Infecting Pulmonary Embolisms.

The name of special embolisms is given to the capillary obliterations due to inanimate foreign bodies, as opposed to microbic infecting embolisms. The first act mechanically ; the second, on the other hand, are endowed with vital properties, which cause suppuration, sloughing, and gangrene. The first group includes the débris of the fibrinous clot, broken up by disaggregation, as in old foci of phlebitis, fibrinous cysts in veins (Verneuil), and accumulations of cellular débris at a given spot (burns, frost-bite, certain intoxications). When the embolisms occur at many points at once, they may end in death by suffocation, unless they pass unnoticed.

More important are **fatty embolisms**, following fractures and osteomyelitis. Dejerine thinks that the increase of the intramedullary pressure consequent upon inflammation causes the little oil-drops of the bony marrow to enter the capillaries. These embolisms cause sudden and extreme dyspnœa. The injured man has air-hunger ; his face and limbs are cyanosed ; he sometimes brings up blood-stained froth, and rapidly succumbs, with or without convulsions. Innumerable fatty droplets mixed with blood are found post mortem in the vessels of the lung, and some authors therefore refer diabetic coma and suffocation in eclampsia to fat embolism.

**Gaseous embolisms** belong rather to the domain of surgery, and usually follow the entrance of air into the cervical veins. This accident shows itself by a characteristic whistling ; severe dyspnœa develops suddenly, and death usually follows.

The infective **microbic embolisms** are daily becoming more numerous as the life-history of micro-organisms becomes better known. Some reach the lungs in the débris of clots ; others make up the embolus alone. The *Streptococcus pyogenes* is the most common ; then come the staphylococci, coli bacillus, and a host of other aerobic or anaerobic micro-organisms, which reproduce *in situ* most of their original processes. Miliary abscesses, suppurative and gangrenous infarcts, admit no other cause. This variety of embolism will be studied under gangrene of the lung. Pulmonary tuberculosis and certain forms of pseudo-tuberculosis (tuberculosis due to *Aspergillus glaucus*, *fumigatus*, etc.), frequently spread by the circulatory system, and can be reproduced experimentally (Renon).\* The secondary cancerous nodules developed in the lung are due to embolisms of cancerous origin, just as the white pulmonary infarcts of leucocythæmia are due to accumulation of hypertrophied leucocytes, and as the pigmentary embolisms of malaria are due to the arrest of pigment granules in the capillaries of the lung.

\* See the chapter on Aspergillary Pseudo-Tuberculosis.



### VIII. GANGRENE OF THE LUNG—GANGRENE OF EMBOLIC ORIGIN—GANGRENE OF AERIAL ORIGIN.

We must first define the meaning of the word **gangrene**. Gangrene is not simply the death of a tissue (necrosis or necrobiosis); it is death, with which putrefaction or fermentation is associated. This fermentation is due to anaerobic organisms (Pasteur). Gangrene, therefore, is not simply the death of a tissue; it is the death of a tissue accompanied by putrid changes.

“Sometimes gangrene pervades an already necrosed area, in which case it is secondary; at other times necrosis and putrefaction of living tissues are caused by the same process, in which cases the gangrene is primary. Gaseous gangrene, symptomatic anthrax, noma, and some cases of pulmonary gangrene belong to the category of primary gangrenes.”

Two great processes are responsible for gangrene of the lung—gangrene of embolic origin, the germs reaching the lung in the form of emboli by the venous channels; and gangrene of aerial origin, the germs reaching the lung by the respiratory tract.

#### 1. Pulmonary Gangrene of Embolic Origin.

**Pathology.**—Whenever a purulent, putrid, or gangrenous centre exists somewhere in the economy (otitis, appendicitis, suppurative phlebitis, osteo-myelitis, etc.), this centre may give rise to specific emboli which end in the lung. Having reached the lung, the embolus provokes a like infection. If it contains germs of suppuration, the infarct suppurates; if it contains those of putrefaction, the infarct becomes putrid; if it carries the germs of gangrene, both these changes occur at the same time. Furthermore, purulent germs may sometimes start from an infected centre; at other times, those of putrefaction and gangrene, as in the examples quoted below.

Let us take otitis, which I have compared to appendicitis, as it resembles the latter affection closely, in that the infection is elaborated in a closed cavity and in the multiplicity of its complications. Whether the otitis be acute or chronic matters little; it may at a given moment give rise to suppurative or gangrenous lesions. The former, such as cervical abscess, meningitis, abscess of the cerebellum, or of the cerebrum, will be studied later. Pulmonary gangrene consecutive to otitis will be studied here, and in order to judge of its importance I give a résumé of cases.

**CASE 1.**—A young woman, who had excellent health in spite of a discharge from the ear for five years, was seized suddenly with sharp pains in the right ear, fever, headache, and vomiting. A week later she came into hospital: prostration, severe headache on the right side, some right otorrhœa, no mastoid pain on pressure, tongue dry, and breath foetid. Some days later intense pain under left breast, subcrepitant râles, high fever, acute dyspnœa, frequent cough, foetid sputum, albuminuria, and

death. Post mortem several gangrenous infarcts in right lung, and gangrenous pleurisy, with 15 ounces of fluid. The tympanic cavity, which was the starting-point of these troubles, contained a small quantity of foetid pus.

CASE 2.—A child, who for three years had discharge from the left ear, was suddenly taken ill with fever, mastoid pain, and vomiting. Brun opened the mastoid, and found foetid pus in the antrum. A diffuse gaseous phlegmon soon developed in the cervico-dorsal region; breath foetid; respiration panting; death. Post mortem thrombo-phlebitis of left lateral sinus, and embolic gangrene of lung, both consecutive to otitis.

CASE 3.—Boy, affected for three years with apparently benign discharge from the right ear. High fever, with intense dyspnoea, rigors, and pain in chest, supervened. Auscultation showed mischief at right base. Pains in chest became most acute; breath was foetid; temperature rose to 104° F.; death. Post mortem both lungs riddled with gangrenous centres of various sizes and miliary abscesses consecutive to otitis.

**Appendicitis**, like otitis (increased virulence of micro-organisms in a closed cavity), may also give rise to remote suppuration (liver, pleura, meningitis, etc.) and embolic gangrene of the lung. I shall here deal with gangrene of the lung.

I saw the following case at the Hôtel-Dieu :

Man admitted for sharp pain at the right base. No expectoration. Signs of pneumonia found at the painful region. Temperature raised. Patient also complained of sharp pains below the right ribs in the flank and iliac fossa. Palpation gave a feeling of thickening and fluctuation. We therefore thought of an abdominal abscess, with consecutive pneumonia. Patient transferred to surgical ward and examined under chloroform, but the abdominal effusion was not confirmed. Death two days later. On opening the abdomen we found a collection of foetid pus, with false membranes and adhesions. This collection was due to appendicitis. It started from the iliac fossa and tracked up below the liver, where a subphrenic pocket was found. At the base of the right lung a gangrenous centre as large as an orange. This centre was composed of two concentric layers—the outer reddish, the inner blackish, excavated, and foul-smelling.

The examples above quoted give an idea of embolic gangrene of the lung. I have chosen my examples from otitis and appendicitis, but many other foci (bone lesions, bed-sores, puerperal lesions, endocarditis, etc.) may end in the same result.

**Pathological Anatomy.**—This form corresponds to Laënnec's **circumscribed** gangrene, with the nearly constant addition of gangrenous pleurisy. The gangrenous centres vary from the size of a cherry-stone to that of an egg; they are mostly present in both lungs, and are multiple; some are superficial and subpleural; others are deep-seated. The subpleural centres have the conical shape of infarcts and a hæmorrhagic tint. An arteriole is usually seen at the apex of the cone. The deep centres have the shape of blackish nodes, distinct from one another. Gangrenous infarcts show the following forms: "Small, firm infarcts, which appear to be quite recent, show on section a yellowish caseous nodule, purulent and extremely foetid, in the centre of a brownish or greenish tissue engorged



with blood. The older infarcts have in their centre softened material of a chocolate-brown, surrounded by a scalloped collarette. Beneath the pleura, infarcts with a liquefied centre are found. The pleura is raised by gaseous, foul-smelling bullæ, forming little caverns, which do not appear to communicate with the bronchi. At its entrance into the infarct the arteriole is filled with blood and with bacteria in enormous numbers. At the centre of the infarct it has necrosed, and its wall is reduced to a shred. The bronchiole which accompanies it is flattened and full of desquamated cells and leucocytes (Guillemot).

The gangrenous cavity contains a greyish pulp, composed of the following elements: pus corpuscles, large cells infiltrated with fatty granules, masses composed of threads of connective or of elastic tissue and débris of capillaries, pigmentary granules, crystals of margarine, leucin, tyrosin, and various micro-organisms. All these elements are found in the sputum.

The cavity is surrounded by a first layer, which serves as a wall and limits the loss of substance. This layer is made up of débris of lung tissue, elastic fibres, and obliterated vessels, which are continuous with the next layer. The second layer is formed of lung tissue in the condition of grey hepatization. It is friable and sanious; the alveoli are filled with pus and large fatty cells, and the vessels are blocked by coagulated fibrin. The third layer, which is continuous with the healthy part of the lung, shows the lesions of catarrhal pneumonia.

## 2. Pulmonary Gangrene of Aerial Origin.

Gangrene of aerial origin corresponds to the **diffuse** form. It presents much the same changes as the circumscribed form, save that they are diffused and very extensive.

In certain cases the well-marked nodules of circumscribed gangrene and the irregular lesions of the diffuse form are found together.

Gangrene of the lung (circumscribed or diffuse) is, as a rule, accompanied by **pleurisy**, which may be gangrenous, purulent, or sero-fibrinous. Pneumothorax is not rare; it may result from perforation or from putrefaction. Lastly, particles of sloughs may pass through the pulmonary veins into the left heart and the general arterial circulation, and set up lesions due to capillary embolisms.

**Pathogenesis.**—Pulmonary gangrene of **aerial origin** may depend on various mechanisms.

Since the cavity of the bronchi, bronchioles, and alveoli are, as it were, the prolongation of the bucco-pharyngeal cavity, it is clear that microbes inhabiting this latter cavity may gradually reach and develop in regions where they do not normally exist. For their growth, however, the **soil must be prepared**; the individual must be in a **state of receptivity**.

For this reason this gangrene is not usually seen in robust people ; on the other hand, it is frequent in alcoholics, diabetics, and in sufferers from Bright's disease, malaria, mental disease, or chronic cerebral affections. It is sometimes met with in the course of acute infectious diseases—measles, variola, typhoid fever, etc.—or in subjects exposed to intense and prolonged chill (Bucquoy).

In other cases it may supervene as a complication in the course of various lesions of the respiratory tract—cavities of phthisis, cancer and sarcoma of the lung, chronic pneumonia, hydatid cysts, foreign bodies in the air-passages, perforation of the lung in chest wounds, foreign bodies carried in through such a wound (bullets, shreds of clothing, fragments of rib) ; opening of an abscess into the bronchi from the liver, spleen, kidney, etc. ; perforation of the œsophagus in cancer, whether spontaneous or due to forcible catheterization.

All the gangrenous or suppurative affections of the bucco-pharyngeal cavity—noma, diphtheria, bucco-pharyngeal abscess, laryngeal necrosis, operations upon the mouth and throat, removal of lingual cancer—may cause gangrene of the lung. It is probable in the latter cases that the obstruction to swallowing explains the entrance of infected particles into the respiratory passages, an accident also seen in lunatics, patients suffering from labio-glosso-laryngeal and diphtheritic paralyses.

A pulmonary infarct, resulting from non-infective embolism, may be invaded secondarily by micro-organisms.

As regards lobar pneumonia, although gangrene is an exceptional termination, and even denied by Laënnec, yet some cases are found (Grisolle, Andral, Bouillaud, Lanceraux). Gangrene is more frequent in lobular pneumonia ; but why in any given case does the infection of the lung tissue end in gangrene ? To answer this question the patient's general condition has been called in question. It is said that in the insane, diabetics, and alcoholics the infectious process, instead of tending to resolution, finds in the general state of the subject, or perhaps in the then existing constitution (Graves, Leudet), a reason for ending in necrosis and putrefaction of tissue. On this account an individual already weakened by typhoid fever, or suffering from an eruptive fever (scarlatina, measles, variola), would run a risk of the pulmonary inflammation ending in gangrene.

**Syphilis** sometimes determines gangrene of the lung.

Lastly, in certain cases a part must be assigned to overcrowding and to contagion, especially in hospital wards.

**Bacteriology.**—The microbic origin of this affection was foreseen as early as 1849 by Virchow, but it is chiefly to Leyden and Jaffé that we are indebted for the first serious work on this subject. Without attributing to the *Leptothrix pulmonalis*—a microbe derived from the *Leptothrix buccalis*—



as great an importance as these authors would do, we cannot hesitate to recognize that this micro-organism exists in most of the foci of gangrene, either alone or in association with the *Monas lens*, *Cercomonas* (Kannenberg), *Proteus vulgaris*, *Micrococcus tetragenus*, and many other aerobic and anaerobic microbes. We may note the *Bacillus ramosus* (Veillon and Zuber), the *Bacillus fragilis* (Veillon and Zuber), and the *Micrococcus foetidus*. "Upon the whole, in the centres of gangrene the aerobic flora is chiefly and sometimes uniquely represented by an almost constant species of streptococcus. In all cases this flora is less numerous than the anaerobic one. It may, indeed, be completely absent. The anaerobic species are, on the contrary, represented by various and very numerous kinds" (Guillemot).

**Symptoms.**—Gangrene of the lung is sometimes ushered in by special symptoms, such as extreme rise of temperature, acuteness of thoracic pain, and adynamia; but in other cases no hint may be given of the imminence of gangrene. I have seen two cases in which the course of events was as follows: An individual is taken ill with chills, nausea, and vomiting; cough, dyspnoea, and pain are next added to the gastric symptoms. Auscultation shows slight pleural rub, scattered râles, and tubular breathing, due to pulmonary congestion or to broncho-pneumonia. The fever is moderate, and the general condition without special significance. At the commencement the disease simulates an ill-defined infection, but the issue is not long in coming. The patient is seized with dyspnoea and fits of coughing, and if there is a communication between the gangrenous centre and a bronchus (this is **more rare** in the case of embolic than of aerial gangrene), he brings up abundant blackish sputum, which is mixed with blood and extremely foetid. It is then evident that a gangrenous focus has formed in the lung, and has just opened into a bronchus.

As soon as the communication between the bronchus and the centre occurs, the expectoration and the breath become horribly foetid, and **hæmoptysis** is not uncommon. The sputum is very abundant, diffiuent, greenish, or blackish; it contains blood, pus, mucus, fat, crystals of margarine, elastic fibres, balls of filaments, and micro-organisms.

When the expectoration is abundant, if collected in a measure-glass, it usually presents three distinct layers. The top is frothy and mucopurulent; the middle one, transparent and viscid, appears to be composed of thickened saliva; lastly, at the bottom of the sputum-glass we find yellowish or greenish balls, giving off an offensive odour, and constituting what are usually called Dittrich's plugs. According to Charcot, foetor of the breath may be lacking in diabetic gangrene; this feature is not absolute. When a gangrenous cavity forms, provided it is superficial or of sufficient extent, auscultation yields the signs of a cavity—namely, cavernous breathing, gurgling, and pectoriloquy. Gangrene of the **pleura**, which begins with

intense pain in the side, acute dyspnoea, and the signs of effusion, is often associated with gangrene of the lung. This aspect of the question will be studied under Putrid and Gangrenous Pleurisy. The existence of pneumothorax (by perforation or by putrefaction) is sometimes recognized. The general symptoms are usually marked—high fever, repeated chills, an earthy tint, loss of appetite, diarrhoea, and rapid wasting. Death usually ensues in gangrene of the lung. Recovery may, however, take place, especially in the circumscribed form, either spontaneously or after operation.

The **diagnosis** of gangrene from bronchiectasis has been already described. Gangrene should not be confused with **foetid bronchitis**, where the mucous membrane alone is attacked. Foetid bronchitis has special symptoms.

The mouth must be examined to make sure that the foetor of the breath is not due to some local affection, such as dental caries, ulcero-membranous stomatitis, gangrene of the tonsils, etc.

We might be tempted to make exploratory punctures for the purpose of finding the gangrenous and purulent focus. This procedure is, however, often uncertain; it is also dangerous, since hæmorrhage and infection of the walls frequently follow. Radioscopy and radiography may furnish very useful information as to the presence of the gangrenous and purulent focus.

The **treatment** of gangrene of the lung is very limited. The chief point is to support the patient's strength by means of tonics, quinine, wine, and alcohol. Balsams also find their indications. The patient should inhale vapour impregnated with carbolic acid.

The gangrenous focus should be opened up, when it is clearly limited and superficial. Cases of **pneumotomy** have, fairly frequently, resulted in cure, especially when the operation was performed at an early date.

## IX. BRONCHO-PULMONARY HÆMORRHAGE—HÆMOPTYSIS.

I shall describe in the same chapter hæmorrhages from the bronchi and from the lung. Hæmorrhage from the surface of the bronchi is called **bronchorrhagia**; that which occurs in the lung tissue itself—that is, in the alveolar cavities—takes the name of **pneumorrhagia**. This distinction is most often artificial. The term **pulmonary apoplexy**, which, by an abuse of language, was, and is still, used to designate hæmorrhage of the lung, should be abandoned.\* Spitting of blood after broncho-pulmonary hæmor-

\* The term **pulmonary apoplexy** was created by Latour in 1815, and adopted by Laënnec. This abuse of language was due to Rochoux, who in his writings on cerebral hæmorrhage had made wrong use of the term **apoplexy**, and employed it as synonymous with hæmorrhage. Several authors have protested against this faulty term—for example, Gendrin, who coined the word **pneumo-hæmorrhage**, and Trousseau, who called this lesion **infiltration of blood**.



rhage, or **hæmoptysis**, is only a symptom which serves to designate the rejection of blood which comes from the respiratory passages, just as hæmatemesis designates the vomiting of blood which comes from the digestive tract.

**Ætiology.**—Bronchial hæmorrhage (**bronchorrhagia**) is caused by congestion (strains, chills, hysteria). It is sometimes supplementary to a flux, or to an habitual hæmorrhage (menstruation, hæmorrhoids), and it is very often associated with pulmonary tuberculosis. According to circumstances, hæmoptysis precedes or accompanies the hatching of tubercles; later, it may be due to rupture of small aneurysms in the cavities. Bronchial hæmorrhage may be associated with enlargement of the arterioles which accompanies bronchiectasis; it very frequently accompanies the growth of hydatid cysts in the lung. Under interlobar pleurisy we shall see the cause of “interlobar hæmoptysis.” Hæmophilia and purpura are sometimes accompanied by hæmoptysis.

**Pulmonary hæmorrhage** (pneumorrhagia) is often passive (blood stasis), and due to diseases of the heart (especially mitral lesions). The occurrence of hæmorrhage in heart affections has been variously interpreted.\* Some invoke the mechanical obstruction to the pulmonary circulation by the lesions at the mitral orifice; others add to this cause the frequent hypertrophy of the right ventricle and the changes in the capillaries of the lung. A more recent theory attributes the hæmorrhage to an embolism which, starting from clots in the right auricle, reaches the small branches of the pulmonary artery, and, after the fashion of capillary embolisms, provokes a hæmorrhagic infarct.

The eruptive fevers (black small-pox), icterus gravis, etc., provoke hæmorrhage which appears to be connected with the alteration in the capillaries and a pathological state of the blood, named, for want of a better term, “state of dissolution.” The causes of the hæmorrhage associated with Bright’s disease are ill understood. In the new-born, pulmonary hæmorrhages connected with sclerema are seen (Hervieux).

In some cases it is difficult to differentiate the origin of one hæmorrhage from another: for example: the hæmorrhages consecutive to broncho-pulmonary lithiasis, syphilis, hydatids of the lung, gangrene, cancer, interlobar pleurisy, etc.

**Pathological Anatomy.**—The changes in bronchial hæmorrhage are superficial; the mucosa of the bronchi may be anæmic or congested, and coagula may be present in the bronchi.

The lesions of pulmonary hæmorrhage are more important. If the hæmorrhage, takes place by rupture, which is rare, the blood tears the lung

\* Atheromatous changes cannot be accused in this case, as they are extremely rare in the pulmonary vessels (Rokitansky).

tissue, collects in a **diffuse focus**, as in the brain, and may burst into the pleural cavity.

As a rule, however, the hæmorrhage proceeds in a different way. The blood infiltrates the alveoli and the parenchyma without much destruction, and the infiltration takes the form of conical nodules, which show a centrally directed apex, recalling the distribution of the bronchi and vessels. These hæmoptoic infarcts, which vary in size and number, are clearly circumscribed and deeply situated, as a rule, in the lower lobes. The cut section of the nodules is blackish and granular, like the hepatization in pneumonia, on account of the fibrinous coagula which make the infundibula prominent. The bronchi and the pulmonary vessels of the area near the infarct are obliterated by clots. The hæmoptoic nodule may undergo or provoke the following changes: It becomes indurated and retains its pigmentation; it passes into fatty degeneration; it causes secondary pneumonia at its periphery, or provokes limited gangrene. When it is subjacent to the pleura, it causes partial pleurisy.

**Symptoms.**—The chief symptom in **bronchial hæmorrhage** is **hæmoptysis**. When the hæmorrhage is violent, the blood pours out of the nose and mouth; it may also flow into the stomach, whence it is rejected by hæmatemesis. As a rule, the hæmoptysis is more moderate, and the patient coughs up bright, frothy blood, which in the spittoon has the appearance “of the foam produced in a basin when an animal is bled” (Trousseau). It may cease in a quarter of an hour or half an hour, and recur some hours later, next day, or on succeeding days. The last sputum coughed up has not this frothy and bright red appearance, but is black and viscid. It is the remnant of the hæmorrhage which has remained longer in the bronchi.

Hæmoptysis is sometimes sudden, and the patient is as much surprised as scared by his spitting of blood. Sometimes it is preceded by a feeling of warmth in the chest and throat; at other times it is preceded or ushered in by epistaxis. When supplementary to the menstrual flow, it is **periodic**, like the hæmorrhage which it replaces.

Bronchorrhagia has no physical signs. I am, of course, referring to signs directly related to the hæmorrhage, and not to those of the lesions which have caused the bleeding.

When the hæmorrhage is **pulmonary**, especially in cardiac cases, the clinical picture is very different. The blood which has infiltrated the lung tissue has not the same tendency to burst into the respiratory channels. Hæmoptysis is not, as in the preceding case, the chief symptom, but is often absent, and its characters distinguish it from hæmoptysis in tuberculosis. The sputum in pneumorrhagia is neither bright red nor aerated; it is blackish, viscid, and more or less mixed with mucus. The quantity of blood brought



up is much less abundant than in bronchorrhagia. The hæmoptysis may last from ten to twenty days, and Grisolles considered this persistence as one of the best diagnostic signs. The differential signs are, however, not absolute, since the blood of bronchorrhagia, if it has remained long in the bronchi, may resemble that of pneumorrhagia.

When the bleeding is abundant or persistent, the patient presents the usual symptoms of great hæmorrhage: pallor of the face, small pulse, tendency to syncope and to dyspnoea, which is proportionate to the extent of the lesion. If the nodules of pneumorrhagia are large and superficial, percussion reveals dullness, and auscultation yields blowing breathing and bronchophony. Broncho-pneumonia consecutive to pulmonary hæmorrhage is rare. Gangrene and perforation of the pleura are almost always fatal.

**Diagnosis.**—The diagnosis of broncho-pulmonary hæmorrhage has only one certain sign—that is, hæmoptysis. Given hæmoptysis, it is necessary to distinguish it from other hæmorrhages (epistaxis, stomatorrhagia, hæmatemesis) which somewhat resemble it. We must, therefore, trace the cause, and for this purpose we inquire into the characters of the blood coughed up, the symptoms accompanying it, and the circumstances in which it supervened. The larynx, the aorta, the lungs, and the heart are subjected to a minute examination.

**Cancers of the larynx** sometimes provoke profuse hæmoptysis.

**Aneurysms of the aorta** may open into the trachea or into the bronchi, and cause sudden death from hæmoptysis, or only slight bleeding, which recurs for several consecutive days or weeks. These cases are exceptional, but still they must be recognized, for both prognosis and diagnosis are involved. Spitting of blood, arising as a supplementary hæmorrhage, must not be taken for hæmoptysis of tubercular origin; nor must we forget that hysterical patients have congestion and hæmoptysis, which has nothing in common with tuberculosis.

The diagnosis between the hæmoptysis of pulmonary (tuberculosis) and that of cardiac origin (mitral lesion) is often made easy by the different characters of the blood and by the existence of the respective lesions in the lung or in the heart; but the matter is not always so simple, and hæmoptysis may appear as an **early sign** before the organic lesions are perceptible by our means of investigation. Furthermore, the distinctive characters of the bloody sputum are not absolutely rigid. Hæmoptysis which has the appearance of a bronchorrhagia has, nevertheless, been caused by heart disease, as Trousseau has clearly shown.

The diagnosis as to the nature of hæmoptysis has been greatly simplified since the discovery of the tubercle bacillus. Careful and repeated examination of the sputum or of the blood in hæmoptysis is performed, and

the presence of the bacillus sometimes allows us to state that the lesion is tuberculous.

Although there is not absolute agreement between broncho-pulmonary hæmorrhage and hæmoptysis, it is difficult clinically to separate them, and the gravity of the **prognosis** often betrays itself by the persistence or the abundance of the hæmoptysis. Fulminating hæmoptysis is caused by the opening of an aortic aneurysm into the trachea or into the bronchi, or by the rupture of a small aneurysm in a tuberculous cavity (Rasmussen's aneurysm). **Febrile** hæmoptysis in tuberculous patients is more grave than the apyretic form. The gravity of the prognosis depends also on the causes (pulmonary or cardiac lesions) which have produced the hæmorrhage.

**Treatment.**—Bronchial is more accessible to treatment than pulmonary hæmorrhage. The initial hæmoptysis of tuberculosis may be arrested by an emetic :

|                      |    |    |    |    |    |         |
|----------------------|----|----|----|----|----|---------|
| Ipecacuanha          | .. | .. | .. | .. | .. | gr. xv. |
| Tartrate of antimony | .. | .. | .. | .. | .. | gr. ss. |

Give at once, as soon as the patient is seized with hæmoptysis.

The expectoration of blood is sometimes arrested before the complete effect of the emetic. Ipecacuanha may then be administered  $\frac{3}{4}$  grain every half-hour, or every hour. Good results are obtained with a mixture of perchloride of iron in a dose of 30 grains in 4 ounces of water.

Iced and acid drinks are given, and a draught of the following composition is prescribed :

|                  |    |    |    |    |    |      |
|------------------|----|----|----|----|----|------|
| Distilled water  | .. | .. | .. | .. | .. | ℥iv. |
| Syrup of rhatany | .. | .. | .. | .. | .. | ℥i.  |
| Rabel water      | .. | .. | .. | .. | .. | ℥ss. |

A tablespoonful to be taken every four hours.

Pills of extract of thebaine,  $\frac{1}{3}$  grain in each, may be given hourly—from 4 to 10 pills in the twenty-four hours. Subcutaneous injections of ergotin may be used. Chloride of calcium, in drachm doses, may be prescribed. An ice-bag is kept *in situ* on the chest.

**Blisters** applied to the thorax or to the actual seat of the congestion, revulsives to the lower limbs, dry-cupping and sinapisms, may also be employed.

This treatment will be considered in detail in the chapter on Pulmonary Tuberculosis.

Digitalis finds its indications in hæmoptysis of cardiac origin.



## X. PULMONARY EMPHYSEMA.

**Definition.**—The name **emphysema** is given to the exaggerated dilatation of the pulmonary tissue by air. When emphysema is limited to the alveoli or the lobules, it is said to be alveolar or **intralobular**; but when the lobule is ruptured, and the air invades the interstitial tissue of the lung, the emphysema is **interlobular**.

**Pathological Anatomy and Mechanism.**—On opening the thorax, the lungs appear distended, and have but little tendency to collapse. The affected parts are greyish-white, crepitate a little, and have a downy feeling to the touch (Laënnec). Emphysema usually involves the apices and the anterior edges of the lungs. Spherical projections of different sizes, due to the emphysematous enlargements of the infundibula, are seen on the surface. The vessels of these parts are for the most part obliterated, and the circulation is deficient, while it is increased in the neighbouring parts, which become the seat of œdematous congestion. On microscopical examination of sections from an emphysematous lung which has been previously inflated and dried, the alveolar walls are often found to be atrophied and perforated. The atrophy of the septa and of the elastic tissue permits dilatation of the alveoli and of the lobules. The dilatation is at first limited to some alveoli or to an infundibulum; later, as the lesion spreads, the infundibula communicate with one another, and the dilatations, which are at first as large as millet-seeds, finally exceed a nut in size. In old people the apex of the lung is thus transformed into a lacunar tissue, in which the air circulates freely. How are the perforation of the septa and the communication between the infundibula brought about? “Under a still unknown influence.” The thinned septa are transformed; in their interior and on their surface they present ovoid masses of fatty granulations, which may come from the alveolar pavement epithelium or perhaps from the capillary vessels, and it is probable that this granular degeneration plays a large part in the perforations of the wall of the alveoli.

The **mechanism** by which emphysema is produced has been variously explained. Two orders of causes are admitted—**mechanical** and **trophic**. Sudden efforts of **expiration**, the fits of whooping-cough, the cough of croup and of broncho-pneumonia, dilate the alveoli to excess, and produce acute emphysema. It is therefore not surprising that the same causes if often repeated (asthma, chronic bronchitis), eventually cause emphysema by **mechanical means**. In other cases—in the aged, for example—**faults in the nutrition** of the lobule, atrophy and perforation of the alveoli, coincide so closely with the spread of emphysema that they appear to be the principal cause. It is therefore difficult to say exactly what part corresponds to

mechanical and what to trophic action, in the production of emphysema. In a good many cases this double process seems to exist. It is perhaps favoured by some special tendency of the lung tissue (heredity).

**Rupture** of the emphysematous vesicles is a grave complication. If the perforation involves the pleural cavity, **pneumothorax** results. If the rupture occurs under the visceral layer of the pleura, or in the interstitial tissue of the lung (interlobular emphysema), the air travels along the connective tissue, reaches the mediastinum, and invades the subcutaneous tissue of the neck, the thorax, and other regions.

**Symptoms.**—The shape of the thorax is peculiar. The chest is rounded and bulging in the clavicular regions, and when the emphysema is very extensive, the intercostal spaces appear dilated, and the chest is enlarged at its base. In the affected regions percussion gives a more ringing sound than in health. This resonance may encroach upon regions which are usually dull, so that the area of cardiac dullness is replaced by hyperresonance. On auscultation, the vesicular murmur is feeble, the respiration has a harsh tone, inspiration is shortened, and expiration is prolonged. The obliteration of vessels produces blood stasis, which may extend to the right ventricle. Dilatation of the ventricle and tricuspid incompetence sometimes follow, and troubles in the cardio-pulmonary circulation may come on and increase the obstruction to respiration. The emphysematous patient has many reasons for his difficulty in breathing. The rarefaction of the lung tissue, the depression of the diaphragm by exaggerated distension of the lungs, the insufficiency of expiration, and the diminution in the field of hæmotosis, lower the respiratory capacity by 50 per cent., or even more, as shown by spirometry.

Besides the continuous dyspnœa which is the result of the emphysema, we must also note **attacks of suffocation**, which often supervene, and are due to the various diseases, such as asthma, pulmonary congestion, or mitral lesions, that are so often associated with emphysema (Woillez). These various types of dyspnœa also exist when emphysema is associated with tuberculosis.

The **diagnosis** is easy, but we must not forget that emphysema, instead of constituting the whole disease, is often only an episode in the course of another malady (asthma, tuberculosis, chronic bronchitis), which must also be diagnosed.

**Treatment.**—The therapeutic indications are chiefly directed to the diseases which have produced the emphysema. As for the emphysematous lesions themselves, we have but little influence over them. Inhalations of oxygen and baths of compressed air are, however, generally recommended.



## XI. ŒDEMA OF THE LUNG—SUPERACUTE ŒDEMA IN BRIGHT'S DISEASE.

Œdema of the lung is due to the transudation of blood-serum into the alveoli and the interstitial tissue of the lung. Congestion is sometimes present as well, and plays a more or less important part. In cases due to nephritis the œdema far outweighs the congestion, while in cases due to cardiac lesions the congestion has the same importance as the œdema, and the lesion is called "congestive œdema," or "œdematous congestion of the lung."

On anatomical examination of the œdematous lung, congestion and atelectasis are found in association with œdema. The lung is heavy, and does not float. On section, abundant frothy, clear fluid exudes when œdema alone is the cause, but red-tinted when congestion is also present.

Many causes provoke œdema of the lung. It may arise suddenly, following thoracentesis, when the operator has made the mistake of withdrawing a large quantity of fluid too rapidly or too completely. The operation is scarcely finished when the patient is seized with suffocation, angina, and fits of coughing, and brings up a rosy, albuminous, frothy liquid, which is the result of superacute œdema. It is fortunate if this terrible accident does not end in death.

Congestive œdema of the lung is seen fairly frequently in the course of diseases of the heart, and during periods of asystole. Many persons suffering from ill-compensated mitral lesions are seized with dyspnœa, due to blood-stasis, elevation of the pulmonary tension, and resulting œdema. This congestive œdema chiefly occurs at the bases of the lungs. On auscultation, numerous fine moist râles are heard; tubular breathing is heard if pleural effusion accompany the œdema. In certain cardiac cases congestive œdema of the lung exists, to the exclusion of lesions in other organs. The lung, which is the first to receive the recoil of the circulatory embarrassment, becomes congested, œdema appears, and dyspnœa is the first indication of the cardiac lesion. In other cardiac cases the œdema is associated with congestion of the liver (cardiac liver), of the kidney (cardiac kidney), or with œdema of the extremities—in short, it forms part of the asystolic syndrome. Appropriate treatment may give good results, as we shall see in the chapter on Diseases of the Heart.

Influenza (Teissier, Rendu) and aortic lesions (Huchard) may lead to œdema of the lung, but the chief cause is **nephritis**,\* either acute or chronic.

\* Superacute œdema of the lung may supervene in the course of pregnancy. Vinay (*Lyon Médical*, 1897), who has studied this complication, rightly considers it due to nephritis gravidarum.

### Œdema of the Lung in Bright's Disease : Slow Form—Acute Form— Superacute Form.

Œdema of the lung is very frequent in the course of acute and chronic nephritis. In many cases of acute scarlatinal or of early syphilitic nephritis, or nephritis *a frigore*, œdema of the lungs may be associated with dropsy of other parts. Sometimes the œdema affects the bronchi rather than the lungs, and is the old "albuminuric bronchitis of Lasèque"; at other times the lungs are œdematous, especially at the bases, and auscultation records a multitude of fine râles. Œdema as slight as this scarcely merits the name of a complication; it forms part of the general dropsy, hampers the breathing, and brings on dyspnoea, but does not occupy the chief place in the course of the acute nephritis. The matter is very different in generalized œdema of the lung, and especially in the **superacute** form, which is a complication as sudden as it is terrible.

The same remarks apply to œdema of the lung in chronic nephritis. In a dyspnoeic patient with Bright's disease we often hear at the bases the subcrepitant râles of pulmonary congestion, which remains stationary for weeks and months. The patient who has attacks of dyspnoea and believes himself asthmatic is sometimes the victim of an error in diagnosis, and goes the round of the thermal cures in his search for the best treatment. In such a case the œdema is not of the first importance; it is more or less extensive, more or less obstinate, takes its share in the respiratory embarrassment, but does not focus all the attention upon itself. It is very different in the other variety of œdema—**superacute pulmonary œdema**—which may burst forth suddenly in the course of chronic nephritis, or more rarely in the course of the acute disease, and in a few hours endanger life. It is on this **superacute œdema**, then, that we shall fix our attention.

The following case gives an exact idea of it :

A man of forty-five was seized with such acute distress that he was brought to hospital at once.\* On admission, death appeared imminent: pale face, dull eyes, livid lips, bluish fingers and nails, hurried breathing, wretched pulse—such was the condition of the moribund patient. Any interrogation was impossible. The heart could not be examined, but both lungs were full of fine subcrepitant râles.

At first sight the condition recalled capillary bronchitis, suffocative catarrh, or acute phthisis. The diagnosis, although difficult, was not unpracticable. He brought up abundant frothy sputum, of a rosy colour. The eyelids were puffy, the legs slightly œdematous; the temperature was subnormal, and the urine very albuminous. The œdema and albuminuria pointed to nephritis; the characteristic expectoration and the fine râles in the lungs indicated superacute œdema.

The treatment was obvious. Charrier cupped the patient, and drew off 10 ounces

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\* This case is taken from my lecture on Superacute Œdema of the Lung in Bright's Disease, in which I have quoted a large number of cases (*Clinique Médicale de l'Hôtel-Dieu*, 1897, p. 23).



of blood. The effect was immediate. In less than an hour the breathing was easier, the expectoration was less profuse, the fine râles disappeared from the upper parts of the lungs, and death was averted. Strict milk diet and lactosed drinks were prescribed, and 5 teaspoonfuls of Trousseau's diuretic wine were given daily.

Next day the breathing was easy, the temperature normal. The heart was examined, and we heard a slight *bruit de galop*. Râles were no longer audible in the lungs except at the bases. The kidneys commenced to act; and the urine, though almost suppressed the day before, amounted to 8 ounces, but was markedly albuminous.

Two days later the patient was able to give us a complete history. He told us that for some time his health had been faulty, and he had suffered from pollakiuria, cramps in the calves, dead fingers, œdema of the eyelids and of the malleoli.

Treatment produced rapid improvement. In a few days the situation had totally changed. Respiration became regular, except that some râles persisted at the bases of the lungs. The puffiness of the face and the œdema of the legs had completely disappeared, and the urine amounted to 50 ounces, although albumin was still present and the depuration was imperfect, for the toxicity of the urine, as experimentally determined, was far from reaching its normal value.

**Symptoms.**—The first point of importance is the suddenness of this œdema. Reference to the cases which I have collected shows that superacute œdema does not, as a rule, result from bronchitis or some pulmonary condition which has gradually become more severe. The onset is nearly always **sudden** and unexpected; the unforeseen accident comes on by day or by night. One patient was seized with superacute œdema while I was asking him questions, and on auscultation, I felt, so to say, the rising of the liquid in his chest. The same sudden onset occurred in three of Giraudeau's patients. His first patient was seized while fast asleep, just as an attack of asthma comes on, although on the previous day she was quite well. His second patient was taken ill quite as suddenly in the night with superacute œdema during a period of apparently good health. The same remark applied to his third patient, who was stricken down two hours after dinner, although there was no premonition of such an accident. A similar thing happened to Bouveret's patients. In one of them superacute œdema appeared suddenly, and death resulted; in the other a first attack of œdema supervened, it is true, after a walk of three miles, but the two other attacks appeared without appreciable cause, when the patient was at rest.

I do not say, of course, that events always have this course. In some cases superacute œdema is preceded by such prodromata as cough, dyspnœa, or râles. In such a case it seems that the soil is prepared; it was so in my first case and in one of Giraudeau's cases. It is, however, an exception. Superacute œdema of the lung in Bright's disease is hardly ever the result of pre-existing broncho-pulmonary lesions. It bursts out suddenly, **like an attack of asthma**, although some hours previously no suspicion has been entertained.

A second point is that superacute œdema of the lung is very often an isolated result of Bright's disease, for, paradoxical as this may appear, it

is hardly ever associated with the severe manifestations of uræmia, or with the marked œdema of Bright's disease. Without knowledge on this point, it would appear that superacute œdema ought to supervene in the patient who is suffering from anasarca. This may be so, especially in acute nephritis. Giraudeau's patient was seized with superacute œdema of the lung in the course of acute nephritis with anasarca. This, however, is a rare occurrence. Nearly all the cases which I have collected show that the mischief appeared as an isolated accident in the course of latent nephritis.

It cannot be said, however, that this œdema attacks persons in *perfect* health. Close examination will show that they are more or less tainted with Brightism. If the "minor complications of Brightism" be carefully looked for, the evolution of an insidious nephritis may be constructed. A group of symptoms, such as the sensation of dead fingers, cryæsthesia, auditory troubles, cramps in the calves, pollakiuria, itching, electric shocks, and epistaxis, will be found in their past history. We shall see that they were not exempt from such a trace of œdema as puffiness of the eyelids and of the alveoli. We shall find that their arterial tension is high, their temporal arteries are tortuous, the second sound accentuated, and a gallop rhythm is present. We shall find that these patients were subject to headache, which they styled migraine; to suffocation, which they took for asthma; and to colds, which they regarded as due to ordinary bronchitis. If their urine is analyzed, albumin will be found; on testing the toxicity, it will be low. We shall find, in short, by an attentive and searching examination of this condition—which for a long time I have named **Brightism**—that the patient whose health appears to have been fairly good has in reality been suffering from mischief in his kidneys; the urinary depuration has been affected, and he has been exposed to the risk of complications which gave more or less warning.

After this digression as to the onset, let us resume our clinical analysis of the symptoms. The attack commences with a tickling in the larynx, jerky cough, and distress, which reaches its limit in some minutes, or perhaps some hours. These symptoms are due to sudden blocking of the pulmonary alveoli by sero-albuminous fluid. As a rule, the inundation begins in the bases, and may affect the whole of the lungs more or less rapidly. As the blood-serum transudes into the alveoli and the bronchioles under pressure, the patient is at once seized with fits of coughing, and then brings up the characteristic fluid, which is frothy and rose-coloured. Some patients may bring up as much as 2 or 3 pints in a few hours, or even more. It may be that the bronchi have not the power of expelling the fluid which has thus accumulated, and the asphyxia varies in indirect ratio to the amount of expectoration.

As soon as dyspnœa appears, innumerable fine subcrepitant and sibilant râles can be heard over both sides of the chest, testifying to the



inundation of the alveoli and of the bronchioles. According to the rapidity and the extension of this inundation, the râles invade the whole, or nearly the whole, of the lungs.

In proportion as the inundation increases, and if the expectoration is ever so slightly insufficient, dyspnœa increases rapidly ; the patient is pale, alarmed, and conscious of his extreme danger ; the pulse is small and quick, the lips are bluish, the nails livid, the limbs cold ; and the struggle may in a few moments end in death (fulminant form), in a day (rapid form), or in three or four days (slow form).

In some patients the condition is not absolutely perilous, so long as the sufferer can empty the inundated lungs. Paresis of the expulsive muscles may supervene, expectoration may be quite absent, and death from asphyxia occurs at short notice. In favourable cases the fluid is coughed up as soon as it forms, the inundation is arrested in time, and after a duration which varies from some hours to some days, the dyspnœa improves, the râles diminish, and the patient wins the struggle. We must, however, not be too sanguine, for the danger, though averted for a moment, may some hours or some days later recur and prove fatal. Sometimes convalescence requires several days ; at other times recovery comes on suddenly, and the patient is able to resume work on the next day.

Superacute œdema of the lung in Bright's disease is made worse by the fact that not only may the patient succumb to an attack in a few hours, but that, although his lungs have recovered, he remains liable to fresh attacks. Several patients have had two or three repetitions of superacute œdema, months or years apart. Giraudeau's first case had three attacks in two years, and Bouveret's second case had three attacks in six months.

**Diagnosis.**—The diagnosis of superacute œdema of the lung in Bright's disease follows from the description which has just been given. A patient with Bright's disease may have severe attacks of dyspnœa, but in this case it is a question of uræmic dyspnœa, which often exhibits the Cheyne Stokes type. It differs from that of pulmonary œdema in that we do not find râles and the characteristic sputum. Dyspnœa of toxic origin may, however, be combined with that due to œdema.

We see a patient who is a prey to the most acute dyspnœa, but it is a case of genuine asthma, and the diagnosis will be easy. The number of respirations is not increased, but rather diminished. Everything points to spasmodic dyspnœa ; inspiration is painful, expiration whistling and very prolonged. The prominent fact on auscultation is not the innumerable râles of pulmonary œdema, but the association of râles with signs of acute emphysema. Expectoration is absent, or composed of shreddy, mucous, or pearly sputum, and in any case the frothy, albuminous, rosy expectoration of superacute œdema is not found. Prognosis and treatment are quite

different, for the attack of asthma is never grave, and blood-letting is unnecessary.

I cannot review all the varieties of dyspnoea which may resemble that of superacute oedema. Such are the severe cases of dyspnoea in suffocative catarrh, acute phthisis, cardiac asthma, and angina pectoris. Let us not forget that we can always make a correct diagnosis in superacute oedema from the following symptoms: Sudden onset of the dyspnoea; fine râles over a large area of, or the whole, chest; incessant cough; abundant frothy, albuminous, rosy expectoration; frequent oedema of the face or of the legs; albuminuria; and previous symptoms of Brightism.

**Pathological Anatomy.**—In a case reported by Bouveret both lungs, from base to apex, were oedematous. The fluid literally streamed out on cutting and squeezing a lobe between the fingers. The bronchi were full of the same frothy rose-coloured fluid. The kidneys were small, granular, and cystic. The heart was enormous, the aorta much dilated, but not atheromatous; the sigmoid valves were healthy. The coronary arteries presented yellow patches of endarteritis.

In Huchard's case nephritis was found post mortem, with small granular kidneys, weighing together  $5\frac{1}{2}$  ounces, and pericarditis, with about 3 ounces of sero-fibrinous fluid in the pericardium. The lungs were so infiltrated with serous fluid that two pints of albuminous rose-coloured fluid were pressed out by gentle squeezing.

At the autopsy on Giraudeau's patient the lungs were bulky and violet-coloured. On section and on pressure a large quantity of frothy rose-coloured fluid, like that brought up in the expectoration, flowed out. The kidneys showed acute congestive nephritis; they were large and violet-coloured; the capsule stripped easily, and in places subcapsular hæmorrhages were visible. The heart showed general hypertrophy, which was most marked in the left ventricle. The valvular and arterial orifices were healthy. The aorta presented no trace of aortitis, either at its origin or in the arch.

Giraudeau observed the following lesions in the lungs of a young girl: The pulmonary alveoli were filled with coagulated fluid, which imprisoned desquamated epithelial cells and some red corpuscles. The capillaries of the septa were gorged with red corpuscles. It seemed that certain septa were thickened by a true interstitial oedema. At these points the bloodvessels were less apparent, as if the circulation had been impeded by the compression of the vessels.

**Pathogenesis.**—What is the explanation of superacute oedema in Bright's disease? The undeniable clinical fact is that patients with Bright's disease are liable to slight oedema in any part—in the lungs, the larynx, and the cellular tissue—just as they are prone to effusion into the serous cavities.



Why, then, this localization in the lungs? I do not know. As Brouardel remarks, from a medico-legal point of view, cold and alcohol appear to have been important factors in many persons who have died from this cause. A drunken man sometimes falls asleep in the open air during intense cold. He dies, and post-mortem superacute œdema of the lung is found, with lesions of nephritis, which are never wanting. Some authors (Huchard and Renaud) assign the principal, if not the only, part to the lesions of aortitis and periaortitis. According to Huchard, the needful intermediary between acute pulmonary œdema and Brightism is aortitis, and especially periaortitis, with its inflammatory or reflex reaction on the cardio-pulmonary plexuses. I am sorry to differ from this view. I do not deny, of course, that some aortic cases may have congestion and œdema of the lungs; but what I maintain, supported by undoubted evidence, is that superacute œdema of the lung has been found in many cases of Bright's disease in which neither aortic nor periaortic lesions were found during life or post mortem. The subject on whom Bouveret made an autopsy had neither aortitis nor periaortitis; Giraudeau's two cases showed neither of these lesions. My patient had neither aortitis nor periaortitis. Brouardel, who at the Morgue performed post-mortems on patients who died of superacute œdema of the lung, says that the aortic lesions are far from being constant, while the renal ones are never lacking. He noted, among others, the autopsy of a man who suffered from latent nephritis, and succumbed to superacute œdema of the lung. The autopsy revealed the **integrity of the aorta**, but showed the lesions of nephritis.

To the anatomical let us add the clinical proofs. Many patients with Bright's disease, whose cases I have reported, have neither before nor after their attacks of pulmonary œdema had any sign of aortitis. The question seems to me settled.

We do not know by what mechanism superacute œdema in Bright's disease is produced. I do not, however, reject the idea of pulmonary vascular troubles of vasomotor origin, the return circulation being for the moment annihilated, and favouring considerable hypertension in the afferent vessels.

Loeper blames the increase of the blood concentration; Widal the increase of the chloride of soda in the blood.

**Treatment.**—The urgent indication is bleeding. In spite of the coldness of the patient and the threatening collapse, which would at first appear as contra-indications, there must be no delay, and, without losing an instant, 10 to 15 ounces of blood must be withdrawn. The marvellous results of bleeding must have been seen to make its importance clear. I do not exaggerate in saying that it produces in the patient a visible change. In a case at the Necker Hospital œdema came on with such rapidity that death

would have speedily followed unless bleeding had been performed at once. The patient, who had not lost consciousness, told us that he felt himself dying and recovering in the space of a few moments. In my patient at the Hôtel-Dieu blood-letting had such a marvellous result that imminent death was arrested, and the râles which filled his chest from apex to base disappeared as by magic, leaving only a residue at the bases. Bleeding gave a similar result in Giraudeau's first case.\*

Of all methods of blood-letting bleeding is, without doubt, the most favourable ; but, in default of bleeding, wet-cupping over the chest may be made, or, better still, two dozen leeches may be applied. Dry-cupping of the thorax and of the lower limbs is also useful.

Subcutaneous injections of caffeine or ether are often indicated. We must remember, however, that the kidneys are inactive, and therefore use caffeine with caution. We may start with an injection of a grain, and be ready to repeat it several times during the following hours. Oxygen in large doses may also render some service. The patient's strength must be supported by milk and weak tea, with a little alcohol.

We must also know what to avoid. Blisters must not be used, for the patient has Bright's disease, and the action of cantharides on the kidneys will rapidly make the situation worse.

For the acute dyspnoea our thoughts turn to morphia, but caution is necessary, for morphia in such conditions may cause grave mishaps. As Brouardel says : " These are cases which it has been my lot to see fairly frequently. A patient begins to choke. It is evening, and one of the physicians on night duty is called. He gives an injection of morphia, following an only too common rule ; the patient gets no relief. A second and third injection at length bring quiet ; some minutes or hours later the patient succumbs, and the family blame the physician who has given the injections. Autopsy and inquest follow. It is found that death was due to superacute œdema of the lung."

Danger once averted, do not lose sight of the patient. Absolute milk diet must be prescribed, chloride of soda, which favours œdema (Widal), avoided, and the urinary secretion closely watched. In short, he must be treated as a case of Bright's disease, and be advised to avoid with the greatest care every cause of overwork and chill.

\* In my wards I have recently seen two more cases of superacute œdema, in which bleeding suddenly arrested asphyxia that was threatening the patient's life.



## XII. ON TUBERCULOSIS IN GENERAL—BACILLUS TOXINES.

## Local Tuberculosis.

**Nature of Tuberculosis.**—By his immortal work on pulmonary phthisis, Laënnec bequeathed to the medical world such an exhaustive description of this disease, with its lesions, its forms, and its signs, that we must all do homage to his genius.

On December 5, 1865, Villemin read a paper before the Académie de Médecine, and upset all previous theories as to the nature of tuberculosis. By numerous and well-conducted experiments, Villemin showed that tuberculosis is a **virulent, infectious, and inoculable disease.**

As soon as Villemin's discovery was known, inoculation of tuberculosis was repeated, and varied to infinity in France and abroad. The tuberculous material was introduced into the serous cavities of the pleura and of the peritoneum. Cohnheim practised inoculation, by means of a fine needle, into the anterior chamber of the eye, and, owing to the transparency of the cornea, was day by day able to follow the evolution of the tubercular process, and in some cases show the generalization of tuberculosis in the animal under experiment. Chauveau produced tuberculosis in the bovine species by mixing tuberculous material with their food, and observed in several cases that the intestine, which had served as the point of entrance for the virus, was much changed by the lesion. Tappeiner and Weichselbaum produced tuberculosis in dogs by making them breathe the powdered sputum of phthisical patients. Krishaber and myself have experimented upon the monkey, in order to deal with the animal which nearest approaches man, and in the numerous experiments made with my intimate and lamented friend I was struck at the autopsy with the severity of the infectious process. Three-quarters of our inoculated monkeys died of tuberculosis in a few weeks, while out of twenty-eight monkeys kept away from all contamination, and not inoculated, only one died of tuberculosis.\*

So great was the importance of Villemin's discovery that it completely changed our ideas of tuberculosis, and gave it a place in the list of infectious diseases. It had also the result of sanctioning, in irrefutable fashion, Laënnec's doctrine of the **identity and the unity** of tuberculo-caseous lesions, and, at the same stroke, destroyed the German teaching, which,

\* Our complete researches concern seventy-eight monkeys. Out of sixteen monkeys inoculated with human tubercle, twelve died with tubercular lesions; out of twenty-four monkeys which were not inoculated, but which lived with the inoculated monkeys, five died from tuberculosis; out of ten monkeys which were inoculated with purulent matter, only one died tuberculous; out of twenty-eight monkeys which were isolated from every source of contamination, only one died from tubercular disease (*Arch. de Physiologie*, Mars, 1884, No. 3).

under the eminent patronage of Virchow and Niemeyer, differed from the work of Laënnec, and looked upon **tuberculous infiltrations** of the lung as simple caseous degeneration, or so-called scrofulous phthisis. This latter part of the question, however, with its developments, will be treated in one of the following chapters.

**Bacillus of Tuberculosis.**—The infectious nature of tuberculosis being demonstrated, the question was to discover the microbe of the disease. The work of Pasteur and his **culture methods**, which had given such marvellous results in the study of other diseases (anthrax), served as a guide. The work was begun, and in May, 1882, Koch discovered the **bacillus** of tuberculosis.

This **bacillus** may be revealed in the sputum by the process described below.\* It forms a very slender, straight, or bent rod, in length equal to the third part of a blood-corpuscle—that is to say, 2 or 3  $\mu$ .

The bacillus is of uniform size throughout its whole length, but ovoid swellings, which may be due to the presence of spores, are seen. The small, colourless, oval vacuoles in the interior of the bacillus have also been looked upon as spores.

In order to stain Koch's bacillus, Ziehl's method is employed at the present day. It consists in leaving the slides for ten to twenty minutes in a solution of carbol-fuchsin which is prepared as follows :

|                  |    |    |    |    |    |           |
|------------------|----|----|----|----|----|-----------|
| Fuchsin          | .. | .. | .. | .. | .. | 1 gramme  |
| Carbolic acid    | .. | .. | .. | .. | .. | 5 grammes |
| Absolute alcohol | .. | .. | .. | .. | .. | 10 „      |
| Distilled water  | .. | .. | .. | .. | .. | 90 „      |

The preparation is then decolorized, either with a 30 per cent. solution of nitric acid, or by a mixture of five parts of absolute alcohol with one of nitric acid. In order more clearly to define the bacilli, the slide is stained a second time with methylene blue in a solution of water and alcohol. By this process the bacilli are stained red, while the tissue and other microbes are of a blue colour.

These characters distinguish it from all other micro-organisms. It is analogous only with that of leprosy.

The so-called pseudo-tuberculous, or acid-resisting bacilli, are more thick-set, and are less resistant to decoloration by acids, especially nitric acid.†

\* Some of the most purulent sputum is taken with a needle and spread out on a slide, which has previously been washed in diluted nitric acid, and afterwards in alcohol. A second slide is placed upon the first, and the two are rubbed together so as to form a film on each of the slides. The albumen is then coagulated by passing the slide three times through the flame of a Bunsen burner, or by placing upon it a few drops of a mixture of alcohol and ether in equal parts. The slides may then be put in the staining fluid.

† The tubercle bacillus is not the only one which remains stained by Ziehl's method after decoloration with acids. A group of other bacilli possesses the same property ;



The bacilli in tubercular sputum are usually free, and are rarely found enclosed in the leucocytes or the epithelial cells. They may be single or in pairs, or grouped *en masse*.

In histological preparations the bacilli are found *en masse* in the giant cells.

The discovery of the tubercle bacillus has been of the highest importance, for its presence is a sure sign of tuberculosis. The proof that the bacillus is indeed the active agent of tuberculosis, as the bacterium is the active agent of anthrax, is that the tubercle bacillus has been successfully **isolated, cultivated, and inoculated**. Koch, inspired by his culture methods, proceeded in the following manner: He took a small piece of tubercular material, and placed it on sterile gelatinized serum, which was then put in an oven at 99° F.

Roux and Nocard have introduced **glycerinated media**, which are an excellent culture material. Good results are obtained by the addition of 1 to 2 per cent. of glucose to the glycerinated media.

After twelve days or a fortnight the appearance of dry or scaly particles, composed of colonies of bacilli, is noticed. These colonies are whitish or yellowish, not shining, clearly isolated from one another, and but little adherent to the culture medium.

The tubercular particles, transported to another culture medium, reproduce new colonies of bacilli, and so on, through several successive cultures.

Bezançon and Griffon have extolled the use of glycerinated blood-agar and yolk of egg with agar, which give earlier and more certain cultures than other media.

From the point of view of **diagnosis**, the discovery of the tubercle bacillus has had considerable influence. We know how difficult the diagnosis often is between pulmonary phthisis and other diseases, such as bronchitis, chronic catarrh, chronic pneumonia, bronchiectasis, chronic laryngitis, pulmonary syphilis, hydatids of the lung, etc. In these different cases the presence of the bacillus in the sputum furnishes the proof of tuberculosis; and its absence, after several successive examinations, enables us to deny, in a more or less certain fashion, the existence of tuberculosis.

In cases where the search for the bacillus is fruitless recourse is had they are therefore called *acid-resisting*, or pseudo-tuberculous. They are met with in milk and in butter, and are found in plants, in earth, and manure. They may be pathogenic in animals. In man acid-resisting bacilli are found in the smegma, the cerumen, the nasal mucus, in certain affections of the eyes, of the uro-genital system, and of the lungs, especially gangrene. They may therefore give rise to errors in diagnosis. They can be distinguished from the tubercle bacillus by the following characters: They are not so resistant to decoloration by acids: 30 per cent. nitric acid, which does not affect Koch's bacillus, renders them colourless; they are more squat, grow in all culture media, and cannot produce tubercles by inoculation of animals (Creuzen et Villaret, *Revue de la Tuberculose*, 1903, p. 188).

to **inoculation** of animals—a procedure which gives most excellent results. The guinea-pig is usually chosen. A fragment of suspected tissue is inoculated under the skin of the animal's belly. A few days later a nodule appears, breaks down, and ends in a **tubercular chancre**. The neighbouring glands enlarge; the tuberculosis becomes general, and the animal dies one or two months later. To gain time, it is better to inoculate the peritoneal cavity, provided, however, pyogenic germs be not associated with Koch's bacillus.

The reaction is still more rapid if the suspected product be injected under the mamma of a female guinea-pig during lactation (Nattan-Larrier).

Every inoculated animal (guinea-pig, rabbit, dog) presents a blood reaction, characterized at first by polynuclear leucocytosis, and later by lymphocytosis.

In some special cases, in order to prove tuberculosis, recourse may be had to the reaction produced by the injection of **tuberculin**, which will be discussed in the chapter on Pulmonary Phthisis.

As regards **tuberculin**, I will content myself for the moment with pointing out the researches, the object of which is the extraction and the isolation of the soluble products secreted by Koch's bacillus. These toxines cause vaso-dilatation, convulsions, and necrosis, and play a considerable part in the symptoms of tuberculosis.

**Pathological Anatomy.**—The tubercular lesions, in whatever organ or tissue they be situated, present two chief forms—the one circumscribed and nodular (**tubercular granulation**); the other diffuse (**tubercular infiltration**). These two forms are frequently associated, and I would point out here that the anatomical peculiarities and texture of the tissue in which the lesions develop may modify their external appearance.

1. The **tubercular granulation**, which corresponds to the macroscopic unit, or the tubercle, properly so called, is a prominent rounded nodule, hard to the touch, grey and semitransparent when it is young, opaque and yellow when it is old. It is visible with the naked eye or with a lens; its size is variable, but it does not exceed  $\frac{1}{20}$  millimetre in diameter.

This **tubercular granulation** is formed of one or of several smaller parts, called **tubercular follicles**, which correspond to the microscopic unit, so that there may be ten, fifteen, or thirty follicles massed together to form the granulation.

The **tubercular follicle** (Schüppel), or **elementary granulation** (Malassez), or **tubercle** proper, has the following **structure**: When a section is made, we find at the centre a giant cell, containing several nuclei, which are most often set like a crown at the periphery. Its dimensions have gained for it the name of **giant cell** (Langhans). The giant cell is surrounded by a zone of fairly large cells, known as **epithelioid**, and by rounded closely-packed



cells belonging to the **embryonic** type—that is to say, having a nucleus which is large as compared with the protoplasm.

All these elements are bound together by a fibrillary or granular ground substance. The circulation is imperfect or absent in the granulations, for the vessels here, as in every tubercular product, are arrested in their development, or obliterated by endarteritis or capillaritis.

The manner in which the tubercle is formed in the tissues—that is, its **histogenesis**—is still disputed. According to Koch, the tubercle is formed chiefly by white corpuscles from the blood, the vessels, or the lymph. According to Metchnikoff and his pupils, the tubercle is really composed of leucocytes, and represents a defensive reaction against the bacillus. The epithelioid and giant cells of the tubercle are formed entirely at the expense of the phagocytes—that is to say, of the large wandering mononuclear leucocytes—and, secondarily, of the endothelial and connective cells. No epithelial cell ever contributes to the formation of the tubercle. “The tubercle is composed of a collection of phagocytes of mesodermic origin, which pass towards the spots where they find and envelop the bacilli.” According to Baumgarten, Ziegler, Cornil, Straus, Brissaud and Toupet, Kostenisch and Wolkon, the primary and fundamental lesion of the tubercle is a reaction of the fixed cells of the tissues, connective and also epithelial cells, which proliferate by karyokinesis, and become converted into epithelioid and giant cells, elements that are very rich in glycogen—a fact which clearly indicates their activity (Loeper). The lesion is only secondarily invaded by migratory leucocytes.

The bacilli are more or less abundant in the tubercular follicle; they are found in variable numbers in the giant cells and the periphery of the follicle. The follicle may be considered as the primary element; the follicles, by their aggregation, form the tubercle, and the tubercles, in their turn, by their aggregation, may form those large tubercles which may be as large as an egg, and are found in the lung, the brain, or at times in other organs. All these products have a common character (but it is not confined to them): they undergo caseous degeneration from the centre to the periphery. The central part of the follicle or of the granulation is infiltrated with fatty granules, becomes opaque, and this degeneration extends to the middle zone of large cells and to the external one of small embryonic cells. The tubercle now becomes yellow, softens, and tends to the formation of ulcers.

It must not be thought, however, that caseation is the only termination: the tubercle may become **calcified**; it may be converted into fibro-plastic tissue, and become **fibrous**—that is to say, harmless. Most often the tubercular change seems to begin in the small vessels or capillaries (tuberculous endarteritis); the vascular origin of the tuberculosis is, however, not absolute.

2. The **tuberculous infiltration** represents the **diffuse form** of tuberculosis. The follicles have no tendency to aggregate into granulations, but are spread out "like a sheet," and infiltrate the tissues, which become thickened, and sometimes take on a lardaceous aspect. These **tuberculous infiltrations** comprise: infiltration of the lung, which plays so large a part in pulmonary phthisis; infiltration of the larynx, confounded, quite wrongly, for several years with œdema; infiltration of the lymphatic glands, considered until lately as being of a scrofulous nature; infiltration of the synovial membranes, joints, and bones, which, under the name of fungous synovitis, white swelling, and caries, were too often regarded as of scrofulous origin.

The tubercular infiltrations described by Grancher may show the same changes as the granulations; they pass into the same caseous state, become softened, have a greyish or a yellowish colour, and when the caseous change is complete differential cellular elements are no longer found. The bacilli themselves are present, but only in very small numbers. Nodules and infiltration are often found together in the same tissue or organ, and are associated with the usual products of inflammation in different proportions, according to the soil in which they develop. Such are the different forms which tuberculosis may assume, and, in spite of the importance and of the value of numerous works, it may be asserted that pathological anatomy alone would often have been unable to affirm or to deny the nature of a tubercular product.

Recognition of the bacillus and inoculation with the suspected product allow us to **affirm** its tubercular nature. We can now distinguish between tuberculosis and changes which resemble it; scrofula will no longer encroach upon a domain which does not belong to it, nor will true bacillary tuberculosis be confounded with false tuberculosis.

It is convenient to mention the rare but undoubted association of cancer and tuberculosis in the same tissue. The old ideas as to the antagonism of tubercular and cancerous lesions must be abandoned, and hybrid formations admitted. Tuberculosis may be grafted upon cancer, but more often cancer is grafted upon tuberculosis.

**Local Tuberculosis.**—By assigning to tuberculosis some of the lesions which had been included under scrofula, the question of tuberculosis has latterly assumed a slightly different aspect. We find **local** manifestations of **tuberculosis** which are confined to the glands, the synovial membranes, the joints, the prostate, testicle, scrotum, ovary, uterus, nipple, skin, pleura, meninges, cerebellum, eyes, or tonsil.

Certain of these local manifestations appear in patients already suspected of pulmonary tuberculosis, and do not merit the name of "local," but others appear as an isolated manifestation. Some remain stationary,



without becoming general or affecting the lung; they may even be cured. Others attack the lung, and may be followed by slow phthisis or by acute tuberculosis. Many people suffer from suppurative adenitis, white swelling, or ossifluent abscess, which were formerly regarded as scrofulous, but are manifestly tuberculous! These folk were considered scrofulous, because the lesion appeared localized and curable, because their lungs were free, and because physicians held to the Louis law, which taught that pulmonary tuberculosis must be accompanied by that of other organs. This question must to-day be looked upon in a different light, and I refer, therefore, to the article on Scrofula.

**Tuberculosis in Animals.**—Monkeys, oxen, and pigs are very subject to tuberculosis. It is fairly common in the monkey; it is rare in the horse, sheep, and goat.

The carnivorous animals, such as dogs and cats, are not liable to tuberculosis.

**Tuberculosis in gallinaceous birds**—that is, **avian tuberculosis**—differs in several respects from the disease in man.

Cadiot, Gilbert, and Royet arrive at the following conclusions: "Avian tuberculosis, which is very frequent in the Gallinacæ, is inoculable in hens, pigeons, and rabbits; it is transmissible, but with more difficulty, to the guinea-pig; it may be seen in the ox and in man" (Kruse, Pansini).

"Tuberculosis of the Mammiferæ attacks mankind, the dog, the ox, and the horse; it is easily transmissible to the guinea-pig and to the rabbit, which, however, is perhaps less sensitive to it than to avian tuberculosis; it is inoculable in the parrot, and sometimes in the hen.

"Both poisons, therefore, attack the same animals, and the terms which designate them are not exact, since the so-called tuberculosis of Mammiferæ is also seen in parrots; and the so-called avian tuberculosis is not seen in all birds, and may sometimes be met with in the Mammiferæ. Parrots infected by human beings become, in their turn, a permanent centre of tuberculous infection."

The doctrine of the unity of human and bovine tuberculosis (Nocard, Arloing) has recently been attacked by Koch, who, in a famous paper read before the London Congress in 1901, attempted to establish, first, that bovine differs from human tuberculosis, as the latter cannot be transmitted to the bovine species; secondly, transmission by the milk or the flesh of infected animals is hardly more frequent than hereditary tuberculosis, which, according to Koch, is almost exceptional. These assertions have been opposed by Arloing, and during the last two years many works have been written refuting them (Ravenel, Spronck, Orth).

### XIII. PULMONARY PHTHISIS—COMMON CHRONIC TUBERCULOSIS.

The word **phthisis** is not synonymous with tuberculosis. It represents the ultimate or last phase of tuberculosis, and characterizes the consumption period of phthisiogenous lesions. These lesions vary. Some are clearly circumscribed—viz., the **tubercular granulation**, or tubercle; others are diffuse—viz., **tuberculous**, or **caseous infiltration**; but their nature is identical, and they are essentially tubercular. When the lesions are slow in growth and ulceration of the lung (**cavity**) occurs late, the patient dies of consumption. This essentially **chronic** form, which is the most common of all, is called pulmonary phthisis, without other designation. If the tubercular products grow rapidly and lead to early ulceration, the disease then takes the name of **acute phthisis**. Lastly, the tubercular granulations may be confluent, killing the patient before softening and ulceration have time to occur; in this case the disease is called **acute miliary tuberculosis**—that is, there is **tuberculosis**, but not phthisis.

**Pathological Anatomy.**—Lesions, which vary according to their form and their age, are found in the lungs, especially at the apices: tubercular granulations and infiltrations, ulcers, cavities, interstitial pneumonia, bronchial dilatations, pleural adhesions, etc., may be present.

We find in the lung, as elsewhere, two chief forms—the granulation or the infiltration—which may be isolated or combined.

**Tubercular Granulations.**—The tubercular granulations in the lung show two slightly different forms—the grey granulation and the miliary tubercle.

The **grey granulation** (so named by Bayle) is a small, hard, prominent, rounded, transparent nodule, that is much smaller than the miliary tubercle. It is sometimes only visible with the lens, and it does not exceed  $\frac{1}{20}$  millimetre in diameter. This greyish granulation may take on a yellowish tint, from degeneration of its elements; it is composed of tubercular follicles, which have been described in the previous chapter. The grey granulation may arise in several spots, but its seat of election is the wall of the blood-vessels and of the lymphatics. It is especially found in the case of **acute miliary tuberculosis**, where it is confluent and generalized through both lungs, while it is more rare in pulmonary phthisis. The **miliary tubercle** (Laënnec) is the form found in chronic phthisis, and is, as a rule, localized the apex of the lung, invading later the lower regions.

The **miliary tubercle** is about as big as a millet-seed: its diameter varies from  $\frac{1}{2}$  to  $1\frac{1}{2}$  millimetres; it is prominent, rounded, greyish, and semitransparent—at first yellowish and opaque, when it is degenerating. Like all tubercles, it is adherent to the neighbouring tissues, and cannot be removed without tearing away particles of them.



The description of the little tubercular granulation does not quite apply to the miliary tubercles, which have a more complex structure, because "the contents of the alveoli and of the bronchioles, and the neoplastic infiltration of the connective tissue of the walls of the bronchi and the vessels, take part in their formation." The miliary tubercle appears to have a well-defined origin; the tubercular follicles, which take the chief part in its formation, develop, according to Rindfleisch, around the terminal intralobular bronchiole, at a point where it becomes acinous and joins the alveolar ducts of the acinus. Such is said to be the origin of the **miliary tubercle**, named by Charcot the **peribronchial tubercular nodule**. It is necessary to add that slightly larger bronchioles may be the centre of formation of the tubercle.

This little mass assumes various shapes. Sometimes it takes that of a crescent, and does not completely surround the bronchiole; at other times it leaves one of the segments free, or sheathes the bronchus like a muff, and the diffuse tubercular elements only present at distant intervals "the agglomerated and circumscribed form, usually regarded as the characteristic of the tubercle." From the bronchus, the tubercular infiltration gradually reaches the neighbouring alveoli, and thus the tubercular nodule arises. Embryonic infiltration occurs in the walls of the bronchi and of the alveolus, while the mucosa of the bronchus, affected by inflammation, loses its epithelium, and its cavity is filled with large cells, of which some show fatty degeneration; in other words, we find tubercular peribronchitis and catarrhal bronchitis. Moreover, **obliterating endobronchitis** is met with, the tubercular nodules projecting into the interior of the bronchus, and finally obliterating its lumen.

The tubercular granulations which develop in the walls of the alveoli have the following disposition: A granulation always envelops several rows of alveoli, and the extension of the tubercular agglomeration takes place by cellular infiltration of the walls and the cavities of the alveoli. The endothelium of the alveolus undergoes changes analogous to those of catarrhal pneumonia. In the midst of this tubercular invasion the walls of the alveoli remain, and form, as it were, the sketch of the tubercular mass, while the elastic fibres still indicate the skeleton of the alveolus.

The bronchial vessels dilate and may become varicose, while progressive obstruction by embryonic proliferation occurs. The condition is really an **obliterating endarteritis**, or **capillaritis** (Martin).

**Tubercular Infiltration—Caseous Inflammations.**—Granulations and miliary tubercles do not form the chief lesion: we first notice a substance which may be greyish and semitransparent, or yellowish and opaque, or at times amorphous and, as it were, infiltrating the pulmonary tissues, though it more often forms centres of variable dimensions. Laënnec called this

**tubercular infiltration**, and it has since been named **caseous material**, because of its resemblance to cheese (**caseum**). It is composed of crystals of fatty matter, epithelial cells, and more or less degenerated lymphatic corpuscles. What is the nature and origin of this caseous material?

Speaking generally, tubercular granulations, whatever be their situation (pleura, peritoneum, meninges, or lungs), cause a surrounding zone of inflammation (Hérard and Cornil). If tuberculosis develops upon the walls of the bronchi, the lesions of endobronchitis result; if it affects the pulmonary alveoli, the changes of catarrhal pneumonia, as well as the embryonic infiltration, result.

Is the caseous substance due to metamorphoses of the ordinary inflammatory products? or is it the remains of broncho-pneumonia arising by contact with the tubercle? or, indeed, is it not a tubercular infiltration, just as the miliary tubercle and the grey granulation are?

Far too much importance has been assigned to the pneumonic products; indeed, the products of ordinary inflammation are not wanting in essentially tubercular lesions, but they are only of secondary importance. The infiltrations called caseous are true tubercular infiltrations, which have undergone the degeneration common to a large number of tubercular productions. Some caseous masses are simply an agglomeration of miliary tubercles, and we shall see later, when dealing with "caseous pneumonia," that the tubercular infiltrations, which are apparently diffuse, are really composed of a more or less confluent aggregation of tubercular products. The tubercular products, which are present either in a circumscribed form (granulation, miliary tubercle) or in an apparently diffuse and infiltrated form, are therefore only the result of a specific inflammatory process; and ordinary phthisis has for its most common lesions **the isolated or aggregated miliary tubercle and chronic tubercular broncho-pneumonia**.

It is to be noted that all these inflammations of tubercular nature and origin are destined to undergo the same process: The vessels, primarily or secondarily invaded (endarteritis, capillaritis), become obliterated; the inflammatory products, which are greyish at first, become opaque, yellow, and caseous; they break down, pass into a neighbouring bronchus, and leave an **ulceration** in their place.

There is, however, a process which tends to cure—viz., the **cretaceous**, or **fibrous**, change in the tubercular products.

**Pulmonary Cavities.**—The formation of a cavity is preceded by **dilatation of the neighbouring bronchi**. This dilatation leads to the destruction of the walls, which lose their resistance; also, when the softened tubercle empties itself into the bronchus, the latter is already dilated, and forms part of the excavation. The primary excavations which result from the breaking down of the miliary tubercles might be called **acinous cavities**;



the union of several acinous cavities would form **lobular cavities**, which, in their turn, would form **lobar** or large cavities.

**Cavities** vary in size from a pea to a nut, or larger. They are often anfractuous, divided into compartments, and traversed by bands of connective tissue and by vessels which have resisted the ulceration. The cavity is lined with a membrane formed of embryonic tissue and granulations. This surface secretes a purulent liquid, rich in toxins, and their absorption leads to hectic fever. The surface of the cavity is sometimes converted into cicatricial tissue; this change may end in cure. The cavity may be filled with blood. **Aneurysmal dilatations** of the pulmonary artery at times project into the cavity, and may rupture, causing fatal hæmorrhage. The shell of the cavity, which is rich in fibro-plastic tissue and granulations, is deprived of pulmonary blood (functional blood), but vessels which communicate with the bronchial arteries (nutritional blood) are present in it (Natalis Guillot).

The **localization** of the tubercular products and their extension in the lung take place in the manner so graphically described by Laënnec: "We very often find in the same lung evident proofs of two or three successive eruptions, and we can then nearly always make out that the primary eruption at the apex of the lung has already become excavated; that the second, situated around the first, but a little lower, is formed by tubercles which, in most instances, are already yellow, though still small; that the third, formed by grey miliary tubercles, with some yellow points in the centre, occupy a still lower zone."

The secondary eruptions are not confined to the lung; similar lesions develop in other organs at the same time.

The lesions of the lung are not always incurable; they may end in **recovery** by calcification or by fibrosis. In persons who have died from some other disease, and especially among the elderly, old tubercular masses are found, varying in size from a pin's head to a pea, and converted into calcified tissue.

**Fibrous** change is also a method of cure which may occur in the granulations and cavities. Fibrous granulations were clearly noted by Cruveilhier, who called them curative; they have lately been carefully studied. The granulations, both in chronic and acute tuberculosis, may pass into the fibrous state. The walls of the cavities may also be surrounded by a fibrous zone, continuous with the fibrous bands in the lung, and part of the apex may thus be converted into fibrous tissue.

**Pleura.**—The changes in the **pleura** (dry pleurisy, or pleurisy with effusion, adhesions, perforation, and pneumothorax) occupy an important place in the history of pulmonary phthisis. The pleurisy is sometimes encysted and apical, interlobar, or diaphragmatic. Often the pleurisy is

dry (false membranes and adhesions) ; in some cases there is a considerable effusion of an extremely variable nature, and the fluid may be serous, sero-fibrinous, hæmorrhagic, or purulent. The more or less adherent false membranes which line the pleura are composed of fibrinous lamellæ, separated by masses of round cells. The superficial layers contain no bacilli. Giant cells are met with, as well as cellular masses and bacilli in the deeper layers, and the vessels of the pleura often show numerous bacilli in their walls. Bacilli are sometimes found in the lymphatic spaces. The granulations are situated on the surface of the lung, or in the pleura.

Tuberculosis of other organs will be studied elsewhere. Tuberculous adenopathy of the **axilla** is sometimes seen in pleuro-pulmonary tuberculosis.

**Bacteriology.**—The tubercle bacillus has been already described. I shall therefore only refer to it briefly. **Bacilli** are found in all tubercular granulations, “between the fibrillæ and the coagulated fibrin, in the interior of the alveoli, and in the thickened connective tissue of the septa ; they are especially numerous at the points where the cells become caseous or granular, and where it is difficult to distinguish the limit of the alveoli—that is to say, in the central parts of the tubercles.” Bacilli exist in numbers in the intravascular clots, in the altered walls of the vessels, and in the perivascular connective tissue.

They are also found on the surface of the pulmonary cavities, but are always associated with other microbes. A veritable flora develops in the walls of the cavities, and we find streptococci, staphylococci, zooglœæ, sarcinæ, the *Proteus vulgaris*, the *Proteus mirabilis*, the bacillus of green pus, etc. These pathogenic agents play a very active part. Some cause supuration and necrosis ; others, by their **toxines**, produce septicæmia and hectic fever ; and, lastly, they all hasten and complete the work of destruction commenced by the bacillus. Many observers, having recognized the streptococcus in the blood of phthisical patients, have asserted that it plays a great part in the production of hectic fever. Straus has shown that this assertion is exaggerated.

**Symptoms.**—Pulmonary phthisis is often preceded by prodromata of long duration—viz., repeated attacks of laryngitis or repeated bronchitis ; pleurisy, which is sometimes of long standing, or which has recurred several months or years apart ; hæmoptysis, which has arisen without appreciable cause, while the patient was apparently in good health. Many patients complain of what they call “a neglected cold,” or have suffered for months from bronchitis, and have never regained their health. They are often anæmic ; they cough, grow thin, lose strength, and bring up sputum streaked with blood ; they easily get out of breath, and suffer from chronic hoarseness and loss of voice ; the appetite is bad, dyspepsia is present, and at times accompanied by regurgitation or vomiting.



On auscultation, we find signs of commencing tuberculosis, such as some roughness on inspiration (Grancher); some dry crackling, which is limited to one apex, or very slight impairment of resonance at the same level. The expectoration is examined, and the tubercle bacillus is often found in it.

At this early period the disease may remain stationary for an almost indefinite period, may improve, or be cured, but more often it pursues its way. Its course, though slow as a rule, is sometimes interrupted by acute attacks of laryngitis, bronchitis, fever, or slight hæmoptysis, and the patient only too often enters the second stage of the disease.

During this period of onset the nutrition has suffered but little: the appetite has been fair, and wasting has not made rapid progress; but if the disease continues to develop, the lesion extends and softens, large moist râles appear, the lung tissue ulcerates, the expectoration becomes muco-purulent, and the mischief is no longer limited to one side, but begins to invade the other lung. In spite of the gravity of this condition, improvement and cure are still possible.

Later, sometimes after many years, **consumption** appears, and sets its seal on the body. The patient has a special facies: his cheeks and temples become hollowed, with a tinge of colour over the malar bones; the eyebrows and eyelashes grow long; the conjunctiva takes on a bluish tint; the fingers become clubbed. The symptoms present comprise fever, profuse sweats (especially during sleep or on waking), vomiting of food, palpitation of the heart, pain in the side (intercostal neuralgia or pleurisy), vocal troubles, and dysphagia (laryngeal phthisis). The fever rises every evening (hectic fever); diarrhoea is frequent (cachectic diarrhoea, or intestinal tuberculosis); the wasting is extreme; the feet are swollen (cachectic œdema); and the tongue is sometimes covered with thrush. In the midst of this general break-up the intellectual faculties are usually intact; illusion is sometimes complete, and it is in this state of consumption, with death at hand, that the patient, trusting in recovery, or believing himself affected by a benign bronchitis, gives himself up to the happiest thoughts.

Such is the course of pulmonary phthisis, when it has not been arrested by a curative process. Let us now study in detail the signs which correspond to the three periods of its course.

During the **first period** we find dullness at one apex; on auscultation, rough breathing, with prolonged, jerky expiration, is heard in the supra-spinous fossa or in the clavicular region, together with dry, subcrepitant râles (**dry crackling**) or sibilant ronchi. These last signs are, however, often absent, and some dry crepitations may be only heard at a limited point after the patient has been made to cough.

The lesion, at its commencement, often appears to be confined to the

most external part of the supraspinous regions ; in many cases I have found slight dullness and dry crackling at this point—*i.e.*, almost upon the shoulder.

During this period, which may be of very long duration, many patients scarcely consider themselves as invalids. They cough, but bring up little expectoration ; they have little or no fever. They believe themselves affected by simple bronchitis, and make no change in their manner of living, until hæmoptysis gives them warning, or an aggravation of symptoms, that must be reckoned with, supervenes.

In the **second period** the lesions are more extensive : the tubercular tissue **softens** ; the ulceration of the lung commences ; the expectoration is more abundant ; the sputum is nummular, and often mixed with blood ; fever may appear ; the appetite fails, and wasting begins. Percussion shows more extensive dullness ; on auscultation, mucous râles are perceptible ; the dry crackling of the first period has become **moist** ; the breathing is rough and blowing, and bronchophony is heard. Pleural friction sounds are often heard in various regions, and tubercular lesions often appear in the apex of the other lung. Laryngitis and tracheo-bronchitis are frequent. The patient always puts them down to a chill or to some imprudence ; as a matter of fact, they are tubercular.

The lesions end in the **third period**—that is to say, **ulcers and cavities in the lung**. The signs of cavity are then evident, their intensity being in proportion to the size of the cavity. If the cavity be only in process of formation, a single sign may exist alone—namely, gurgling. If the cavity be of sufficient size, new signs are evident—namely, on percussion, the cracked-pot sound ; on auscultation, gurgling, cavernous breathing, and pectoriloquy. If the cavity be very extensive, it may give the same signs as pneumothorax : amphoric breath and voice sounds, and metallic tinkling. This period corresponds to the term “ **phthisis**,” or consumption with hectic fever, profuse sweats, extreme wasting, and cachectic œdema.

I must now dwell at greater length on some symptoms, such as expectoration, fever, and hæmoptysis, which are of great importance.

The **expectoration**, which has no special characters at the onset, becomes gradually thick and opaque ; the sputum is homogeneous and nummular (from its resemblance to the shape of a coin) ; it floats in a clear liquid. During the last period the expectoration is quite puriform (liquid from cavities). Nummular sputum is also seen in the bronchitis of measles or of influenza, and in bronchiectasis. In order that muco-purulent sputum may take the nummular form, it is sufficient to expectorate into a sputum-glass containing water. The sputum of phthisical patients contains a large amount of phosphates and of chloride of soda.

Under the **microscope** pus corpuscles, epithelial cells, and elastic fibres,



all indicating destruction of the lung parenchyma, are seen in the sputum. The **microbes** met with in the sputum are very numerous; I may mention the staphylococcus, the streptococcus, the pneumococcus, the pneumobacillus, the *Bacillus pyocyaneus*, sarcinæ, leptothrix, and micro-organisms which colour the sputum green, etc.; but the important microbe is Koch's bacillus. In the preceding chapter I have given a description of this bacillus, and the means of detecting it in the sputum, so I shall not reproduce it here. We shall see later that the presence of Koch's bacillus in the sputum is sometimes the only positive proof of tuberculosis. The bacillus may exist in the sputum from the onset of pulmonary tuberculosis; it is, however, more abundant a little later. As a rule, the gravity of the disease is in proportion to the number of the bacilli.

**Fever.**—Fever is of very great importance, for it is the **almost certain index of the gravity of the prognosis**. Febrile tuberculosis is always grave, while the non-febrile form may go on for an indefinite period without compromising life. Fever usually shows itself during the second period, with softening of the tubercles or with broncho-pulmonary inflammation; it may, however, appear at the onset of tuberculosis. Some patients, indeed, have fever before the apparent onset of the lesions; in their case it may be said that the fever comprises, for a time, almost the entire disease, and may therefore be called pretubercular fever.

The early fever in tuberculosis is due to successive crops of tubercles, and also to the special virulence of the bacillus and the poisoning of the economy by tuberculin, so that the patients in question are rather **tuberculinized than tuberculized**. It is certain, I repeat, that every tuberculosis which is febrile at its onset is very grave; every hæmoptysis which is febrile is very serious; fever is an unfavourable element in the prognosis.

The symptoms of fever are rise of temperature, abundant perspiration, and an **acceleration of the pulse**, which may reach 100 or 120 beats, while the temperature may be but little elevated. The local temperature on the diseased side is higher than at a symmetrical point on the healthy side.

At a later stage, during the period of softening, with ulcerations and cavities, the fever grows worse; it is intermittent, usually begins about five or six o'clock in the evening, and ends during the night with profuse sweating. The attack is sometimes ushered in by a feeling of cold, and the thermometer frequently reaches 104° F. This variety is **hectic fever**, which is usually accompanied or followed, at a more or less early interval, by diarrhœa, wasting, and consumption—in short, phthisis.

The fever of the advanced period of tuberculosis is due, not only to the causes already mentioned, but also to the association of other microbes which give rise to most complicated infections. The cavities and caseous

centres are invaded by an army of microbes ; they establish themselves in the conquered country, and produce secondary infections, which are largely responsible for the symptoms of this period.

**Hæmoptysis.**—Spitting of blood is one of the most important symptoms in pulmonary tuberculosis, and causes the patient the most distress. It may supervene at any period of phthisis, but it is usually an early symptom, or appears late in the stage of cavity.

Early hæmoptysis shows different forms. Sometimes the patient brings up only sputum tinged with blood ; at other times the fits of coughing are very violent, and he brings up bright red frothy blood, which in the sputum-glass has the appearance “ of the foam that is present in a basin into which an animal has been bled ” (Trousseau). The duration is variable : it may cease after a quarter of an hour or half an hour ; it may reappear some hours later, on the next day, or on the following days. The last sputum brought up has not a bright red, frothy appearance, but is viscid and dark, and represents the remains of the hæmorrhage which has remained longer in the bronchi.

In some cases hæmoptysis is from the outset very profuse. The patient starts coughing ; warm fluid rises in the throat, and the blood gushes forth in such quantity that the patient does not appear to spit, but rather to vomit, blood.

Hæmoptysis may supervene without prodromata, and the patient is as surprised as he is frightened by his spitting of blood. Sometimes it is preceded by oppression, by a feeling of warmth in the chest or tickling in the throat, puffiness of the face, and epistaxis. Before or during the bleeding, and as long as the hæmorrhagic phase has not ended, the pulse is usually hard and tense ; in some cases fever is marked, and the prognosis is usually bad.

Hæmoptysis may **precede** the other symptoms by many years, and may be repeated during apparent good health, while auscultation shows no sign of the mischief. This proves that, in its embryonic stage, the tubercle produces congestion, which may extend, directly or reflexly, to the respiratory channels, or even to the nasal mucosa ; and it is a notable fact that tubercular patients sometimes have attacks of epistaxis which precede or accompany the hæmoptysis. It is the *molimen hæmorrhagicum* associated with the formation of tubercles.

Pulmonary congestion of tubercular origin is, then, a cause of hæmoptysis. This assertion must be admitted without dispute, as it rests on facts, verified post mortem ; but it is more probable that the obliteration of vessels by the tubercular products produces collateral congestions, which are largely responsible for the bronchial hæmorrhages. Perhaps the violent congestion which results in the rupture of capillaries and in hæmoptysis is favoured



by the vaso-dilatory toxine of the tubercle bacillus, to which Bouchard has given the name of *ectasine*.

It is upon cases of early hæmoptysis that Morton's theory was based—*Ab hæmoptæ tabes*. According to this theory, the hæmorrhage itself becomes the starting-point of the tubercularization of the organ. This theory is dead. When an individual who has had hæmoptysis becomes phthisical two or three years after the spitting of blood, the bronchorrhagia is not the cause; it is only an early sign of the tubercular lesion. We have, besides, the proof of this in the examination of the semisanguineous sputa which follow hæmoptysis. Tubercle bacilli have several times been recognized in the sputum, although other signs of tuberculosis were absent.

Let us now consider late hæmoptysis, which occurs in the period of *cavities*. This late hæmoptysis may be due to the various causes which I have just enumerated above, and is then the result of new attacks of tuberculosis; but sometimes, also, it is caused by the rupture of small aneurysms in the wall of the cavities. These aneurysms, which we call Rasmussen's aneurysms, from the name of the Danish physician who first clearly described them, are found in the pulmonary and bronchial arterioles, whose calibre is from 1 to 5 millimetres. They vary in size from a pin's head to an almond. The rupture of the aneurysm may be delayed by the formation of clots, but when the aneurysm bursts, the hæmorrhage is so abundant that the patient vomits mouthfuls of blood, and death may supervene in a few minutes. I have seen such a case: the young girl was seized with fulminating hæmorrhage, and succumbed in less than five minutes.

The above description gives an idea of the early and late hæmorrhages of pulmonary tuberculosis. The early cases never prove fatal by their abundance, but the late ones may carry off the patient in a few minutes. The absence or presence of **fever** is important in prognosis. Non-febrile hæmoptysis, especially if early, is not severe; but hæmoptysis which is febrile or associated with the febrile forms of tuberculosis has an exceptional gravity.

Hæmoptysis is present in about 60 per cent. of cases, and may be for years the **sole** symptom of almost latent tuberculosis. The case quoted by Andral is well known: A man who had hæmoptysis all his life died of a disease other than phthisis; at the autopsy cretaceous tubercles were found. All his children died of phthisis. I have often seen such cases; more often than we think, hæmoptysis is the **only evidence** of pulmonary tuberculosis. Hæmoptysis is not one of the symptoms of tuberculosis in infancy, and is rare in patients under fifteen years of age. It is more frequent in women than in men, and many cases of so-called **supplementary** hæmoptysis are tubercular.

The **genital functions** are less affected in men than in women. At a more or less advanced stage in phthisis the menses become irregular, and

may be suppressed, or at times replaced by leucorrhœa. These troubles explain why pregnancy is rarer in phthisical women. The puerperal and nursing states have, furthermore, a bad influence on the course of the disease.

The **œdema** seen in phthisis is of diverse origin: cachectic œdema, which begins in the lower extremities; painful œdema, which is due to venous coagulation (**phlegmasia alba dolens**), and is generally limited to one limb; and diffuse œdema, with lesions of the kidney and albuminuria, are seen.

**Lesions of Other Organs.**—I have so far described the lesions of the lung, and discussed the most usual symptoms of pulmonary phthisis. There is hardly an organ which escapes tubercular infection. Whether tubercular infection be present alone, or whether it be aided in its work of destruction by secondary infections from the toxins of other microbes, many and varied troubles result, modifying the picture of the disease or hastening its course. The study of these complications will be discussed fully when dealing with the tuberculosis of each organ; I shall here content myself with a rapid enumeration.

**1. Digestive System.**—The changes in the digestive system comprise tubercular ulcerations of the tongue, the mouth, and the larynx; tuberculosis of the tonsils; gastritis, which is sometimes ulcerating; all varieties of dyspepsia; chronic enteritis; ulceration and fistula of the anus; tuberculosis of the mesenteric glands; acute and chronic peritonitis.

The lesions of the **liver** present the most diverse forms; for the liver, like every other organ, has its own peculiar tuberculosis. The tubercular granulation is not the only lesion present. We find also tubercular lesions affecting the type of cirrhosis or of fatty or amyloid degeneration.

**2. Circulatory System.**—The troubles of the circulatory system include palpitation, which is very frequent; dilatation of the right heart; tubercular pericarditis and endocarditis; the formation of clots in the veins of the limbs; and thrombosis of the pulmonary artery, which is a possible cause of rapid death.

**3. Genito-Urinary System.**—The testes, the prostate, the bladder, the kidney, the ovaries, and the uterus, may be affected by tuberculosis. It may remain as a local condition, or precede that of the lung.

**4. Nervous System.**—Here we find extensive or localized meningitis; lesions of the cerebrum, with all their train of symptoms; tubercular lesions of the mesoencephalon and the bulb, and those of the spinal cord (tubercular leptomyelitis), which do not differ in their clinical description from other varieties of myelitis.

In opposition to these lesions of the nervous centres, I would mention **peripheral neuritis**, which has of late been carefully studied. Clinically, it shows itself by sensory (neuralgia, hyperæsthesia, anæsthesia), motor (paralysis, paresis), and trophic troubles (amyotrophia, zona).



**5. Organs of the Senses.**—Otitis in phthisical patients is a catarrh of the drum (otorrhœa), with perforation of the membrana tympani. The ulceration of the mucosa causes caries and necrosis of the petrous bone. This otitis may be consecutive to pharyngeal catarrh, which has spread to the drum through the Eustachian tube (Bellière). Tuberculosis of the nose and of the nasal fossæ has been already described.

**Tuberculosis in Children.**—I shall now mention tuberculosis in children and elderly people. Children may become tubercular at all ages, but the very young child (one from a few days up to two years old) presents a familiar form of tuberculosis. It has been summed up by Landouzy and Queyrat as follows: Infantile tuberculosis often appears as a broncho-pneumonia. It may show all the lesions of tuberculosis seen in adults, including cavities surrounded by fibrous tissue and aneurysms, which predispose to fatal hæmoptysis. Tuberculosis is transmitted to the new-born either by mediate contagion or by heredity.

In elderly people tuberculosis is more frequent than is usually supposed, but it has not the characteristics of phthisis in the adult. It is more torpid, and its symptoms are less marked.

**Course—Prognosis—Termination.**—The course and duration of pulmonary phthisis are extremely variable. One individual, for example, may for many years bear the growth of tubercular products without passing into the third stage; while another, after six or eight months' illness, is already a victim to sweating, wasting, diarrhœa, and hectic fever. Some people who have had apparently insignificant hæmoptysis show no symptoms of proved tuberculosis until ten or fifteen years later. Others may have had several attacks of hæmoptysis and inveterate bronchitis, but yet have dragged out a more or less invalid existence, without ever reaching the third stage. Many causes hasten or retard the course of pulmonary phthisis. Firstly, there is the nature of the soil on which the disease has developed; then there are the cares of every kind, hygiene, questions of climate, nourishment, and comfort, and hence the much quicker course of tuberculosis amongst the poorer classes, while among those in easy circumstances we can often check or modify its progress. It has been said that when emphysema occurs in a tubercular lung, it arrests the progress of the tuberculosis for the time being.

Between the local changes in the lung and the general condition of the patient there is, of necessity, some parallelism. We see people with local signs of advanced tuberculosis (softening and cavities), but they live on, without reaching the stage of consumption. These examples must be known in order to avoid grave errors in prognosis.

The natural end of pulmonary tuberculosis is often death by phthisis; yet cases of cure are frequent (Grancher). I have often seen recovery from

pulmonary tuberculosis when it is treated at an early period. The tubercle may recover (cretaceous and fibrous condition), and the small cavities may cicatrize. In certain conditions death supervenes from complications, such as laryngeal phthisis, purulent pleurisy, pneumothorax, enteritis, peritonitis, meningitis, etc. Syncope, embolism, or thrombosis of the pulmonary artery are causes of sudden death.

**Ætiology.**—**Heredity and contagion** are the two great causes of pulmonary phthisis, and in this respect tuberculosis shows an analogy with syphilis. In both heredity may betray itself by early or by late manifestations. The early ones are, in the one case, syphilis in the new-born; in the other, tuberculous broncho-pneumonia of infancy, meningitis, and tuberculization of the peritoneum and of the bronchial glands. The late manifestations are, in the one case, the multiple lesions of hereditary syphilis; in the other, pulmonary phthisis and the various local tubercular lesions till lately considered as scrofulous.

Sometimes the **parents** are notably phthisical; at other times they show only such imperfect evidences of tuberculosis as inveterate bronchitis or suppurating adenitis, wrongly regarded as scrofulous; they drag out a more or less invalid existence, without going on to confirmed phthisis. They may be cured, or think themselves cured. These troubles favour tuberculosis, and children born of such stock may, unfortunately, inherit the original taint. Sometimes, in a family where tuberculosis or scrofula reigns (and they are identical), the parents in whom the germ is latent beget children who develop tuberculosis, while the parents themselves are only affected later.

We may consider heredity in two ways: either the subject inherits the infectious principle—that is to say, the seed; or he only inherits the predisposition to contract tuberculosis—that is to say, the nature of the soil which is favourable to its growth. Many authors incline to this latter opinion. Peter says that people are not born tubercular, but tuberculizable. Bouchard says that the tuberculosis which parents transmit to their children is prospective, and not actual.

This opinion is admissible, but it is none the less true that the hereditary lesion has been caught red-handed in the fœtus. The inoculation of guinea-pigs with blood from a fœtus conceived by a phthisical mother has caused tuberculosis analogous to that which a fragment of tubercular lung produces.

We possess to-day undeniable cases of **congenital tuberculosis**. In two cases out of five, Koch's bacillus has been found in the blood of the umbilical vein of infants born of tubercular mothers (Bar and Rénon). Tuberculosis has been recognized in the fœtus at various ages. It may be, says Kuss, that the germs reach the fœtus a little before delivery, or at the moment of birth (thanks to placental depletion, produced by the first



inspirations), and only succeed in setting up appreciable foci at the end of several months. It is therefore reasonable to admit that hereditary tuberculosis is transmitted directly by the germs. The whole question consists in recognizing what the conditions will be which, sooner or later, will favour the growth of the germs, which may long remain in a latent state. This latency of the bacilli is not at all surprising.

The contagious nature of tuberculosis had been long supposed, but it was clearly established by Villemin. To-day it is based upon a considerable number of cases. Cases of contagion between husband and wife are relatively frequent: a healthy husband becomes tubercular through contact with his wife who is dying of phthisis. He marries again, and, in his turn, gives tuberculosis to his second wife, who was quite well. I could give many more cases; the more intimate the living in common with phthisical persons, the more is contagion to be dreaded.\*

In the preceding chapter I describe the modes of transmission of tuberculosis by experimental measures. How can we explain its transmission in the human species? It is possible that the bacillus enters the digestive tract by means of food and drink, and in this connection milk, both from a tubercular animal and from a tubercular wet-nurse, has been incriminated. Calmette thinks that the intestinal tract is often the entrance-gate of pulmonary tuberculosis. Verneuil thinks that the contagion may take place through the genital tract. In some cases the bacillus has entered the economy through a wound in the skin.

The respiratory passages are the usual entrance gateway of the infectious germ (spore or bacillus), and in most cases the lung is the first organ attacked. The germs exist in abundance in the sputum of phthisical patients, and experience has proved that the sputum may be dried, powdered, and kept for several weeks without losing its virulence, since the dust, when inhaled by animals, causes tuberculosis (Tappeiner). Transmission to man probably occurs in this way: the débris of sputum, reduced to powder, floats in the air and enters the bronchi. In order that the germ may produce its noxious effect, the bronchus must have first lost its epithelium, and it may be asked if bronchitis and broncho-pneumonia (measles, whooping-cough) do not especially favour the entrance of the germ. In all cases the contaminated person must be in a condition of receptivity, for in case of **contagion** we find favourable soils and soils refractory to the growth of germs.

Children of tubercular stock, patients with diabetes or convalescent from

\* These propositions are general, and apply equally to animals and to man. Kris-haber and myself, in 1883, made experimental researches upon tuberculosis in the ape, and lost only one monkey out of eighteen which were living together, but were kept isolated from every source of contamination, while we lost five monkeys from tuberculosis out of twenty-four which lived with tubercular animals. They had contracted tuberculosis by contagion (*Arch. de Physiologie*, 1883).

acute diseases (measles, whooping-cough, typhoid fever) are in a state of receptivity. The soil is prepared by malnutrition, failing health, excesses of every kind, exhaustion, fatigue, trouble, and repeated pregnancies. "It may be said that tuberculosis is the common end of all constitutional degenerations in families as well as in individuals" (Jaccoud).

**Traumatism** and contusions of the thorax may be placed in the category of predisposing causes, by tilling the soil in which the bacillus was in a latent condition. Since attention has been called to this point many cases have been collected, and I know of several in which injury to the thorax has been followed by tuberculosis.

Tuberculosis is most common in youth; it appears, however, at other periods. It is fairly common in advanced age and in children during earliest infancy. It is more common in warm climates, and is comparatively rare in high-lying countries.

**Diagnosis.**—The diagnosis of pulmonary phthisis is sometimes difficult, not only at the onset of the disease, but even at more advanced periods. Sée divides the disease into latent, larval, and pseudo-phthisis. In **latent** phthisis we find ill-marked symptoms, such as pallor, wasting, with or without fever, catarrhal cough, or hæmoptysis. The cases of **larval** phthisis are those which assume the mask of an acute affection of the respiratory tracts, such as bronchitis, pleurisy, broncho-pneumonia, or laryngitis. The cases of pseudo-phthisis are diseases such as bronchial dilatation, syphilitic gummata of the lungs, hydatid cysts of the lung, aspergillary tuberculosis, and neoplasms of different kinds that simulate phthisis, but are not of tubercular origin. In these different cases the diagnosis is not always simple, and the recognition of Koch's **bacillus** renders the greatest service.

As regards **diagnosis**, I think it useful to state the following precepts:

Every youth or adult who wastes much or rapidly, with or without fever, must be suspected of having tuberculosis, in the absence of diabetes or Basedow's disease.

Every girl or young woman who has neither genuine chlorosis, Bright's disease, nor syphilitic anæmia, but yet has the appearance of chloro-anæmia, must be suspected of having tuberculosis.

Every individual who has hæmoptysis must be suspected of tuberculosis. The cases of so-called **supplementary** hæmoptysis are very often tubercular; but, on the other hand, we must remember that there is a group of **non-tubercular** cases, such as hæmoptysis in bronchitis, hydatids, or syphilis of the lung, false tuberculosis, etc.

Koch's tuberculin has undoubted value in diagnosis, and may reveal even the most hidden trace of tuberculosis. It has therefore attracted the attention of hygienists, and has been employed in veterinary practice for the early diagnosis of tuberculosis in cattle.



In man great care must be taken in using injections of tuberculin for purposes of diagnosis. The amount must not be more than  $\frac{1}{10}$  or  $\frac{1}{2}$  milligramme; two or three injections may be made at intervals of two or three days. In case of positive reaction, the temperature rises about  $1^{\circ}$  or more. The rise is often accompanied by lassitude and headache.

The cuti-reaction does not give constant results. I have verified this fact at the Hôtel-Dieu.

**Prognosis.**—I shall not dwell at length on the prognosis of pulmonary phthisis. The preceding description shows how serious the disease is. There is, however, a point to which I would call attention—viz., that tuberculosis is cured much more often than we suppose. Not only is it curable at its onset, but it is still curable at an advanced period. In people who have died from quite another disease we often find old fibrous or cretaceous tubercular lesions, which prove that they have at some time been affected by tuberculosis.

**Treatment.**—Let us first consider **prophylactic** treatment, the object of which is : (1) To modify as far as possible the evil results of heredity in a person of tubercular stock ; (2) to remove the causes of **contagion**. The individual should from infancy live in the open air, in the country, and in a high-lying locality, take much exercise, have abundant nourishment, and avoid all causes of contagion ; for, thanks to his origin, he is, above all others, in a **state of receptivity**.

The means employed to avoid contagion are as follows : Choose for the child a nurse who has no tubercular taint ; never permit a child to sleep in a room with phthisical parents ; do not allow husband and wife to share the same room. Collect, as far as possible, the products of expectoration in a basin, which is emptied and washed several times a day ; and do not allow sputum to be left on linen, pocket-handkerchiefs, or on the floor, where it dries, is reduced to dust, and becomes a potent cause of contagion. Purify rooms and bedding after the death of a phthisical patient ; expose clothing which the patient has used, to steam at a temperature of  $212^{\circ}$ , or, better still, burn it. If these precautions were taken, the balance-sheet of phthisis would be much diminished.

We may next consider the different medicines and their respective efficiency in phthisis.

Cod-liver oil, in sufficient doses, builds up the constitution. Intolerance on the part of the stomach and diarrhœa are contra-indications. Tolerance may sometimes be established by giving the oil in increasing doses, after it has been well cooled in a freezing mixture. I have obtained excellent results with cod-liver oil in large doses—*e.g.*, a tumblerful. Some patients take 10 to 15 ounces daily for several weeks. I repeat that the results obtained are often surprising. When the oil disagrees, I substitute fatty and oily foods ; caviare, sardines in oil, tunny-fish, slices of bread and butter, *pâté de*

*foie gras*, etc. Excellent results are sometimes obtained. I would especially recommend eggs and raw meat in large quantities.

Meat-juice has been used with benefit. The good effects of this régime do not rest solely upon pure and simple super-alimentation, but on the particular quality of the food.

**Glycerine** is a very useful and economical drug, and is given daily in doses of 1 to 2 ounces, with a little rum, and flavoured with a drop of essence of peppermint (Jaccoud). Preparations of **arsenic**, because of their trophic action, are much indicated: arseniate of soda is given in doses of  $\frac{1}{80}$  to  $\frac{1}{12}$  grain daily in distilled water; or arsenious acid, in doses of  $\frac{1}{20}$  to  $\frac{1}{10}$  grain daily, in the form of granules, each containing  $\frac{1}{80}$  grain. Cacodylate of soda, in daily injections of about 1 grain or more, is an excellent medicine. (See Appendix on **Therapeutics**.)

**Creosote** stimulates the appetite and lessens the bronchial secretion; it is given in capsules or in pills, each containing 1 minim creosote. The patient takes as many as ten or twenty during his meals.

**Counter-irritation**, by repeated blisters or by the cautery, is useful in dealing with inflammation and congestion. In my own practice I make much use of counter-irritants. It is well to use the cautery, and to allow suppuration to go on as long as possible. A good way is to apply the cautery-point 200 or 300 times a week, and to continue this treatment for a long period.

The **fever** of tuberculosis, especially in the hectic form (absorption fever), may be checked by salicylic acid (Jaccoud's method). Twenty grains of salicylic acid are given during the morning in three cachets, at intervals of half an hour. According to circumstances, the dose is diminished, stopped, or repeated some days after.

**Aspirin** (acetyl-salicylic acid) in 5-grain doses, repeated three or four times a day, is useful for the fever of phthisis (Rénon); abundant perspiration limits its use.

**Cryogenin**, in doses of two or three cachets, each containing 1 grain, has a remarkable antithermic action.

Antipyrin is efficacious, in daily doses of 30 to 40 grains. For the fever, I have often prescribed bathing with tepid water or cold baths, with great benefit to the patient.

Hæmoptysis should be dealt with in the following manner: If very abundant, it is sometimes arrested by an emetic, such as ipecacuanha (30 grains). If it is more trifling, ipecacuanha is prescribed in nauseating doses, and one or two of the following pills are administered every hour, or every two hours:

|                  |    |    |    |    |    |                    |
|------------------|----|----|----|----|----|--------------------|
| Ipecacuanha      | .. | .. | .. | .. | .. | gr. i.             |
| Extract of opium | .. | .. | .. | .. | .. | gr. $\frac{1}{30}$ |



Other means are also used for hæmoptysis : subcutaneous injections of ergotin, solutions containing perchloride of iron, and counter-irritation to the chest by means of blisters. The patient should be kept absolutely at rest, given iced and acidulated drinks, and have abundant cold nourishment, which should be administered in small quantities. The following draught may also be prescribed, in tablespoonfuls, every three hours :

|                  |    |    |    |    |    |        |
|------------------|----|----|----|----|----|--------|
| Distilled water  | .. | .. | .. | .. | .. | ℥iv.   |
| Syrup of rhatany | .. | .. | .. | .. | .. | ℥i.    |
| Rabel water      | .. | .. | .. | .. | .. | ℥xliv. |

The **sweats** are relieved by white agaric (Trousseau), in doses of 3 grains, taken every evening, in a cachet ; by atropin (Vulpian), in doses of  $\frac{1}{30}$  grain ; or by camphorate of pyramidon, in doses of from 6 to 8 grains, in a cachet.

**Diarrhœa**, which is often profuse, may be treated with chalk in large doses ; by opiates (five to ten pills, each containing  $\frac{1}{8}$  grain opium, given in the course of twenty-four hours) ; by methylene blue, given daily, in three cachets, each containing 3 grains of lactose and 1 grain of methylene blue.

**Vomiting** may be relieved in some cases by 2 drops of laudanum, taken before a meal ; or by a tablespoonful of lime-water, with the addition of  $\frac{1}{30}$  grain of hydrochlorate of cocaine ; at other times by lavage of the stomach, or, better still, by artificial feeding. This artificial feeding (super-alimentation, gavage), used by Debove, has often proved effective, not only in vomiting, but also in malnutrition. Milk, lentil-flour, and powdered meat are given through a tube. Some patients will swallow, without the help of the tube, 6 ounces of powdered meat in the twenty-four hours, the powder being suspended in milk or broth, given at short intervals. Super-alimentation sometimes produces remarkable effects.

**Koch's Tuberculin.**—I must now give some details of treatment by Koch's tuberculin.

On November 13, 1890, Koch announced that he had found an efficient remedy for tuberculosis of the skin, bones, glands, or lungs ; and this remedy — Koch's lymph — which was immediately tried by many physicians, remained a secret until the beginning of 1891, when Koch, in a second paper, stated that it was tuberculin.

Tuberculin is a glycerine extract of pure cultures of the tubercle bacillus. It is a clear brownish fluid, without action when introduced by the mouth, but very active when injected under the skin or into the blood-stream. In a healthy man a dose of 0.25 c.c. produces very marked effects, while in a guinea-pig a dose of 2 c.c. remains inactive. The human being is therefore 1,300 times more sensitive to its action than the guinea-pig.

In a tubercular guinea-pig tuberculin, given in gradually increasing doses, has no action on the bacilli, but produces a marked effect on the

tissues around the tubercle. These tissues show abundant exudation of serous fluid and active diapedesis. They may or may not become necrosed. When necrosis occurs, the cast-off tissue contains bacilli in more or less considerable numbers, and the ulceration thus produced may be converted into a simple wound, covered with fleshy granulations, which may form scar tissue.

In a healthy man 1 c.c. of lymph is necessary to produce a rise of temperature to 100° F. In a tubercular man a dose of from 1 to 3 c.c. of lymph is sufficient to produce, at the end of two or three hours, a rigor, a temperature of 104° F., cough, vomiting, acute pain, enlargement of the spleen, and slight delirium. These symptoms last twelve to fifteen hours. In the case of **lupus**, local reaction can be made out at the site of the tubercular lesions. At the end of five or six hours the lupoid areas swell, become red, and sometimes necrose. The redness diminishes after two or three days, and is accompanied by scaling. Two or three weeks later the eschars become detached, leaving in favourable cases a lasting cicatrix. The same thing happens in tuberculosis of the glands, bones, or joints, but the reaction is less clear. In favourable cases of pulmonary tuberculosis, after subcutaneous injections of lymph in very small doses, the cough is less frequent, the expectoration becomes mucous, the bacilli diminish, and finally disappear, and the general condition improves.

Such are the results of tuberculin in favourable cases; but these cases are **exceptional**, and, after many well-conducted observations, the infatuation first aroused has given place to the most profound discredit.

Most serious accidents, and even death, have occurred after inoculations of tuberculin. Amongst these accidents, I would note meningitis, endocarditis, oedema of the glottis and of the lung, broncho-pneumonia, etc. After inoculations performed in my wards with the greatest care, I have seen perforation of the tympanic membrane from suppurative otitis. Such gloomy results have caused the use of injections of tuberculin in phthisis to be abandoned.

The commission of physicians at the St. Louis Hospital, which for two months and a half tried tuberculin on thirty patients suffering from **lupus**, decided against Koch's method of treatment. In some cases temporary reduction of the lupoid mass and temporary attenuation of the tuberculous lesion were seen, but cure was never obtained. In the course of these experiments no patient, fortunately, succumbed, but very grave complications were seen in the heart, brain, and kidneys, as well as the most alarming general symptoms. In some cases tubercular centres in the lung, till then latent, were reawakened, and it was very difficult to arrest the lesion which had once more become acute.

**Tuberculocidin.**—Klebs has separated the noxious principles and alka-



loids of Koch's tuberculin from the active principle which is an albumose. This purified lymph, or tuberculocidin, does not give rise to febrile reaction ; it causes retrogression in the tubercular tissues, without necrosis, and brings about the destruction of the bacilli. The dose, at the commencement of treatment, is  $\frac{1}{13}$  grain, and it may be raised to 7 grains later. The results are encouraging.

C. Spengler (of Davos) has combined the action of tuberculin with tuberculocidin. He injected a mixture of from  $\frac{1}{500}$  grain of tuberculin with  $\frac{1}{3}$  grain of tuberculocidin. Injections were made several days following, and in some cases the fever yielded.

Denys has used bouillon, freed from the tubercle bacillus by filtering, in the treatment of tuberculosis in man. He gives hypodermic injections in doses varying from 1 to 25 c.c. He has seen good results, but the value is not yet exactly known.

Let us now consider thermal and climatic treatment in pulmonary tuberculosis. Speaking generally, **thermal cures** are absolutely contra-indicated while the patient is suffering from hæmoptysis or from fever. In patients affected with slow tuberculosis, who are but little prone to sharp reaction, hæmoptysis, or broncho-pneumonia, Mont-Dore, la Bourboule, and the sulphur-waters, such as Cauterets, Luchon, Eaux Bonnes, etc., may be recommended; but in patients suffering from pulmonary tuberculosis in the irritative form, the warm alkaline springs of Royat and Ems, or the cold sulphur springs of Allevard, are advisable.

A similar distinction should be made when choosing a place of residence for tubercular patients. In cases liable to febrile attacks and palpitations of the heart, high altitudes, such as Davos and Saint-Moritz, will be most beneficial. These elevated situations give wonderful results, as regards improvement and cure, from the purity of the air and the activity which they cause in the breathing. All forms of phthisis, however, are not suited to these high altitudes ; stations of moderate altitude, or the maritime ones of Cannes, Mentone, Arcachon, Algiers, Madeira, or Pau, are then to be considered.

Surgical treatment of tubercular lesions of the lung, interstitial injections of drugs, and pneumotomy, are questions which are under consideration, and upon which it would be premature to pronounce an opinion.

#### XIV. FIBROID PHTHISIS.

The tubercles in the lung, instead of undergoing caseation, may be converted into fibrous tissue. They form isolated granulations, which are prominent and very hard to the touch ; their structure is completely modified by the fibrosis. Fibrous tubercles had already been noted by Bayle, but

Cruveilhier was the first to show the importance of this change : “ Pulmonary tubercles are too generally considered as being incurable ; they may become cicatrized.” He noted “ granulations and **tubercles of recovery** ”—that is, inert granulations and tubercles—perfectly distinct from those in process of development, with which they had been confounded. Cruveilhier also noted that tubercular lesions may recover at any stage, from the miliary granulation to the cavity, by a fibrous change ; and that, further, “ part of the lung tissue around the tubercles may be converted into fibrous tissue.” This change constitutes a means of isolation and cure of the tubercular centres. The condition constitutes the curative phlegmasia, or dark slaty induration which we now call interstitial pneumonia in phthisical patients.

Later, Grancher and Charcot showed that this fibrous change is very common, and may occur at a very early stage ; it must be considered as one of the modes of evolution of the tubercle.

Furthermore, the works of these authors, and those of Rénaut, Bard, Cornil, and Thaon, made it plain that Cruveilhier’s dark slaty induration may end in true pulmonary fibrosis or fibroid phthisis ; and that while the fibrous change in the young tubercle is a true process of recovery, the pulmonary cirrhosis which surrounds the caseous tubercles has not always that happy result. Indeed, we now know that Koch’s bacillus may remain alive for a very long time in caseous tubercles surrounded by fibrous tissues, and that pulmonary fibrosis of an **invading** character may cause disastrous results, and be in itself a serious condition.

Lastly, Cruveilhier showed that large fibrous tubercles, especially in old people, may contain not only caseous substance, resembling dry putty, but also stony granules. Very small calcareous granules have been noted by Schüppel and by Ziegler in the tubercular follicles, where they assume quite a special disposition, and are made up of concentric layers. Metchnikoff found calcareous bodies of the same character in the tubercles of Algerian gerbil (*Meriones Shawi*), an animal in which the infection spreads very slowly after inoculation. In the centre of these calcareous bodies we find tubercle bacilli, which in the early stages of the lesion appear perfectly normal, and are surrounded by a layer of amorphous substance. Later they lose their faculty for taking stains, degenerate, and disappear. According to Metchnikoff, the stratified layers are multiple cuticles, secreted by the bacillus for defensive purposes, and the phosphate of lime is probably deposited in this cuticle by the giant cell itself, in its reaction against the bacillus.

Although these facts are not applicable to the calcareous bodies in the tubercle of man, they are very interesting as reconciling the tubercles of recovery with the ideas we have concerning them.

The histological changes are as follows :



**1. Fibrous Tubercle.**—When the tubercle reaches its stationary stage, we find new connective fibres, which interpose themselves between the cells, and finally make up most of the granulation in which giant cells may still be met with at the centre or at the periphery. “These giant cells are often situated in a cavity which holds them exactly, and is made up of fibrous bundles, forming a circle around them. The isolated fibrous tubercles, and the periphery of the large confluent tubercles of the same nature, have in their interior capillary vessels permeable by the blood. The wall of the vessel is often thickened, especially in the sclerosed tissues. While the tissue of the granulation becomes fibrous, “the septa of the pulmonary alveoli have a tendency to become thickened, and interstitial pneumonia develops around the granulations that have become fibrous.” In the thickened walls of the alveoli, small round cells, which infiltrate the bundles of the connective tissue, are seen in large numbers. In the oldest, and especially the confluent, fibrous tubercles the giant cells disappear at the centre of the islet, which is now only formed of fibrillary connective tissue, without vessels and cellular infiltration; fibrous tubercles, with giant cells and permeable vessels, are found at the periphery.

The old fibrous tubercles may be infiltrated with black pigment. These black granulations, composed of particles of carbon, and also of altered blood-pigment, may occupy the whole granulation, or only a part of it. They are then situated at the periphery, for they are found chiefly in the cells; they may be seen in the giant cells.

These fibrous tubercles, even when they contain giant cells, may be considered as absolutely arrested in their evolution.

**2. Interstitial Pneumonia.**—The fibrous, pigmented tubercles often cause thickening of the septa of the lung, so that they may be situated in the middle of an indurated connective tissue which no longer presents alveolar cavities. Around these masses, where the lung structure has quite perished, the pulmonary tissues show the lesions of interstitial pneumonia: thickening of the alveolar septa, which are made up of fibres of connective tissue and flat cells containing black pigment; alveolar exudate, made up of leucocytes and pigmental epithelial cells; alveolar septa covered with layers of large swollen epithelial cells; narrowing of the alveoli, which may take an elongated form, while their direction is perpendicular to that of the thickened interlobular septa. In certain places distended vessels, which form a kind of cavernous tissue, are found in the connective tissue. Side by side with the lesions of interstitial pneumonia we sometimes see opaque formations, which to the naked eye resemble tubercles. This condition is really one of venous thrombosis, surrounded by fibrous zones, possessing a rich collateral circulation (Cornil). This interstitial pneumonia may be found around caseous centres and healed cavities with thickened walls.

This condition is seen in the lung, where the fibrosis has made slow progress. In these cases it is often found associated with chronic adhesive pleurisy and thickening of the subpleural connective tissue. In this way arises pulmonary fibrosis, which not only deprives a part or the whole of the lung of all function, but which may also result in deformity of the chest and dilatation of the bronchi.

In other cases fibroid phthisis does not run the same course. The induration may affect a whole lobe, or even both lungs, in the form of islets, or of large masses around indurated or caseous miliary tubercles without or with merely small cavities. Interstitial pneumonia then presents a form of subacute phthisis, and at times matures fairly rapidly: in six months or in a year (Cornil).

To sum up: the fibrous change may affect young, isolated tubercles, and thus bring about a true cure of these lesions. The interstitial pneumonia which is often present, and the fibrous change in the more advanced lesions, are frequently of good omen. On the one hand, however, they do not always arrest the course of the lesions; and, on the other hand, they may go beyond the mark, and give rise to pulmonary fibrosis, with its results.

#### XV. PNEUMONIC PHTHISIS—TUBERCULAR PNEUMONIA— CASEOUS PNEUMONIA.

Since Laënnec traced the anatomical and clinical history of phthisis two chief schools of thought have arisen—the one affirming the **unity**, and the other the **duality**, of phthisiogenous lesions.

According to the former, pulmonary tuberculosis and caseous pneumonia are one disease; they are both traceable to heredity, go on to the same termination—*i.e.*, phthisis—and if their lesions, tubercular granulations and caseous infiltrations present any difference, such difference concerns only the form of their morbid products, and not their nature. Laënnec therefore bequeathed to us the doctrine of their unity, both from the anatomical and the clinical point of view.

According to the **dualists**, on the other hand, pulmonary tuberculosis and caseous pneumonia represent two distinct diseases. In 1850 Reinhardt affirmed that the lesion described by Laënnec as tubercular infiltration was nothing else than “caseous pneumonia.” According to the dualists, the pathological anatomy, course, and prognosis were different in the two cases. Tubercular granulations and caseous infiltration had neither the same origin nor the same nature; heredity was the chief element in the case of the tubercular patient, while in the case of the patient with caseous disease, he got off with a pneumonia which Virchow called scrofulous, “if he had not the ill-luck to become tubercular” (Niemeyer).



You see, said the dualists, that this scrofulous pneumonia (Virchow's) has nothing in common with pulmonary tuberculosis, since there is a difference in form, seat, origin, and nature between the two maladies—difference in form, because the tubercular granulation is nodular, while caseous pneumonia is diffuse; difference in situation, because the granulation arises outside the alveolus, while caseous pneumonia arises inside; difference in origin, because the tubercular granulation develops at the expense of the connective tissue, and caseous pneumonia at the expense of the pulmonary endothelium.

Such were the anatomical and histological arguments invoked by the dualists, and it is on this ground that each of their conclusions has been vigorously attacked and successfully routed in various works, and notably by Grancher, in a thesis which forms an eloquent plea in favour of Laënnec's work.

But what definitely ruined the dualist doctrine and confirmed the triumph of the French school is Villemin's discovery, on the one hand, and Koch's discovery, on the other. Since Villemin made his memorable experiments, we know that all tubercular products are virulent and inoculable, **whatever their form may be**; and we know also, since Koch's great discovery, that all these products contain the specific bacillus of tuberculosis.

The experiments of Auclair deserve quoting: In the rabbit caseous pneumonia follows the intratracheal injection of an ethereal extract of bacilli from tubercular patients; interstitial pneumonia may be the result of inoculation with a chloroform extract of the same bacilli. The double fibro-caseous evolution of the tubercle appears, then, to depend upon a double toxine, which explains the incidence of the lesions to one or to the other process, or to both at once.

There is no longer any reason for preserving the term "caseous pneumonia." The infectious malady called tuberculosis, which runs an acute, sub-acute or chronic course in the lung and other organs, shows itself anatomically by products of a tubercular nature. The products are sometimes of small size and nodular in shape (grey granulations, miliary tubercles); at other times they are of large size (discrete or confluent nodules, tubercular infiltration). The granular form may exist alone—*i.e.*, acute granular tuberculosis. The pneumonic form, whether discrete or confluent, may exist alone—*i.e.*, caseous pneumonia, or, better, tubercular broncho-pneumonia, which may more or less quickly give rise to acute and subacute phthisis.

**Pathological Anatomy.**—Tubercular pneumonia is usually limited to one lung; it may be limited to a few lobules or invade a whole lobe. It attacks the lower as well as the upper lobe.

The lung presents different appearances, according as the lesion is more or less advanced. In the more advanced stage the lung tissue resembles

Roquefort cheese—hence the name “caseous pneumonia”—and the greyish areas form with the yellowish and brownish ones a kind of mosaic. The cut section does not present the granular condition of lobar pneumonia, but is more opaque, smooth, dry, homogeneous and anæmic.

The lesions of commencing tubercular pneumonia are seen side by side with the caseous masses. We find masses of rosy-coloured, quivering, and semitransparent substance, called by Laënnec “colloid infiltration,” but since termed “caseous colloid pneumonia” (Thaon); masses of greyish homogeneous substance are also seen: this is Laënnec’s grey infiltration. These tubercular infiltrations may go on to caseation.

If we study caseous infiltration closely, we see that it is not disseminated at random through the lung; it is composed of more or less confluent nodules, leaving between them lung tissue that is healthy, or showing the lesions of ordinary broncho-pneumonia. The caseous nodules have a special texture, and are, for the most part, developed around a small bronchus, which is the centre of their formation (peribronchial nodules). They are composed of (1) a central region, and (2) a peripheral zone. The older the central zone, the more caseous it is. Some vestiges of lung are seen—*i.e.*, arterioles—rings and bands of elastic tissue, which represent the bronchioles and the alveolar walls. Around this central region, which comprises the caseous centre of the tubercular follicle, we see a zone that is well defined on the side of the central region, but irregular at the periphery. This embryonic zone (Grancher) is formed of embryonic cells, that infiltrate the walls of the alveoli and their cavities; and giant cells that are irregularly disposed in the form of a crown (Charcot), and complete the analogy between the caseous nodule and the tubercular follicle. Here, as in the tubercle, the degeneration begins at the centre, and the lesion extends at the circumference. These caseous nodules which are seen in the different varieties of caseous pneumonia are collections of a tubercular nature; the condition is Laënnec’s tubercular infiltration in its most absolute sense. The caseous inflammation is tubercular, and contains Koch’s bacillus.

The softening of the caseous substance, the ulceration that results, and the cavity, are produced by the process we have already described in the case of tubercle. Caseous pneumonia, however, may last a long while, without giving rise to ulceration of the lung; considerable masses may long remain in the same condition, although the lung is impermeable to air and blood (Cornil and Ranvier).

Chronic caseous pneumonia, like chronic tuberculosis, is always accompanied by interstitial pneumonia (Grancher), and often by fibrinous pleurisy. We shall see later in what relation tubercular granulations are associated with it.

**Bacteriology.**—The tubercle bacillus has always been met with in



caseous pneumonia. In the lobar form the bacilli are found in the centre of the infundibula, in the embryonic cells that fill the alveoli, and in the walls of the alveoli. A similar distribution obtains in the lobular forms. The tubercle bacillus, however, is not alone in evidence in the lesions of tubercular pneumonia and broncho-pneumonia. Mixed infections are present, just as in all broncho-pneumonias; the pneumococcus, the pneumobacillus, and the streptococcus predominate in the diseased zone around the caseous masses. This peripheral zone represents the less advanced stage of the lesion, while the caseous centre represents its completion. The secondary or associated microbes predominate in the peripheral zone, and it is probable that they engender centres of broncho-pneumonia and start the lesion, which the bacillus finally caseifies.

**Description.**—Tubercular pneumonia may be lobular or pseudo-lobar (Vulpian). A lobar form has also been described, but, according to Charcot, tubercular pneumonia is never lobar, and the cases which have been called lobar are confluent lobular or pseudo-lobar.

Tubercular pneumonia, whether it be **lobular** or **pseudo-lobar**, sometimes resembles an acute phlegmasia, and at other times a **chronic** disease; but all intermediate forms—notably the **subacute** variety—are found between these two extremes.

The **acute** pseudo-lobar form begins suddenly with fever, chill, pain in the side; and, indeed, the violence of the chill, the high temperature, the coloured and viscid sputum, may closely resemble the onset of true lobar pneumonia. I saw, some years ago, a medical student who was carried off in a few weeks by tubercular pneumonia of the middle lobe; his sister had died some time before from chronic tuberculosis. The **acute** lobular form, in its initial symptoms, resembles broncho-pneumonia.

The acute forms may carry off the patient very quickly, within a few weeks, even before pulmonary ulceration has had time to develop. At other times cavities appear early, and auscultation allows us to follow the course of events. When the progress of the disease is a little slower, the clinical picture of **subacute** tubercular pneumonia is seen. These acute and subacute forms answer to the terms “acute phthisis” and “acute pneumonic phthisis.”

**Chronic** tubercular pneumonia may follow the acute condition, or be chronic from the first. In the latter case the disease begins silently, preceded or not by some suspicious symptoms, such as laryngitis, bronchitis, or hæmoptysis. The patient coughs and complains of distress, which is often **paroxysmal**, and comparable to the dyspnœa of heart disease, or of an attack of asthma. He loses strength and wastes. On auscultation, we find in one or in several lobes tubular breathing, subcrepitant râles, and marked diminution of the vesicular murmur. These signs are sometimes predominant; they remain stationary in the invaded regions; but the

lesion, in spite of its extent, may only cause moderate fever, and trifling, or purely catarrhal, expectoration.

It is exceptional for caseous pneumonia to recover; in most cases the chronic form leads to ulceration of the lung, cavities, slow consumption, and phthisis, in the true sense of the word. In other circumstances, and in spite of several months' duration, the patient succumbs without ulceration of the lung. He is carried off by asphyxia, with symptoms of hectic fever, sweating, and diarrhoea. I have seen two cases of this kind: one, with Tardieu, in a young girl whose brother died two months later of ordinary tuberculosis; the other, with Krank and Leudet, in a young woman in whom caseous pneumonia complicated, as often happens, fibrinous pleurisy. In both patients almost the whole of the right lung appeared to be converted into a huge caseous block; the dullness was absolute, and the **absence of every normal and abnormal sound** alternated in places with slight tubular breathing, or with subcrepitant râles. The growing dyspnoea and the asphyxia were the chief symptoms throughout the disease.

During infancy, as I have said in the chapter on Pulmonary Phthisis, tuberculosis often shows itself as a broncho-pneumonia.

**Diagnosis.**—The diagnosis of tubercular pneumonia is exceedingly difficult at the commencement. The acute forms simulate broncho-pneumonia or genuine pneumonia, and it is soon evident that we are dealing with acute phthisis. It must be said, however, that cases of acute caseous pneumonia rarely show **the symptoms of genuine inflammation**. Thus, in the form which simulates lobar pneumonia the sputum contains more blood, or is accompanied by genuine hæmoptysis; defervescence does not occur; wasting is rapid; signs of cavities appear in some cases, and the patient is carried off by acute consumption.

The **chronic** and **subacute** forms of caseous pneumonia are quite as insidious in their course. The inflammation is ill defined; the stethoscopic signs, râles, and tubular breathing remain stationary; secondary pneumonia is thought of, and the primary cause is sought elsewhere. The question is asked whether the patient may not be diabetic, or suffering from Bright's disease, or cardiac mischief; but the appearance of further symptoms soon makes the diagnosis clear.

In these doubtful and difficult cases too much information cannot be obtained. The patient's antecedents (hæmoptysis, suspicious bronchitis), and the question of heredity (phthisis among forebears or relatives) will have great weight in diagnosis.

The presence of **tubercle bacilli** in the sputum of patients with caseous pneumonia is less frequent than in cases of common tuberculosis. Bacilli may be wanting in the expectoration, although they may be found in the lung tissue at the autopsy.



## XVI. ACUTE GRANULAR TUBERCULOSIS—ACUTE GENERALIZED TUBERCULIZATION.

The terms "galloping" and "acute," applied to phthisis and tuberculosis, have been variously interpreted by authorities, and this confusion has certainly complicated the study of these diseases. We must abandon the term "galloping," and keep only the epithet "acute," in opposition to that of "chronic." Phthisis is chronic when the lesions slowly and gradually end in ulceration of the lung, cavities, and consumption; it is sub-acute or acute when these lesions run a rapid course, as in certain cases of tubercular pneumonia: the patient then passes through the stages of phthisis in a few months or weeks, instead of several years.

These different forms—chronic phthisis, which is the most common, subacute and acute phthisis—have been described in the preceding chapters. I shall now give the description of another form of tubercular infection—viz., **acute granular tuberculosis, acute granulosis, or granulia.**

In the different varieties of phthisis which we have already reviewed, the grey granulation was of minor importance, while the miliary tubercles and the diffuse or circumscribed tubercular inflammations almost entirely made up the lesion. In **acute granular tuberculosis** the chief lesion is the tubercular granulation, while the hyperæmia and broncho-pulmonary infection only occupy a second place. The tubercles are so confluent, asphyxia and death are so rapid, that the secondary degenerations and the ulcerations of the lung that accompany phthisis have not time to develop. We must, however, remember that the different forms of tubercular infection may exist simultaneously in the same subject, in which case we find post mortem the lesions of chronic phthisis or of acute tubercular pneumonia, and the confluent granulations of acute tuberculosis.

**Pathological Anatomy.**—The lesions of acute tuberculosis differ according as the granulations are or are not accompanied by congestion, bronchitis, broncho-pneumonia, pleurisy, or old tubercular lesions.

1. In some cases the granulations constitute the only lesion, or, at least, the neighbouring inflammation which accompanies them is insignificant; the parenchyma of the lung is riddled with granulations, both on its surface and in its deep parts. On section, the lung appears riddled with granulations; these are almost always quite young, grey, and semitransparent; when they are older they undergo degeneration in the centre. In chronic tuberculosis (ordinary phthisis) the miliary tubercle develops chiefly around the bronchiole, which serves as its centre of formation; but in acute tuberculosis the grey granulation develops chiefly around the bloodvessels and the lymphatics. The structure of the granulations has been described elsewhere.

2. In other cases the granulations of acute tuberculosis are accompanied by intense congestion, capillary bronchitis, broncho-pneumonia, and pleurisy. If the disease has lasted some time, the tubercular granulations and the broncho-pulmonary infiltrations have already undergone initiatory softening and caseation.

3. In a large number of autopsies we find both acute and quite recent crops of granulations, and an old tubercular lesion, or a caseous nodule of broncho-pneumonia. On these facts Bühl founded his theory, according to which the crops of acute tuberculosis are only secondary lesions, grafted on the chronic lesion which has favoured their development.

Cases of secondary infection, well known from Laënnec's time, are very frequent; they demand serious consideration, and prove that an individual attacked by acute tuberculosis has been already in the power of the tubercular infection, because old silent lesions are found in the lung, the brain, or other organs. Cases of secondary acute tuberculosis have been noted in patients primarily affected with tuberculosis of the eye, or with tubercular infiltration of the spine, of the lymphatic glands, etc. Still, acute tuberculosis may be primary, as has been noted, in a large number of cases.

Acute tuberculosis does not, as a rule, remain limited to the respiratory system. It often invades other organs and systems, and then merits the name of **acute tuberculization**. Few organs escape: peritoneum, meninges, pericardium, endocardium, synovial membranes, liver, spleen, kidneys, choroid, thyroid gland, bronchial and mesenteric glands, etc., may be invaded by the tubercular granulations.

The **glands** may rapidly reach an enormous size, and cause certain complications, such as "compression of the bronchi, with symptoms of asphyxia; compression of the mesenteric arteries, with intestinal gangrene; compression of the common bile-duct, with jaundice."

In acute tuberculosis the granulations are usually found in the vascular and lymphatic tissue. Giant cells and bacilli have been recognized in the internal coat of the meningeal vessels and in the fibrinous thrombi of the vessels; they have been seen in the lining membrane of the pulmonary veins (Mügge); in the tunica intima of the endocardium of the right heart; in the vegetations of the endocardium (Weigert); in the walls of the veins; in the tunica intima of the inferior vena cava; in the blood; in the wall of the thoracic duct (Ponfick); and in the walls of the lymphatic vessels: so that acute tuberculosis "results, in all probability, from the entrance of bacteria into the general circulation of the blood or lymph plasma."

**Description.**—Acute tuberculosis shows such different forms that an attempt has been made to classify them, either by the anatomical lesion or by the leading symptom. Although the different classifications proposed



may be purely fanciful and but little in agreement with the clinical data, I feel that they are necessary for a pathological description.

1. There is a catarrhal form, of benign appearance, in which acute and **almost latent** tuberculosis (Leudet) resembles a seasonal fever, with some pulmonary or gastric symptoms; but the patient is rapidly carried off by attacks of suffocation or of syncope after a more or less lengthy illness, which did not appear serious, and was supposed to be bronchitis or influenza. These latent forms are the more insidious inasmuch as they may be apyretic. I have seen a case at the Necker Hospital.

2. The **suffocative** form, which Graves calls acute tubercular asphyxia, and which, says Andral, resembles an acute attack of asthma, is characterized by increasing dyspnoea and asphyxia, which come on without warning, or, at least, in the midst of trifling symptoms. Pain, cough, and expectoration are absent; the fever is moderate, and auscultation shows only some râles. This form, which is often accompanied by broncho-pulmonary congestion, may carry off the patient in four or five days. If anything can give a clue to the diagnosis, it is precisely the absence of signs, or, at least, the disproportion between the gravity of the symptoms and the levity of the physical signs.

3. Acute tuberculosis may be associated with the infections of the respiratory system (diphtheria), and is then partly masked by them. Thus when it takes the form of **capillary bronchitis**, we find cough, dyspnoea, and expectoration. There are the same fine, sibilant and subcrepitant râles; and if other symptoms, such as bloody sputum, enlargement of the spleen, abdominal or cerebral troubles, are not present, the diagnosis is most difficult. When acute tuberculosis is associated with **broncho-pneumonia (acute phthisis)**, the difficulties in diagnosis are the same as in the preceding case. The disease runs its course in a few weeks, or in two to three months; we see the rapid formation of ulcers in the lung and the consumption of the patient, and this variety is confounded with pneumonic phthisis.

In some cases the broncho-pulmonary inflammations give place to pleural inflammation, and the sero-fibrinous or **hæmorrhagic pleurisy** is so prominent that the acute tuberculosis passes unnoticed, until it shows itself by symptoms that have nothing in common with simple pleurisy.

4. The **typhoid** form answers especially to **acute generalized tuberculation**, and closely resembles typhoid fever: headache, insomnia, pulmonary congestion, epistaxis, abdominal and cerebral troubles, and lenticular rose spots, are present. There exist, nevertheless, some distinctive signs which may assist in the diagnosis. In acute tuberculosis the stupor is less profound; hyperæsthesia of the chest is acute (Bouchut); ophthalmoscopic examination sometimes reveals tubercles in the choroid (Bouchut); the stethoscopic signs are more marked at the apex of the lungs, and the sputum is

sometimes mixed with red blood; the patient has attacks of dyspnoea, and the temperature curve presents some differences. While the febrile cycle of enteric fever is made up of three periods, in which the temperature successively ascends, remains stationary, and descends, the temperature in acute tuberculosis is much higher at night than in the morning, and scarcely deviates from this type during the whole disease. Sero-diagnosis (Widal) will remove all doubts between enteric fever and acute tuberculosis.

5. The **cerebral form** may show itself suddenly by loss of consciousness, followed by coma and symptoms of meningitis; these symptoms recall the description of acute hydrocephalus, and, indeed, abundant effusion is found post mortem in the ventricles. The cerebral symptoms do not supervene as a simple episode, as is seen in the other varieties of acute and chronic tuberculization; here they draw all the attention to themselves.

6. **Unusual Forms.**—There are cases which are, indeed, more rare and insidious than the preceding ones, in which tuberculosis shows itself by such unusual symptoms that the nature of the disease is not recognized at first. Colin has published the history of a patient in whom the miliary tubercles primarily appeared in the kidneys, and the disease was at first taken for acute nephritis. In a case of Laveran the tuberculosis first invaded the articular synovial membrane, and for the first few days the disease was looked upon as acute rheumatism.

7. In some cases the tubercular lesions are little marked, but the virulence of the bacillus is exaggerated, and the disease truly deserves the name **acute tubercular infectious fever**. The case looks like typhoid fever, but there is no stupor, and the fever may improve under the influence of antipyrin.

**Diagnosis—Prognosis.**—I have indicated the difficulties in diagnosis with regard to each of the forms which acute granular tuberculosis may assume. A search for **bacilli** in the sputum should never be neglected. It is the most valuable method, and in many cases has cleared up a doubtful diagnosis.

Acute tuberculosis and acute generalized tuberculization are nearly always fatal, and, indeed, some forms carry the patient off in a few days. There are exceptions to this rule. Sometimes the tubercular granulations occur in intermittent crops; the disease is not very acute, and lasts several months. In other cases the acute attack gives place to a chronic state, and acute tuberculosis becomes ordinary pulmonary phthisis. In some cases, when the tubercles have not been confluent, they pass into the **fibrous state**; the disease becomes chronic, and may then be almost harmless, if the patient is not affected with other acute or chronic manifestations of tubercular infection.

Acute tuberculosis especially attacks young people and soldiers, and



the military medical officers have insisted on its **quasi-epidemic** character. It is not rare in very young children, up to the age of fifteen or eighteen months, and although in them it is nearly always generalized, the absence of local symptoms renders the diagnosis very difficult (Parrot).

The **treatment** of acute tuberculosis varies, according to the form that the disease assumes: tannin, in daily doses of 15 grains, and iodide of soda, in daily doses of from 120 to 240 grains (Lépine), appear to have given good results.

## XVII. FALSE TUBERCULOSIS OF THE LUNG.

The **tubercle**, which is a mode of reaction of the tissues against pathogenic agents, is not in any way specific to Koch's bacillary tuberculosis. It may be met with in different affections, characterized histologically by tubercles; these are cases of pseudo-tuberculosis.

The causes of pseudo-tuberculosis are multiple. I would mention certain animal parasites—the *Strongylus vasorum*, whose eggs produce in the dog a tubercular reaction in the pancreas (Laulanié); the *Ollulanus tricuspis*, and the *Pseudalius ovis pulmonalis*, one of which produces pneumonia in the cat and the other in the sheep. At other times bacterial parasites are seen—for example, the bacilli of Malassez and Vignal's zooglœic tuberculosis; also tubercles are produced by fungi, such as *Favus cladothrix* (Sabracès and Dubreuilh), Eppinger's streptothrix (Picot, Rivière, and Sabracès), and the aspergilli, which include *Aspergillus subfuscus* (Olsen and Gade), *Aspergillus nidulans* (Lindt), *Aspergillus flavescens*, and, above all, *Aspergillus fumigatus*. Pseudo-tuberculosis is especially due to the latter fungus, and as it is the only one that has been observed in anything like a complete manner in man, it is the only one we shall describe here, under the name of aspergillary pseudo-tuberculosis.

### Aspergillary Pseudo-Tuberculosis.

In man aspergillary pseudo-tuberculosis (pulmonary aspergillosis) has been met with chiefly in **pigeon-feeders** and **hair-combers**. I have seen it several times in my wards at the Necker Hospital.\*

**Bacteriology.**—The *Aspergillus fumigatus*, which belongs to the order of Ascomycetes and to the family of the Perisporiaceæ, is composed, in the adult state, of a mycelium made up of short alternating hyphæ, which are slightly dilated at their extremities, and give off sterile, septate, and colourless branches, and colourless or slightly coloured fruit-bearing ones. These latter support the spores, which rest on the receptacle or spore-bearing head,

\* Dieulafoy, Chantemesse et Widai, "Une Pseudo-tuberculose Mycosique" (Congrès de Berlin, 1889).

from which they emanate by the strangulation of small cells having the form of quills, and called basidia.

The spores of the *Aspergillus fumigatus* are green or brownish, according to the media. Their maximum development takes place at 98° F.—that is, at a temperature nearly the same as that of the human body. These spores grow very well on Raulin's liquid, on beer wort, and Sabouraud's maltose; their colour is brown on the former and green on the latter culture medium. In certain cases they may take on a yellowish colour. Gelatine is liquefied by *Aspergillus fumigatus*.

In animals the pathogenic action of this fungus is well marked. Pigeons die three or four days after inoculation of the spores in the axillary vein; rabbits die in six or eight days, and guinea-pigs in four or five days, after injection into the veins of the ear. The ape is less sensitive; and this pathogenic action, which is nil in the sheep, is present in the case of dogs and cats (Saxer). Inhalation into the trachea kills pigeons in from twelve days to a fortnight. The ingestion of spores produces exceptionally in the rabbit tubercular lesions of the intestine, which may go on to perforation (Rénon).

Spontaneous aspergillosis is fairly frequent in birds; it may invade the eggs during incubation, and transmit the disease to the chick (Leucet). It is fairly rare in Mammiferæ, where it takes the form either of chronic phthisis or of superacute hæmorrhagic septicæmia (Leucet). The spores of the *Aspergillus fumigatus* are present in the air, upon trees, in the most superficial layers of the soil, and are also found in the nasal mucosa and the saliva of persons who are healthy, or who are suffering from the most diverse affections. The surface of seeds is, however, their chosen haunt (Rénon).

The resistance of the spores is considerable, and their vitality is enormous. They still reproduce themselves after two or three years' sojourn in an old culture, but their virulence is, in consequence, found to be attenuated. Their vitality is weakened by a more or less prolonged sojourn in organic membranes (Rénon). Heat kills them, and they then become harmless to animals; but the animal is none the less susceptible to the injection of virulent spores, and appears to succumb the more quickly, as the degree of heat necessary for the sterilization of the primarily injected spores has been high. By a progressive inoculation with virulent spores, rabbits can be made to stand considerable doses, that would rapidly kill the control animals.

The *Aspergillus fumigatus* does not produce toxins. Neither culture fluids nor substances extracted from the mycelium possess any vaccinal powers (Kotliar, Rénon). In the rabbit the spores traverse the placenta, and are directly transmitted from the mother to the foetus.

**Ætiology—Pathogenesis.**—It is interesting to consider how pigeon-feeders may take the disease. Infected pigeons present on the floor of the mouth a small tumour, or chancre, which may cause a mouth-to-mouth



contamination. It is probable that more usually pigeon and feeder find the common cause of their disease in the spores of the *Aspergillus fumigatus*, which are present on the grains of millet and vetch.

Hair-combers may also contract aspergillary tuberculosis. They are infected from the flour which they rub on the hair to remove the grease. This flour contains many spores, while the undressed hairs collected every morning in the dirt-boxes by the rag-pickers of Paris scarcely contain any of them. Birds living in this dusty atmosphere succumb in a fortnight to three weeks; pigeons that are made to inhale this dust die of aspergillary tuberculosis.

In some cases aspergillosis finds in man a soil perfectly prepared by previous inflammation of the broncho-pulmonary system. The affection is then secondary; its progress is slow, and almost always masked by the symptoms of the primary disease; it is not recognized, as a rule, until the autopsy. In other cases aspergillary pseudo-tuberculosis is a primary or autonomous affection that is identical with the disease seen in animals, and it is this form that I have especially in view in this chapter. The French conception of primary aspergillosis (Dieulafoy, Chantemesse and Widal, Potain, Rénon, Gaucher and Sergent), after having been actively attacked in Germany, is now completely admitted; it is indisputable, for it corresponds exactly with anatomical, experimental, and clinical facts (Rénon). Cases have been multiplied during the last few years, and, according to Saxer, primary aspergillosis is much more frequent than is usually supposed.

The only difference that still separates the French from the German school consists in the term "pseudo-tuberculosis," which is applied in France to the primary aspergillary ulcerative process in the lung. If we remember that this affection, which destroys the lung tissue, presents all the clinical signs of tuberculosis, we see that we are very nearly in agreement, and that in every case the idea of aspergillosis as a primary disease receives a striking confirmation.

**Symptoms.**—Aspergillary pseudo-tuberculosis presents various forms. The disease may begin with slight or with abundant hæmoptysis, followed in general by other attacks at intervals of several months, or of one or two years.

At the same time, fatigue, loss of strength, with dyspepsia and anorexia, appear. The cough is dry, and recurs in fits. The expectoration, at first frothy, becomes greenish and purulent, and the sputum is often streaked with blood. The signs of pulmonary tuberculosis in its first stage are found—namely, slight induration at one apex, with harsh breathing and sometimes prolonged expiration. A rise of temperature to 102° F., with or without night sweats, may be noted; sometimes, also, pleurisy, with or without effusion, may occur. In some patients hæmoptysis is rare, and the signs of bronchitis are most prominent. The cough is incessant, and suffo-

cation is intense, especially during the night. Indeed, attacks of "pseudo-asthma" (Rénon) are seen; the breathlessness diminishes during the day, but only if the patient makes no active efforts. The sputum is greenish, purulent, and sometimes nummular. During the attacks a *bruit de tempête*, with snoring, sibilant and subcrepitant râles, is heard; between the attacks, symptoms of pulmonary induration at the apex of the lung may be found.

As a rule, the other organs are healthy, the liver and spleen being specially immune.

The course of the disease is not always progressive, and slight improvement of variable duration may alternate with transient aggravation. There is not, as in ordinary tuberculosis, a gradual and progressive extension of the lesions. After a period of transitory cachexia, the patients regain their good condition, and sometimes resemble tubercular patients so little that they would not be considered sick if auscultation were not performed. Retrogression is therefore nearly always the rule; the aspergillus gradually disappears from the expectoration, and a definite cure may be caused by fibrosis, as is seen in the case of animals.

The most formidable complication is the invasion of the lungs by Koch's bacillus, which gradually takes the place of the fungus; the course is then that of an ordinary pulmonary tuberculosis, accompanied by local lesions, such as anal fistula or cutaneous tuberculosis. This fibro-plastic action of the aspergillus, however, helps the struggle against the new parasite; but the curative process may overshoot the mark, and in one case Rénon and Sergent have noted that fibrosis became, in its turn, the chief complication, and the patient died of dilatation of the heart, with asystole.

The duration of aspergillary tuberculosis is very long; it has lasted three, six, eight years and more in the cases that have so far been observed.

The *Aspergillus fumigatus* may not harm the lung, and may only invade the bronchial system. The result is a peculiar membranous bronchitis, which was essentially chronic in the two known cases. The membranes, composed solely of mycelium and spores, were situated in the large bronchi, and expelled almost every month, with a crisis of acute dyspnoea (Obici, Rénon, and Devilliers).

**Diagnosis.**—It is important to make an early diagnosis, but this is impossible by clinical methods alone. We should think of the aspergillus when, in a patient who has tubercular lesions, recognized as such by physical examination, the course of events is slow and the general health remains good. The probability will become much greater if the patient be exposed in his work to handling grain or flour (pigeon-feeders, hair-combers, millers, seedsmen, etc.); and it will become a certainty if Koch's bacillus be absent from the sputum and the mycelium be present. The diagnosis, therefore, entirely rests on bacteriological examination.



In searching for the bacilli, the Ziehl-Kühne method should be employed. If the result is negative, we must prove the absence of the bacilli by inoculation of a guinea-pig with the sputum. If thirty or forty days afterwards the animal presents no tubercular lesion (verified bacteriologically) at the point of inoculation, the question is decided: it is not a case of Koch's tuberculosis.

The search for fragments of mycelium in the sputum should be made with an aqueous solution of saffranin, or, better still, by staining with thionin. If the search is negative, recourse may be had to cultures. If it is positive, the same procedure should be employed, to make certain that the fragments are really those of the aspergillary mycelium. The fresh sputum, collected aseptically, should be sown in tubes of sterilized Raulin's liquid, and placed in an oven at 98° F. If the sputum contains spores or mycelium, we shall, by the second day, see that isolated filaments, which are united into a tuft of mycelium, rise up from the sown particle; the mycelium will rise gradually, and take from three to six days to reach the surface. Some hours later it will form a whitish, velvety, and absolutely characteristic carpet, which twenty hours later is covered with greenish spores, that assume a smoky-black colour in a few days. We must then verify the pathogenic action of the fungus thus found on the rabbit, and absolutely prove the *Aspergillus fumigatus*, since the two other species, *Aspergillus niger* and *Aspergillus glaucus*, which develop under these conditions, are non-pathogenic. The animal will succumb in a few days to a generalized aspergillary tuberculosis of all the viscera, but especially of the kidneys, and a fragment of the latter organ, sown in a tube of Raulin's liquid, will in five or six days reproduce a culture of the *Aspergillus fumigatus*. The cycle will be complete, and absolutely no room will be left to doubt the existence of the fungus in the sputum.

We can thus eliminate asthma, chronic bronchitis, and Koch's tuberculosis. The pseudo-tuberculosis produced by the *Rhizomucor parasiticus* (Lucet and Constantin) closely resembles aspergillosis; minute examination of the parasite found in the sputum and cultures will alone prevent error. Actinomycosis of the apex of the lung is often accompanied by chocolate-coloured expectoration, which consists of a mixture of blood and pus; the peculiar grains of the actinomyces are found in it. In the exceptional cases of pulmonary mycosis, due to Eppinger's streptothrix, the form of the mycelium in the sputum is different, and cultures decide the question.

**Prognosis.**—The prognosis of aspergillary pseudo-tuberculosis is relatively good; this does not obtain in pseudo-tuberculosis complicated by Koch's bacillus. The remissions are less frequent and less prolonged; the pulmonary signs are more marked, and death may supervene at a distant date.

**Pathological Anatomy.**—When aspergillary pseudo-tuberculosis is secondary, and occurs as a complication of chronic bronchitis or of previous pulmonary tuberculosis, it is, as a rule, a post-mortem discovery: small velvety, greenish, or brownish tufts, composed of adherent mycelium, are found. The cavity contains full-blown spores. Outside this infiltration of the walls of the cavities by the fungus we may see (Lichtheim, Cohnheim, Fürbinger) tubercles of the size of a nut or of a millet-seed, which, under the microscope, show an abundant mycelium extending from the tubercle to the alveoli.

The lesions of simple and primary aspergillary pseudo-tuberculosis have been studied in man (Ribbert, Boyce, Saxer), and also in animals (Dieulafoy, Chantemesse and Widal, Ribbert, Rénon, Obici, Saxer). Microscopically there is no difference between the tubercle due to the aspergillus and that due to Koch's bacillus. In pigeons the lesions affect especially the lung and the liver; in rabbits they affect the kidneys; and pleurisy, enteritis with perforation of the gut, peritonitis, cystitis, and osteitis of the vertebræ, with congestive abscess, simulating Pott's disease, may be noted. All these lesions are tubercular in form. The mycelium is passed in the urine, when the renal changes are marked (Rénon).

The tubercles vary in size from a pin's head to a small pea; they may undergo vitreous degeneration and calcification, with formation of true cavities; at other times we find a tubercular infiltration *en nappe*. Aspergillary tuberculosis may pass into a fibrous condition, which is one of its active modes of cure. This process is also seen in man, even when bacillosis is present as a complication. At the autopsy of a pigeon-feeder, affected in succession by these two maladies, Rénon and Sergent noted marked lesions of chronic pneumonia; the fibrous tissue extended from the bronchi to the pleura, choking the lung tissue proper.

"The histological lesions are in every way comparable with those of bacillary tuberculosis. In a section of the lung we see a large number of tubercular nodules, surrounded at their periphery by giant cells. The growth of these nodules can easily be followed. The young ones are formed by an agglomeration of leucocytes or of epithelial cells around one or several branches of mycelium. The older granulations present in their centre a feltwork of mycelium, the interlacing branches of which stain better at the periphery, in the immediate neighbourhood of the giant cells. In certain cases the tubercle is solely represented by a very large cell with multiple nuclei; while the protoplasm contains a ramification of mycelium, either alive and well stained, or altered in structure: moniliform, unstained, and partly digested by phagocytes" (Dieulafoy, Chantemesse and Widal). In chronic cases we sometimes find in the tubercle tufts of abundant mycelium, presenting a great likeness to actinomycosis (Laulanié, Rénon,



Ribbert, Boyce) and to the actinomycotic forms of Koch's bacillus (Rénon).

Indeed, according to the German school, the fungus plays the chief part in the production of the histological lesions in the human lung. The aspergillus is said to provoke foci of necrosis, which, by elimination of their contents, cause cavities. This process is said to be specific (Saxer). We see, however, that two at first irreconcilable opinions are almost brought into harmony, although the primary pathogenic action of the parasite has remained a subject of active discussion.

**Treatment.**—The treatment is symptomatic and general. Hæmorrhage must be treated by the means given under Pulmonary Tuberculosis. Bronchitis may be alleviated with creosote and terpene, and tincture of lobelia, with iodide of potash, which has given fair results in animals, should be employed for the attacks of suffocation (Rénon). The general condition should be maintained by superalimentation and by large doses of cod-liver oil (3 to 5 ounces daily), and residence in the country, at the seaside, or in a climate at high altitude should be advised.

### XVIII. CANCER OF THE LUNG.

**Ætiology.**—Cancer of the lung may be primary or secondary; the former is rare, the latter common.

The growth is frequently secondary to cancer of the breast, which extends to the parietal pleura, when the subpleural lymphatics carry cancer cells to the lung. The mechanism is the same in the propagation of cancers from the mediastinum to the lung.

Cancer of the lung is sometimes secondary to that of the abdominal organs—glands, stomach, intestines, liver, and ovary. The spread of cancer from these organs to the lung takes place in different ways: by venous emboli, following the course of the portal vein, vena cava, right heart, and pulmonary artery; by way of the lymphatics, the cancer reaching the peritoneum over the diaphragm and passing through this muscle by means of the lymphatic communication existing between the peritoneum and the pleura, and invading the visceral pleura and lung. When cancer is consecutive to that of the limbs or of the head, the propagation takes place by the venous channels.

**Pathological Anatomy.**—Cancer of the lung may be lobar or diffuse. The lobar variety forms a bulky mass, which may involve or compress the neighbouring organs (trachea, œsophagus, arteries, and veins); in the diffuse form the growth is disseminated in the form of nodules in the deep layers, or on the surface of the organ.

Lobar cancer is usually primary and unilateral. Diffuse or nodular cancer is nearly always secondary, and affects both lungs; the cancerous nodules may be superficial (subpleural) or deep (intrapulmonary). They are of all sizes; some are no larger than a pin's head, and the condition is called miliary carcinosis, from its resemblance to tuberculosis; other nodules are as large as a pea or a walnut.

Primary cancer nearly always assumes the **encephaloid** form. Secondary cancer is a reproduction of the parent growth, which may be scirrhous, melanotic, colloid, or adenomatous (Marfan). The cancerous mass may finally become softened; in some cases it forms a bloody pulp, the elimination of which may give rise to a cavity.

Microscopic examination in primary cancer shows the alveoli blocked by spherical or polygonal cells, with large ovoid nuclei. The alveolar walls are usually normal. "There is no stroma of new formation in cancer of the lung, and it is the fibrous framework of this organ which takes its place." The epithelial origin has been definitely shown by Malassez. Cancer arises in the epithelium; it is uncertain whether the bronchial or glandular epithelium may not give rise to it.

All the structures in the mediastinum may be invaded by cancer of the lung. The lymphatic vessels and the corresponding glands (cervical and axillary glands), especially the bronchial ones, may show simple inflammation or cancerous change. The pleura is usually involved in cancer of the lung; the result is pleurisy, with effusion, which is very often hæmorrhagic (see Hæmorrhagic Pleurisy).

**Description.**—In a description of cancer of the lung it is necessary to distinguish the symptoms which properly belong to it from those which depend on invasion of the mediastinum and of the pleura; this distinction, however, is very difficult, for cancer rarely remains confined to the lung without affecting the pleura or the glands of the mediastinum.

The symptoms proper to cancer of the lung are somewhat limited. The patient complains of pain (pain in the side, which may or may not be radiating) of increasing intensity; the pain may be brachial, cervical, or intercostal, and accompanied by zona. Cough is a usual symptom. Dyspnoea may be slight, severe, continuous, or paroxysmal, with or without stridor, depending on the multiplicity of its causes. Compression of the trachea and the bronchi, compression of the vagus and recurrent nerves, lesions of the pleura, and pleural effusion, may all cause dyspnoea.

**Hæmoptysis** is fairly frequent, and some authors have given as characteristic the currant-jelly-like expectoration which contains cancer elements and elastic fibres from the lung. I have lately seen a typical case of this expectoration, with Dr. Marcano, in cancer of the lung, secondary to that of the breast. According as the cancer is lobar or diffuse, the dullness yields



more or less precise information ; auscultation may sometimes show tubular breathing and bronchophony.

When the bronchial glands are invaded by cancer, the symptom-complex, which we shall study under Tumours of the Mediastinum, is found. I shall here simply mention two of these symptoms : paroxysmal or intermittent dyspnoea (compression of the vagus and phrenic nerve) and cough, which is often analogous to the fits of whooping-cough.

The cancerous mass may also cause compression of one recurrent nerve (dyspnoea and spasm of the glottis), of the œsophagus (dysphagia), or of the venous channels (œdema of the face and neck and supplementary circulation).

It sometimes happens that acute, subacute, or latent pleurisy masks the development of cancer of the lung ; the patient only complains when dyspnoea, due to effusion or to other causes, has become severe. Pleurisy is discovered ; thoracentesis is performed, and fluid, which is most frequently blood-stained, is drawn off ; and yet, in spite of the operation, pain, cough, and dyspnoea continue. I have seen several cases—one, among others, with Dr. Auburtin. The patient had a considerable pleural effusion. I evacuated seven pints of blood-stained fluid in four sittings. The improvement was of short duration, and, though the effusion did not recur, the cancer continued its progress. In one of the following sections we shall see the importance of hæmorrhagic pleurisy in cancer.

After a duration of from one month to two years, cancer ends in death. Death which supervenes from increasing dyspnoea and asphyxia is terribly painful ; injections of morphia are in such cases the only palliative. Rapid or even sudden death has often been noted. In other cases the patient dies in a state of asystole, with general œdema, cyanosis, and coma. Sometimes hectic fever supervenes and ends the scene.

The **diagnosis** of cancer of the lung presents serious difficulties, especially when it is primary and runs an acute course. We are so used to the slow progress of cancer, to the gradual breaking-up of the individual, and to the characteristic colour of the skin, that the diagnosis is often at fault when cancer runs an acute course. This acute course is not rare in cancer of the lung. An individual in good health shows symptoms which might quite well be put down to acute phthisis. He dies in a few weeks, and cancer of the lung is found post mortem.

In doubtful cases we must never omit to look for cancer in other organs—*e.g.*, cancerous nodules under the skin, cancer of the liver, rectum, testis, or uterus. A previous operation or the presence of a scar (breast) may give a clue. I have twice seen cancer of the lung in patients who had undergone operation—the one for disease of the left testis, the other for an osteo-

sarcoma of the knee.\* It is also necessary to make sure of the condition of the corresponding axillary or cervical glands; this evidence of cancer, although inconstant, is none the less valuable when it exists.

### XIX. BRONCHO-PULMONARY LITHIASIS.

**Pathological Anatomy and Pathology.**—"Lung stones," or broncho-pulmonary lithiasis, has been well described by Poulalion. From the histological point of view, they may be divided into three categories—cartilaginous or cartilaginiform, bony, and calcareous bodies. We must also differentiate the growths and the changes which take place in the thickness of the broncho-pleuro-pulmonary tissues, and constitute parenchymatous, cartilaginous, bony, or calcareous concretions, from those which occur in the interior of normal or accidental cavities in the respiratory system, and are always calcareous in nature, constituting calculi, properly speaking (broncholiths).

1. The **cartilaginous** or **cartilaginiform** growths are made up of cartilaginous or other dense fibroid tissue; they may be situated in the walls of the bronchi, in the pleura, or in the lung tissue. They have the appearance of cartilage, and are resistant, elastic, whitish, opaline, and of a bluish sheen.

2. **Bony** growths are characterized by the existence of osteoblasts and the presence of newly-formed Haversian canals. These bony calculi arise in ossified bronchial cartilages (bronchial dilatation, pulmonary phthisis), in ossifications of the tracheo-bronchial mucosa, in ossifications of the pleura, developed in the fibrous shell of old pleurisies.

3. **Calcareous** growths result from calcification of the different tissues in the respiratory system, by incrustation with particles of tribasic phosphate of lime and carbonate of lime. Among the lesions of the lung which may undergo calcareous transformation we must notice, in the first place, the caseous tubercle, then infarcts, broncho-pneumonic nodules, miliary abscesses, pseudo-tubercles of actinomycosis or of the aspergillus, and, lastly, cysts and tumours of the lung.

The lung may only contain some isolated, calcareous concretions, as is the case in the caseous nodules of ordinary tuberculosis which is in process of cicatrization and cure. At other times, on the contrary, the concretions are in considerable number, and the lung tissue is, as it were, riddled by them; this condition is called calcareous granulositis of the lung (Poulalion).

The **parenchymatous** concretions may become stationary and remain latent, but in other cases they may undergo a process of enucleation which

\* This patient, whom I saw with Dechambre, was suffering from cancer of the upper left lobe, which presented the appearance of encysted pleurisy.



causes their migration into the tissues, and generally ends by their passing into the air-passages.

**Intracavitary** calculi present the most marked analogy with biliary and urinary calculi; they may, like the latter, have as their nucleus of origin foreign bodies from without, or parenchymatous concretions which have been set free; they are formed in the bronchial channels, or in cavities accidentally developed.

**Symptoms.**—In some cases broncho-pulmonary lithiasis, especially in its parenchymatous forms, may be latent, and only be discovered post mortem. As a rule, the presence of calculi in the respiratory passages produces troubles similar to those which occur in the biliary or in the urinary tracts. True crises of bronchial and pulmonary colic occur. Sometimes the expulsion of the concretion takes place without the patient perceiving it, and he expectorates the calculus while coughing; at other times, on the contrary, as happened to a patient in my wards, the expulsion is preceded by heaviness, dyspnœa, pain, constriction, anguish, and a feeling of tearing, either in the sternal region or at the sides of the chest. These pains are almost always followed by obstinate, jerky cough, during which the patient suddenly experiences a sharp, tearing sensation in the larynx, and increase of the dyspnœa, followed immediately by the expulsion of a hard body, which may strike against the back of the incisor teeth. The expectoration of the calculus being accomplished, the **bronchial colic** ends, and the cough and pain frequently yield at the same time. Sometimes it is only a case of abortive bronchial colic; the patient, while coughing, experiences a painful sensation or feeling of a foreign body rising in the trachea, and then passing back again into the bronchi and lung. The duration of the crisis is very variable. It may last a few moments, or even some hours—as many as forty-eight (Poulalion). The number and size of the calculi brought up are also variable; as many as 400 have been counted, and may be as large as a pin's head, a millet-seed, or a nut.

The expectoration of calculi is often accompanied by hæmoptysis, which, though generally slight, is sometimes fulminant; the bleeding may precede the expectoration of the calculus by some days, but as a rule accompanies it.

We may see fever due to neighbouring inflammation, or to the action of the calculus on the altered mucosa, with consecutive absorption of septic products. Examination of the chest before, during, or after the crisis most often yields but trifling information; bronchitic râles are heard, and it is only in obstruction of a large bronchus by a concretion that we can recognize below the obstacle more or less extensive absence of breath-sounds.

Broncho-pulmonary lithiasis may occur in the course of tuberculosis, and favour the development of the latter trouble.

In other cases the chronic course of lithiasis simulates phthisis, although absolutely no tuberculosis is present ; this is known as **pulmonary pseudo-phthisis of calcareous origin** (Poulalion). During a more or less lengthy period the patient suffers from cough, which is at first dry, but later is accompanied by mucous or muco-purulent sputum. These symptoms become worse ; signs of induration, of pulmonary congestion, or of localized bronchitis, and even those of small cavities, are often found. The general condition becomes bad ; wasting and night-sweats appear. Hæmoptysis is common. The attention, however, is chiefly attracted to the dyspnœa and the pain ; both come on in more or less intense attacks, until in a more violent fit of coughing than usual the patient brings up the calculus, with or without hæmoptysis. The symptoms now show marked improvement, when only one calculus exists ; but the relief is temporary when there are several calculi, and their expulsion is always preceded by a period of aggravation. In this form Koch's bacilli are never found in the sputum.

Cure is the rule when there are no complications. As complications, I may note acute bronchitis, which generally ends favourably ; pleuropneumonia, which is often fatal ; abscess of the lung, which sometimes opens up the bronchi, or ends in perforation of the pleura and pyopneumothorax. Sudden death from obstruction of a large bronchus by a calculus has been noted (Tice).

**Diagnosis.**—The diagnosis is almost always impossible before expulsion of the calculus. Cough, dyspnœa, and pain in the chest are quite insufficient signs, and the rejection of the calculus must be waited for. We must then ascertain whether the patient is tubercular or not, and whether the concretion is of intraparenchymatous or intracavitary origin. Examination of the sputum for bacilli and inoculation of the guinea-pig will help to decide the first point ; as regards the second, histological examination of the cut section will establish it.

The differential diagnosis must be made from fragments of bone that are coughed up, but do not come from the respiratory system ; from portions of the vertebræ in Pott's disease (Chénieux) ; from a sequestrum from the larynx ; calcareous concretions formed in the ventricles of the larynx (Pravaz) ; concretions from the crypts of the tonsils ; rhinoliths which have fallen into the pharynx ; and, lastly, foreign bodies which have reached the lung from without.

The **prognosis** of calculous pseudo-phthisis is not grave when the patient brings up the foreign body ; yet apart from the complications mentioned above, the prognosis is much affected by the weakened condition of the lung and the possible development of tuberculosis.

**Treatment** can only be symptomatic ; surgical intervention appears hardly possible, because precise indications are wanting as to the seat of the



calculus. If calcareous change in tubercles is favourable to their cure, we should assist this calcification by the use of soluble phosphates and by nourishment which contains much vegetable matter.

## XX. HYDATID CYSTS OF THE LUNG AND OF THE PLEURA.

**Pathological Anatomy.**—In frequency hydatid cysts of the lung come next to those of the liver—that is to say, we see them fairly often. As I have described in detail (see Liver) the life history of the hydatid, I shall here notice only the characters peculiar to hydatid of the lung.

The cyst usually affects the right base. It is sometimes associated with a cyst in the liver. The pulmonary cyst is unilocular, the alveolar cyst being extremely rare. In order to reach the lung, the embryo follows various routes. It may enter the respiratory tract by aspiration of dust; it may be ingested with food and drink, pass from the intestine into the portal veins, traverse the liver, the subhepatic veins, the vena cava, and the heart, to be arrested in the lung. The embryo may perhaps enter the hæmorrhoidal veins, pass through the pudic and the internal iliac veins, reach the inferior vena cava, without passing into the liver (Chachereau), and travel through the heart into the lung. In the case of coexistence of hydatid of the liver and of the lung, it may be asked if the embryo has not migrated directly from the one organ to the other.

The adventitious covering of hydatid cysts of the lung is **very thin**; it may be completely absent, and this fact will explain why the cyst so readily opens into the bronchi. Hydatid cysts of the pleura are rare, unless the pleura has been invaded secondarily by a pulmonary cyst.

**Description.**—As the lung is less tolerant than the liver, the early growth of the pulmonary cyst is rarely quite latent. In the liver, cysts may be present for a long while and may attain large proportions without producing symptoms or results; enlargement of the right hypochondrium is sometimes the first sign of the hydatid cyst. In the lung, on the other hand, it is exceptional for the hydatid to remain quiet long; indeed, its presence may be revealed early by important symptoms, of which hæmoptysis is the most striking.

**Period of Onset.**—Dry, jerky **cough** may be the only symptom for weeks. It is the result of a reflex, and simulates the cough of tuberculosis, with this difference, however—that the cough in tuberculosis is almost always followed by some expectoration.

The **pain**, which is rarely sharp at this stage, may simulate pleuritic pain or intercostal neuralgia; in some cases it is obstinate, and radiates into the neck, the shoulder, and the epigastrium.

**Dyspnœa** is present at an advanced stage of the malady, and in complications ; although it is rare during the early growth of the hydatid, it has been noted in some cases.

**Hæmoptysis**, from its importance and its frequency, deserves careful attention, and, while it may be either slight or severe, and more or less repeated, plays a large part in the history of pulmonary hydatids. Early hæmoptysis, arising at the onset of the malady—before any other symptom, indeed—and late hæmoptysis, coincident with the opening of the cyst, are both seen.

**Early hæmoptysis** comes on as a precursory sign in this disease, as in many pulmonary affections. It is, indeed, remarkable that the first cry of revolt on the part of the lung against the invader is perhaps a means of defence. Since phagocytosis is insufficient to meet the attack, the vessels take part, and it may be said that the lung seeks to get rid of its adversary by the ejaculation of blood ; it sometimes succeeds, and the hæmoptysis, having no ill results, is then termed “ essential.”

I have named this early hæmoptysis “**defensive.**” It is very frequent in **pulmonary tuberculosis** ; tubercular hæmoptysis may, indeed, arise in the course of apparently excellent health, when no suspicion of tuberculosis exists. Parents who have suffered from hæmoptysis may beget tubercular children, although the former may have had no other sign of tuberculosis than the hæmoptysis, which has left no traces.

We also find **early hæmoptysis** in false pulmonary tuberculosis ; I have seen it many times in pigeon-feeders affected with aspergillary tuberculosis (*vide* Chapter IV., section xvii.).

Early hæmoptysis is also seen in patients with broncho-pulmonary concretions, who are suspected of tuberculosis, until they bring up the concretions during an attack of bronchial colic. Hydatid cysts of the lung especially provoke early hæmoptysis, as will be seen from the following examples, taken from my clinical lecture on the subject :\*

A case sent to me by Dr. Leroy :

On May 22, 1898, the first hæmoptysis came on, without appreciable cause, and a so-called pleuritic pain appeared on the right side. Four months later fresh hæmoptysis (about a pint of bright frothy blood) suddenly took place ; obstinate cough supervened, and the patient was convinced that he had tuberculosis. He was treated without success, for his strength gradually decreased. At intervals pain reappeared on the right side ; appetite diminished, and six months later he had lost 32 pounds in weight.

Next year the same symptoms were present : frequent fits of coughing, and further spitting of blood. Four large hæmoptyses were recorded. On each occasion the hæmoptysis was treated with ergotin, Rabel-water, and applications of ice, with absolute rest in bed. Each bleeding left the patient still more feeble ; though he had no fever, he coughed continually. The situation became worse, and the diagnosis of hæmoptoic tuberculosis appeared no longer doubtful.

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\* Dieulafoy, “ Les Hémoptysies des Kystes Hydatiques du Poumon ” (*Clinique Médicale de l'Hôtel-Dieu, Paris, 1905*), 16<sup>me</sup> leçon.



A decisive incident, however, revealed the true nature of the affection. On November 12, 1899, the cough became more violent than ever, and he coughed up much blood-stained sputum and a large piece of hydatid membrane. The thoracic pain, the cough, the numerous hæmorrhages, were due, not to tuberculosis, but to a hydatid cyst of the lung, which had previously given no definite sign.

The expulsion of hydatid membranes and the attacks of hæmoptysis recurred on several occasions. The expulsion of hydatid membranes was almost always heralded, twenty-four hours in advance, by fits of coughing and by more or less abundant hæmoptysis. On the other hand, some of the hæmorrhages were not followed by expulsion of hydatid membrane. From March 24, 1900, to the beginning of April, 1901, thirteen large hæmorrhages were counted, without expulsion of pus or of membrane. They continued during May, but it was not till August 25 that pus and large membranes were brought up. During the last four months of 1901 the hæmorrhages recurred, and were always followed by the coughing up of hydatid membranes, with or without purulent sputum. In 1902 hæmoptysis, membranes coughed up; from May 22, 1898, to April 12, 1902, hæmorrhage on sixty different occasions, and membranes coughed up on forty-three. He is now cured.

#### Case published by Lavéran :

A soldier, twenty-six years of age, was in excellent health up to the end of October. He practised fencing a great deal, and in the latter part of October, during an assault-at-arms, he suddenly felt sharp pain in the chest, and brought up about a tumblerful of bright red blood. After a few days he resumed his duties, but soon felt pain on both sides of the chest, and had fresh hæmorrhage. On December 5 he was sent to a military hospital, where phthisis was diagnosed, and he was invalided out. Next April the cause of these hæmorrhages was discovered: the patient coughed up pus containing hydatid membranes, and thus got rid of his hydatid cyst seven months after the first spitting of blood.

#### A medical student has published his own case :

After an attack of pleurisy the patient had slight hæmoptysis, in April. The sputum was frothy and tinged with bright blood, while signs of congestion and crepitant râles, were found at the right apex. During May the patient was treated with quinine, creasote, etc. Fever appeared, and he lost his appetite. On May 25, fresh hæmoptysis. Finally, after a series of troubles, thought to be tubercular, in January 13 of the next year, he found a piece of hydatid membrane, 2 inches square, in the sputum.

#### Watelet relates the following case :

A man of forty was taken ill with hæmoptysis and wasting; râles in the left lung; tuberculosis suspected. Four months later fresh hæmoptysis, fœtid sputum, and expulsion of enormous hydatid membrane.

#### Landouzy writes :

A woman had five hæmorrhages three months before the rupture of the pulmonary cyst. The blood was red and frothy; the quantity about half a tumblerful on each occasion. She had been considered tubercular.

Fenger and Hollister speak of a patient who had attacks of hæmoptysis for twelve years. Later he brought up the cyst; pneumotomy became necessary, and he recovered completely.

In a case of hydatid of the lung reported by Delgrange hæmoptysis, which was sometimes trifling, sometimes very profuse, persisted for five months.

In his admirable lecture on hydatids of the lung Trousseau has been careful to say that hæmoptysis has been noted in almost every case, and, among other examples, quotes Mercier's case :

A man was subject to hæmoptysis for several years, but showed no other signs of tuberculosis ; he was suddenly seized with acute pain in the right side ; examination of the chest revealed hydropneumothorax. Post mortem hydatid of the lung was found ; it had caused perforation of the pleura and ulceration of a bronchus.

A gentleman from the Argentine Republic, thinking himself affected with pulmonary tuberculosis, consulted me at the beginning of 1902. He complained of obstinate cough for two months and frequent hæmoptysis. No sputum. On auscultation, I discovered no trace of tuberculosis, neither râles nor dullness being present. Examination of the sputum failed us, because the patient brought up none. The appetite was bad, and the man was wasting. I left the diagnosis open. After an interval of some weeks I saw the patient a second and third time, and the most minute investigation revealed nothing. The hæmoptysis continued.

One day the patient brought me a bottle containing a cloudy liquid, with a quantity of hydatid shreds, which he had brought up during the night, after terrible fits of coughing and of breathlessness, which almost amounted to suffocation. The diagnosis was clear, and I took the offending body to the Hôtel-Dieu, telling my pupils the history of this patient.

Hearn, in his work, which comprises 144 cases of hydatid of the lung, gives prominence to the frequency of hæmoptysis. "In less than a fifth of my cases," says he, "there is no mention of it."

In Iceland, where hydatid disease is so frequent, Finsen says that it is almost possible to diagnose pulmonary echinococcus from spitting of blood.

Vegar and Cranwell, in their monograph on hydatid cysts in the Argentine Republic, say that hæmoptysis constitutes one of the most important symptoms in cysts of the lung.

Widal has reported a case of hydatid cyst of the lung in which daily hæmoptysis without fever and expectoration remained the chief symptom for four months.

To sum up, hæmoptysis, whether early or anterior to the rupture of the cyst, presents different forms. In some cases hæmoptysis is reduced to a minimum. We see bloody, brownish, or reddish sputum of a gooseberry or currant colour. This sputum is coughed up, and may recur several times in the day for weeks or for months, with or without periods of arrest. Sometimes bright red blood is brought up at more or less definite intervals for months or even years.

On examining the cases, we see that hæmoptysis may precede the other signs. I think that the embryo may, from the moment of its fixation in the lung, cause hæmoptysis, and I believe that hæmoptysis may be repeated when the hydatid cyst is only as big as a pin's head, a small pea, or a nut, and may go on during the growth of the cyst before its rupture. Such is the early hæmoptysis. It is evident that we must recognize it.

The above details will, I think, suffice as regards **early** hæmoptysis



which accompanies the growth in the lung; subsequently we shall have to study the late hæmoptysis which accompanies rupture of the cyst.

Let us now consider pleurisy—a very rare complication, it is true, but one which may nevertheless supervene from the first.

It is important to remember that pleurisy may supervene at the onset of a pulmonary hydatid. I do not speak, of course, of hydatid of the pleura, which is extremely rare, as we shall see later; I allude to those cases of pleurisy which develop in the ordinary way from the onset of the pulmonary cyst. I do not know its exact pathogeny, but it is certain that pleurisy may develop in hydatid of the lung, as in that of the liver—*e.g.* :

A hospital attendant, who had been treated for a month for pleurisy, which was cured, coughed up four months later foetid sputum and hydatid membranes.

A medical student, Marconnet, was taken ill with pleurisy, which preceded the other symptoms of hydatid. “The numerous attacks of pleurisy,” says Marconnet, “from which I suffered during my disease were assuredly due to the formation of the cyst. They were, moreover, so strange that they puzzled my doctors. The effusions disappeared as if by magic. If it be admitted that my first pleurisy may have been primary, and that it was not the result, but the cause, of localizing the hexacanthus, how can the pleurisies which were consecutive to it be explained? Is it not more rational to consider that they were all caused by the parasite?”

Another medical student, Chachereau, who also reported his own case, suffered at the age of twenty-three from pleurisy, which was the first symptom to appear. At the end of 1872 he suffered from left pleurisy, with much effusion. Dr. Leonardi recognized the gravity of his condition, and thought of puncture. The pleurisy showed a most insidious course. The effusion was absorbed rapidly, and the other symptoms of hydatid made their appearance much later.

From the first, hydatid of the lung may excite general symptoms. Loss of strength, anorexia, and wasting have been noted in a large number of cases.

In short, it is seen that, in its first period, hydatid of the lung shows its presence by symptoms which **simulate pulmonary tuberculosis to such an extent as to be mistaken for it.** A patient comes to us with hæmoptysis, and says that for some time past he has been coughing and wasting. It is quite natural to suppose the onset of tuberculosis. However, he does not bring up sputum, and the râles are not clearly localized to the apex of the lung, while the search for bacilli in the blood coughed up is negative. This is quite true, and yet, in the face of repeated hæmoptyses, cough, anorexia, wasting, and attacks of pleurisy, we cannot eliminate the idea of early tuberculosis. Perusal of the reported cases shows that the mistake has almost always been made. In such cases the sero-diagnosis of tuberculosis must not be neglected, for a negative sero-diagnosis is of great value. Radiography may sometimes be of use.

**Evolution of the Cyst.**—We have studied the onset of hydatid of the lung; let us now follow the other phases of its growth. If the growing cyst reaches the size of the foetal or adult head, and spreads towards the walls

of the thorax, they may become **arched**. According to the localization of the cyst, the arching occupies the lower, lateral, or upper part of the chest. It simulates in different cases intrathoracic tumour or encysted pleurisy. Sometimes the cyst, if it be very large, may simulate general pleurisy. Examples of these different varieties are as follows :

Moutard-Martin says : " In a patient who had already had several attacks of hæmoptysis, arching of the lateral and inferior part of the thorax appeared on the left side. This arching rose as high as the seventh intercostal space. Over this area dullness was complete, tactile vibrations were abolished, and the normal vesicular murmur was replaced by tubular breathing. With all reserve, it was thought to be a case of encysted pleurisy. Thoracentesis was performed, and it was found that a hydatid cyst of the lung had been punctured."

" A patient," says Danlos, " presented arching of the lower lateral and posterior part of the thorax on the right side. This arching reached as high as the fourth intercostal space. At this level the intercostal spaces bulged outwards. Percussion gave complete dullness, and auscultation showed abolition of the vesicular murmur. The patient had a hydatid cyst of the lung."

In a case which was reported by Debove all the signs of encysted pleural effusion on the left side were found—namely, abolition of the thoracic vibrations, dullness on percussion, absence of vesicular murmur, and displacement of the heart. The case was one of hydatid of the lung.

Landouzy writes : " The patient, who had had violent hæmoptysis, showed dilatation of the chest on the left side, with bulging below the clavicle. Dullness on percussion, abolition of thoracic vibrations, tubular breathing, and marked deviation of the heart were found. Puncture confirmed the diagnosis, and 6 pints of liquid were drawn off from a hydatid cyst of the lung."

Large cysts may, then, produce bulging of the thorax, displace the heart, and simulate encysted or extensive effusion. In some cases they produce other symptoms, of which the most important are pain, dyspnœa, myosis on the same side as the cyst (Widal), and pressure signs, such as œdema of the lower and upper limbs.

**Diagnosis.**—In the early stage hydatid cyst of the lung, especially if it be deeply situated, escapes our methods of investigation. It produces no dullness, no deformity, and no bulging of the thorax. Some râles, due to the pulmonary congestion around the cyst, may be heard, but they do not help us in diagnosis. Radiography may furnish some information. Hæmoptysis must be taken into careful consideration. If, in an individual suffering from repeated hæmoptysis, there is no reason to suspect tuberculosis, aspergillosis, calcuosis, or bronchiectasis, hydatid cyst of the lung must be especially thought of. The diagnosis will be almost certain if the hæmoptysis is accompanied by **urticaria**, as in Chachereau's case.

The **diagnosis** of hydatid cysts, when they produce deformity or bulging of the chest, is also very difficult. To facilitate the discussion, let us divide them into two categories. In some cases the bulging is so clearly limited that the cyst **forms a tumour**. If the bulging occurs at the tense and antero-lateral part of the chest, hydatid cyst of the liver is thought of ; if it deforms



the upper region of the thorax, aneurysm or tumour of the mediastinum will first enter our minds ; if it is posterior and lateral, the idea of an inter-lobar pleurisy presents itself ; if the thoracic deformity occupies the postero-inferior part of the chest, basal effusion is diagnosed. In other cases the cyst does not form a tumour, but by its large extent simulates pleurisy of the great pleural cavity, and presents most of its signs and symptoms, such as dilatation of the thorax, absolute dullness, abolition of the vibrations, tubular breathing, and deviation of the heart—in fact, everything tends to produce mistakes. Aspiratory puncture would remove all doubts. This is true, but we shall see later the mischief which may result from puncture. In making a decision, we must inquire carefully into the symptoms which have marked the onset of the disease and have accompanied its progress. Hæmoptysis occupies the chief place.

As the hydatid antibodies cause deviation of the complement, we may employ this method (Weinberg's reaction) in the diagnosis of hydatid cysts of the lung.

**Rupture of the Cyst.**—All hydatid cysts of the lung do not grow towards the walls of the thorax, neither do they acquire a large size. Many cases are not large enough to betray themselves by any bulging of the chest ; but, whatever be the size of the cyst, be it large or small, complications will certainly appear. They comprise inflammation of the neighbouring regions, infection and suppuration in the cyst, and its rupture into the bronchi or into the pleura.

In many cases rupture of the cyst is preceded or accompanied by broncho-pulmonary lesions, which we must recognize. Pulmonary congestion, broncho-pneumonia, and pleuro-pneumonia have been observed either during the growth of the cyst or at the moment of its infection (Walske, Lorieux). This side of the question has been neglected by some writers on hydatid of the lung. I shall sum it up by describing a case under my care at the Necker Hospital :

One of my attendants, who had had hæmoptysis several months before, was taken ill with fever, cough, pain in the chest, and blood-stained sputum. On examining the patient, we found subcrepitant mucous râles and faint tubular breathing over the middle third of the chest behind on the right side. This condition simulated a bastard pneumonia, or pulmonary infarct. For several days these signs did not change, with the exception of some friction sounds which blended with the diffuse râles.

The expectoration continued to be abundant, viscid, and hæmoptoic ; but then it changed in character, becoming muco-purulent, and one day the patient coughed up several shreds of hydatid membranes, which cleared up the diagnosis. The fever fell. The hydatid cyst had suppurated, and the neighbouring lung tissue had become affected by a bastard pneumonia.

These **pneumonic attacks** (lesions of the pneumococcus) may appear at **different periods in the evolution of the cyst.** They are as important as the attacks of pleurisy mentioned at the beginning of this article.

As long as the cyst does not suppurate rupture does not occur. Exceptions to this rule are extremely rare (Marconnet's case). The cyst may open into the bronchi (*vomica*), as most often happens, into the pleura, or into the pleura and the bronchi at the same time (*pyopneumothorax*). Let us study these different modes of rupture.

Rupture **into the bronchi** is sometimes preceded by a febrile stage, with bronchitis, pulmonary congestion, incessant cough, and purulent, blood-stained, or foetid expectoration. At this time the cyst is **fissured**, but not yet freely open. If the cyst is small, or if the communication with the bronchus is of small size, the opening of the cyst does not quite take the characters of a *vomica*. The patient, after fits of coughing, brings up homogeneous, purulent, sputum, like currant jelly. Sometimes the expectoration is genuinely blood-stained, and hooklets, shreds, or hydatid vesicles, that prove the case, are found in the blood or in the expectoration, which is sometimes foetid.

If the cyst is large, and the communication with the bronchus is well established, the patient is seized with terrible fits of coughing, a feeling of tearing, and suffocation bordering on asphyxia, which is caused by the liquid and the membranes that block the air-tubes. He brings up, as if by *vomica*, a large quantity of fluid, which is clear and transparent, like spring-water if the cyst is not suppurating (Marconnet's case), but muddy, sero-purulent, blood-stained, and of nauseous odour and taste, if the ruptured cyst has suppurated. In the fluid of the *vomica* hydatid vesicles are sometimes found, from the size of a pin's head to that of a walnut, but more often membranous shreds of various sizes are seen.

The first *vomica* is generally the largest, but it is rarely the only one. We sometimes meet with a series of *vomicæ* which are repeated for days, weeks, and months. In the space of a year Chachereau had fifteen *vomicæ*, or at least hydatids were expelled fifteen times. As a rule, I repeat, the first *vomica* is the most severe, and the others are rather a purulent expectoration with shreds of membrane. The breath and the expectoration are often foetid. So long as the hydatids are not all brought up, the purulent expectoration does not cease.

In many cases rupture of the cyst is announced or accompanied by a more obstinate and abundant **hæmoptysis** than the early attacks seen in the first period of its growth. Chachereau had fifteen in eighteen months. They were trifling before the rupture of the cyst, abundant at the moment of rupture, and persisted for five months after the last *vomica*. Marconnet had slight hæmoptysis before rupture of the cyst, but almost fatal hæmorrhage at the moment of rupture. In Habershon's case hæmoptysis caused the death of the patient, and at the autopsy the pulmonary vein was found cut through, causing the hæmorrhage.



**Urticaria** fairly often accompanies rupture of the pulmonary hydatid, like that of hydatids in every region. Arnault has cited a case of general urticaria after rupture of the cyst into the bronchi. Chachereau had his first attack of urticaria some days before the cyst opened into the bronchi, and had ten attacks in succession, which were sometimes so severe that sleep became impossible. Delagenière quotes the case of a woman who was seized with urticaria after the cyst perforated the pleura. Urticaria is, therefore, a symptom which fairly often accompanies rupture of hydatid cyst of the lung.

New signs appear after rupture of the cyst into the bronchi. On auscultation, multiple râles and, at times, cavernous breathing are heard. As secondary infection often occurs in the cyst, fever is not rare, and with it the usual train of symptoms—viz., sweating, anorexia, wasting—appears. When we see these wasted, anæmic patients, who spit up pus and blood and have clubbed fingers (Trousseau), we would not be able to eliminate the idea of phthisis if bacteriological examination did not rectify the diagnosis.

The **diagnosis** of the hydatid vomica is quite simple when membranes and shreds are found in the voided matter. In default of hydatid membranes that present a characteristic shape, it is necessary to search carefully for hooklets, because the presence of a single hooklet makes a doubtful diagnosis certain. In many cases the vomica does not appear with characteristic symptoms, and we then lack a clue to the diagnosis. Most patients think they are suffering from bronchitis, and say that for a week, a fortnight, or longer, they have been coughing up muco-purulent or blood-stained sputum. They do not mention the shreds of hydatid, which may have passed unnoticed. These patients are examined, and in one we find signs of bronchiectasis; in a second, those of chronic bronchitis, with bronchorrhœa; in a third, signs of pulmonary cavity, with repeated hæmoptysis; in a fourth, signs recalling the vomica which follows encysted interlobar pleurisy.

So far it is sometimes difficult to make a diagnosis. Urticaria, when it exists, is a point of great value; but the histological examination of the expectorated matter must never be neglected, for the presence of hydatid membrane and hooklets can alone give positive information.

Rupture of the cyst into the bronchi is sometimes a mode of cure, but often the pulmonary fistula, which is a centre of infection, becomes an inexhaustible source of purulent expectoration and hæmoptysis. The patient continues to cough up membranes and much purulent foetid sputum. Fever supervenes, with its train of septic symptoms, including diarrhœa, loss of appetite, wasting, sweats. We see next true hydatid phthisis produced by secondary infections. **Tuberculosis** may also develop as a secondary infection in the course of hydatid of the lung.

**Rupture into the Pleura.**—Let us consider rupture of the cyst into the pleura.

I must first, however, discuss an interesting question : Can the hydatid cyst develop primarily in the pleural cavity ? Primary hydatid cysts of the pleura are extremely rare. Laënnec, Cruveilhier, Davaine, and Trousseau scarcely admit the possibility of the **primary** development of a hydatid cyst in the pleural cavity. Davaine, in twenty-five cases of thoracic hydatid, only once found a primary hydatid of the pleura. Dupuytren and Joffroy reported a case of pleural hydatid ; careful consideration, says Trousseau, shows that it was a cyst of the lung which opened into the pleura, because the patient had had hæmoptysis. Vigla, in his work on hydatids of the thoracic cavity, quotes only one case of pleural hydatid, and it is a very doubtful case, as it lacks post-mortem verification. Maydl published in 1891 a memoir upon echinococci of the pleura ; after having analyzed in detail each of the cases contained in this work, I find that there is no case of primary hydatid of the pleura, but only of hydatids secondary to those of the lung or of the liver. Further, frequent as primary hydatid is in the parenchyma of organs, it is exceptional in serous cavities. The hydatid is found in the liver and the subperitoneal tissues, but very seldom in the peritoneum. The hydatid is found in the brain, but very seldom in the meningeal cavity ; in the heart, but very seldom in the pericardium ; in the lung, but very seldom in the pleura. Cysts of the serous and of the pleural cavities have in most cases penetrated these cavities by effraction. A cyst of the lung may work through the thickened pleura, and thus simulate a cyst of that tissue (Trousseau).

From these facts we must conclude that hydatid of the pleura is nearly always secondary to one that starts in a neighbouring organ and perforates the pleural cavity. The thickened pleura often opposes this invasion, and when perforation takes place it may consist of a simple slit or a large tear in the pleura.

In some cases the invasion of the pleura may be insidious, but more often it is accompanied by dyspnoea and pain. We find signs of dry pleurisy (rub) or of pleural effusion. The following case is typical :

A boy, of tubercular appearance, was suddenly seized with stabbing pain in the right lung. On auscultation, friction sounds were heard. The whole right side of the chest became enlarged, and was absolutely motionless during respiration. The dullness was absolute. Post mortem, the right pleura was found to be ruptured, and filled with sero-purulent fluid and membranes from a huge hydatid of the lung, which had burst into the pleural cavity.

In other cases, which are the most frequent, the cyst of the lung shows two perforations, which communicate with the pleura on the one hand and with the bronchi on the other. I have carefully analyzed these cases, and have found that perforation almost always takes place first into the bronchus, while the other perforation occurs a little later. The perforation into the



bronchus is generally a small fissure. It does not produce the large vomica with acute symptoms, and the patient appears to have only foetid bronchitis or broncho-pneumonia with gangrene. He brings up purulent, foetid, blood-stained sputum. Perforation of the pleura now occurs, and pneumothorax appears. If the perforation of the pleura takes place before that of the bronchi, and the lung is adherent to the pleura, the symptoms of pneumothorax may not be violent; in other cases the pneumothorax is accompanied by terrible pain, acute dyspnœa, and threatening asphyxia. The subjoined cases give a clear idea of these different modes of perforation :

In a case reported by Bucquoy, perforation of the pleura preceded that of the bronchi. The patient had a hydatid cyst of the right lung. Very abundant effusion into the right pleura (opening of the pulmonary cyst into the pleura) appeared. Later, signs of pneumothorax (opening of the cystic cavity into the bronchi) supervened, and the patient brought up much foetid purulent fluid by repeated vomicæ. The situation became very serious. Thoracotomy was performed, and the incision gave exit to infectious, purulent fluid, and to a hydatid cyst as large as an orange.

Danlos relates a case in which the perforation of the bronchi preceded that of the pleura. The onset of the pneumothorax was dramatic, and followed by death. In a man aged forty-five years deformity and bulging were present at the lower lateral and posterior part of the right side of the chest. Hydatid cyst of the lung was diagnosed. The patient was seized with fits of coughing, and brought up purulent and foetid sputum (small opening of the cyst into the bronchus). Ten days later he felt terrible pain on the right side, with dyspnœa. On auscultation, amphoric breathing was heard (perforation of the pleura and pneumothorax). The patient died, and the autopsy revealed a huge hydatid cyst at the base of the right lung, causing the above complications.

In a case given by Fouquier the pneumothorax came on suddenly. The patient died, and the autopsy showed a hydatid cyst of the lower right lobe of the lung, communicating on the one hand with two bronchi, and on the other with the pleural cavity by a rounded opening with raised edges, into which the end of the index-finger could easily be introduced.

**Summary.**—The diagnosis of hydatid of the lung is very difficult before perforation of the cyst and vomica. Hæmoptysis, pleurisy, and attacks of pneumonia do not clear up the diagnosis. Bulging of the chest, dullness which is clearly defined, and previous or simultaneous hæmoptysis not due to tuberculosis, are valuable signs in diagnosis. We know that great precautions are needed in exploratory punctures, which are, however, the only means of clinching the diagnosis before the rupture of the cyst.

When a patient has cough, is wasting, and has repeated hæmoptyses and pleural complications, we think first of pulmonary tuberculosis. Examination for bacilli is negative, and the diagnosis remains in doubt. The patient later brings up hydatid vesicles or membranes, and the diagnosis becomes certain.

In some cases, however, even after purulent fluid has been rejected

through the bronchi, diagnosis is very difficult, because the membranous shreds may escape the patient's notice, and the lesion would pass unperceived if we did not find vestiges of membranes or hooklets on microscopic examination. Lastly, in other cases the disease, at a given moment, assumes the appearances of broncho-pneumonia, as in my hospital attendant, and a hydatid of the lung would not be recognized if membranes and hooklets were not found in the expectoration.

The same remarks apply to the passage of the hydatid into the pleural cavity and to the question of pyopneumothorax.

As I have discussed the **diagnosis** in regard to the different complications which may arise in the course of hydatid of the lung, further reference is superfluous.

**Prognosis.**—In this chapter I have reviewed the complications of hydatid of the lung, and we have seen its gravity. In exceptionally fortunate cases, the cyst may be cured spontaneously—that is to say, it may undergo necrobiosis, which is equivalent to cure. In some cases rupture of the cyst into the bronchi results in cure, but before recovery is definite the patient runs the risk of most grave complications—viz., at the moment of rupture hæmoptysis, vomicæ, and pyopneumothorax; and later secondary infections, hectic fever, and tuberculosis.

**Treatment.**—As medical treatment has no effect on hydatid of the lung, surgical intervention must be resorted to.

Certain authors have recommended expectant treatment, but this method has scarcely given good results, as the following statistics prove: Hearn, in 128 cases of pulmonary hydatid, left to run their own course, counts 82 deaths—a death-rate of 64 per cent. Madelung, in 19 untreated cases, records 6 deaths.

Thomas Davies records 31 deaths in 133 cases of pulmonary hydatid which opened into the bronchi. Such figures are not encouraging, and explain the necessity for surgical intervention.

Aspiratory puncture is insufficient and frequently **dangerous** in hydatid cysts of the lung. Mirallié has collected 43 cases in which simple puncture was performed; 11 cures, 22 deaths, and 10 negative results are recorded. Punctures are frequently followed by sudden or by rapid death. Thus, in the 22 fatal cases noted above, we find that they may be divided as follows: In 1 case the patient died after a blank puncture; in 10 cases death was sudden—death came on in one minute (Acland, Philippe, etc.), in five minutes (Holden), in seven minutes (Bristowe), in half an hour (Lansdale and Holden), in two hours (Hector Mackenzie), in nine hours (Cornil and Gibier), in thirteen hours (Duffey). In almost all these cases the accident was accompanied by some attacks of coughing, with cyanosis and asphyxia, coldness of the extremities, and sometimes rejection of blood and fluid.



Puncture, therefore, must be abandoned ; it is dangerous even for purposes of exploration.

**Pneumotomy** is the best method, and is absolutely indicated. It gives such good results that the recoveries amount to 90 per cent.

## XXI. SYPHILIS OF THE LUNG.

**Discussion.**—Syphilis of the lung is of the highest importance. Too many mistakes in diagnosis occur from want of care in looking for this malady. It endangers the patient's life ; but, on the other hand, it gives us an opening to score a therapeutic success, for we can sometimes restore health in a few weeks or in a few months to people who appear to be in the last stage of phthisis.

The chief manifestations in the lung include broncho-pneumonia of tubercular aspect, cavities simulating phthisis, gangrene, fibrosis of the lung, and dilatation of the bronchi, without counting mediastinal adenopathies and pleurisy. Before we undertake the clinical study of these syphilitic lesions, we must consider an important point : At what date may syphilis attack the lung ? Is it soon or long after infection ? This division into early and late lesions is clearly marked in such organs as the brain, the spinal cord, the kidneys, etc. Take, for example, cerebral syphilis. Its lesions are sometimes late and at other times early. The late lesions, such as obliterating or dilating arteritis, and gummatous lesions of the bones, meninges, or brain (general paralysis), appear many years after infection. We also see early cerebral syphilis which is almost exclusively confined to the arteries of the circle of Willis. It may, some months after infection, give rise to endarteritis obliterans, with cerebral softening and hemiplegia, and to arteritis ectasians, with rupture of an aneurysm, meningeal hæmorrhage and death from apoplexy.

This distinction between early and late lesions is nowhere more marked than in the kidneys. The tertiary disease usually takes the form of chronic nephritis, with or without gummatous, sclero-gummatous, and amyloid lesions, while early renal syphilis appears some months after the chancre, and causes acute or hyperacute nephritis, which is sometimes most dangerous.

It was formerly thought that syphilis only attacked the organs in the so-called tertiary stage. This view is incorrect. Some organs are affected by syphilis at a very early stage, and the lesions, though early, are far from being benign. They are quite as serious as the lesions of the tertiary stage.

I will, therefore, put the question afresh : Do we find in the lung early syphilis, that appears within the first few months after infection, and late

syphilis, that appears only after a long interval? The answer is categorical. Syphilitic lesions of the lung are, practically speaking, never early. I would ask you to remember that I am speaking of the lung, and not of the bronchi. Bronchitis may be one of the earliest manifestations of syphilis. Many people, when once infected, take cold with the greatest ease. They suffer from laryngitis or tracheo-bronchitis; they cough and expectorate. On auscultation, sibilant and mucous râles are heard, and the trouble is put down to influenza or to an ordinary cold, and not to recent syphilis. Tracheo-bronchitis may recur several times, even during the early years of infection, and sometimes arouses unjustifiable suspicions of tuberculosis. These so-called attacks of bronchitis lead us to send patients to take a cure at Mont-Doré, Cauterets, Eaux-Bonnes, and Luchon, when we should have begun by prescribing mercury. Laryngo-tracheo-bronchitis is, then, one of the early manifestations of syphilis. Lesions in the lungs, on the other hand, occur late, and not one of the other visceral determinations, as Mauriac rightly remarks, develops at a later period. This rule appears to me absolute, and, save for some exceptional cases, it may be said that syphilis of the lung only appears in the advanced tertiary stage.

After these explanations, let us enter upon our subject. Syphilis of the lung, as of other organs, presents various forms—gumma (circumscribed syphiloma), diffuse syphiloma, and fibrosis. These different forms may be isolated or associated. For the clear arrangement of this question I propose the classification which I have followed in my clinical lectures at the Hôtel-Dieu.\*

Syphilis of the lung may show the following forms :

**First Type.**—Pulmonary syphiloma, with acute febrile course, simulating acute tuberculosis or tubercular broncho-pneumonia.

**Second Type.**—Pulmonary syphiloma of slow course, simulating ordinary chronic tuberculosis and phthisis in the stage of cavity.

**Third Type.**—Broncho-pulmonary syphiloma, with fibrosis or sclero-gummatous lesions, simulating chronic pneumonia and cirrhosis of the lung, with or without bronchial dilatation, pleurisy, and tracheo-bronchial adenopathy.

**Fourth Type.**—Syphilitic gangrene of the lung.

**Fifth Type.**—Syphilitic pneumopathy, complicated by pulmonary tuberculosis.

**Sixth Type.**—Hereditary pulmonary syphilis.

\* Dieulafoy, "Syphilis du Poumon et de la Plèvre" (*Clinique Médicale de l'Hôtel-Dieu*, 1898, leçons 18 et 19).



### 1. Pulmonary Syphilis, with Acute Course, simulating Acute Tuberculosis.

In some cases—which are rare, it is true—syphilis of the lung shows an almost faithful picture of acute tubercular broncho-pneumonia—*i.e.*, fairly sudden onset, acute fever, incessant cough, violent dyspnoea, muco-purulent expectoration, rapid wasting and profuse sweats, dullness on percussion, râles and tubular breathing on auscultation. The above symptoms may be common to syphilitic and to acute or subacute tubercular broncho-pneumonia. The following example was seen by Giraudeau in Hayem's wards :

Patient, thirty-five years old. She had cough, and for a week fever was continuous. Temperature up to 104° F. On admission, dull zone, with tubular breathing and subcrepitant râles over the middle part of the left lung. Nothing elsewhere. Right lung healthy. Frequent cough and profuse expectoration. During the next few days, rapid formation of a cavity. The breathing soon became tubular, and the expectoration muco-purulent. Later, in addition to tubular breathing, large mucous râles appeared, wasting was considerable, and night-sweats occurred. On February 21—that is to say, in twenty days—a cavity had formed in the lung. Cavernal breathing, pectoriloquy, and bronchophony were audible over the greater part of the middle lobe. Gurgling was present; the sputum was nummular, and streaked with blood. The patient appeared to be suffering from **acute phthisis**.

On February 28 vaginal examination revealed in the right cul-de-sac a rounded ulcer, which was as big as a sixpence, punched out, and covered by a greyish, adherent layer. This **gummatous** ulcer showed the need for specific treatment. The lung trouble was then thought to be specific in nature, and Gibert's syrup given in doses of two tablespoonfuls daily. After some weeks, the situation improved, the appetite came back, the nummular sputum, sweats, and fever disappeared. The cavernal breathing and gurgling were replaced by rough breathing. **The cavity had cicatrized**. As the treatment had caused stomatitis, it was stopped. Ninety grains of iodide of potash were prescribed daily, and when the patient left the hospital she had regained her healthy look, and the gummatous ulcer in the vagina was undergoing cicatrization. A little while after, this woman came back for treatment, not for the lung, which was healthy, but for osteoperiostitis of the frontal bone, which yielded to specific treatment. It was, therefore, undeniable that this woman had been attacked in a short time with vaginal gumma, syphilis of the lung, and frontal osteoperiostitis. Judging by the rapid and exceptionally serious course of the lesion in the lung, she was doomed to die. She was saved by specific treatment.

I have seen a similar case.

I attended a patient supposed to be suffering from influenza. He coughed, had slight fever, and complained of pain in the chest, especially at the apex of the left lung, where I found tubular breathing and râles, due to pulmonary congestion. This man, who was up to then in robust health, asked for a blister, which I prescribed, as well as a draught containing kermes. During the next few days the situation became worse. The cough was incessant, and at night the dyspnoea was unusually severe. The sputum soon became muco-purulent and nummular. The early râles changed to gurgling. His strength failed, and I thought of acute tubercular pneumonia.

The prognosis was very grave. Incidentally, the patient told me that his left testis had been affected for some days. I found it enlarged and painful. Orchitis was

present, but not epididymitis. As he had no discharge, I thought at once of tertiary syphilis. I questioned the patient, who replied that he had had a chancre ten months before. This revelation threw light on the subject. Was it not possible that syphilis was at once the cause of the lesion in the lung and in the testis? I prescribed iodide of potash in large doses, and also asked the advice of Fournier. He considered the orchitis and the lung trouble to be syphilitic, and advised inunction of mercury in addition to the iodide of potash. The picture changed so rapidly that after a few nights the dyspnoea disappeared. A fortnight later the fever had subsided, the general condition was excellent, tubular breathing and gurgling had disappeared, and the only local sign remaining at the apex of the right lung was slight dullness with some râles, which finally disappeared. The lesion of the testis was also cured. The case was undoubtedly acute phagedænic syphiloma, which rapidly caused breaking-down of the apex, and was clearly arrested in its course by specific treatment. I have never lost sight of this man, and I have treated him since that time for a syphilitic whitlow, but there has never been any further question of pulmonary lesions.

In my clinical lectures, several cases of this acute and subacute broncho-pneumonic syphiloma with fever will be found. In nearly all these cases it has simulated acute tubercular broncho-pneumonia: onset fairly sudden, acute fever and dyspnoea, muco-purulent expectoration, rapid wasting, profuse sweats, with dullness, râles, tubular breathing, and gurgling, are the signs common to acute broncho-pneumonic syphiloma and to acute or to subacute tubercular broncho-pneumonia.

It has been said that syphilitic pneumopathy may occur without fever, wasting, or any sign of hectic. This is true in a certain number of cases of slow course, which we shall study later, but not of the acute forms, in which the diagnosis is extremely difficult. If we carefully think over the acute cases, we shall agree that patients who suffer from fever, accompanied by signs of pulmonary softening, purulent sputum, and night-sweats, and pass into rapid consumption within a few weeks, clinically resemble those who are suffering from acute phthisis.

In them, it is true, dyspnoea is often severe and out of proportion to the lesion, but this symptom is not sufficient to decide the diagnosis. The physical signs—râles, tubular breathing, gurgling—are those of acute phthisis. It has been said that the localization of the lesions may reveal the true nature, because the syphilitic lesion is often situated in the middle part of the lung, and especially on the right side. This special topography is, indeed, worth remembering: it may lead us to think of syphilis; still it is not constant, for in a case of Raymond, and also in one of my cases, the lesion was at the apex of the lung, which is the usual seat of tuberculosis.

Acute syphiloma and acute tubercular broncho-pneumonia have, therefore, very many points of resemblance, and hence it is only possible to make a diagnosis of pulmonary syphilis if the patient present other syphilitic lesions, such as osteitis or painful periostitis, ulcerating gumma, specific eruption, or syphilitic testis. If we find some such stigmata of syphilis as



an exostosis of the tibia, frontal periostitis, the presence of scars, and the repeated absence of Koch's bacillus from the sputum, we can give a diagnosis of acute syphilitic pneumopathy. In short, if pathognomonic symptoms of acute pneumonic syphiloma do not exist, it is at least necessary to collect all the information which may give a clue to the diagnosis. In one of my patients at the Hôtel-Dieu pneumonic syphiloma was diagnosed, thanks to tertiary syphilis of the shoulder and the back. In the patient whom I saw with Fournier syphilis of the testis revealed the nature of the pulmonary lesion. In Giraudeau's patient an ulcerated gumma of the vagina led him to discern acute syphilis of the lung.

What is the cause of acute syphiloma of the lung? The syphiloma may be circumscribed (syphilitic gumma) or diffuse (broncho-pneumonic syphiloma). Gummata of the lung may be as large as a lentil or an egg. They are more or less numerous, and may be situated on the surface of the lung, under the pleura, or in the deep parts of the organ. They are indurated and greyish in an early stage of their growth, but finally soften and become converted into a yellowish pulp, which may pass into a bronchus, and leave in its place a gummatous cavity, with fibrous walls. When cure sets in, the walls of the cavity become covered with granulations, the cavity contracts, and is replaced by scar tissue.

**Diffuse syphiloma** of the lung assumes a special form, and causes a kind of syphilitic broncho-pneumonia, which may be acute, subacute, or chronic. The acute form, which has been discussed in the examples quoted above, is somewhat rare. Microscopic examination has given the following results:

"Microscopic examination," says Rémy, "shows that the tumour is composed of a number of nodules of broncho-pneumonia in various stages (catarrhal, fibrinous, and caseous). There is no well-marked general capsule formed by a zone of fibrous tissue, as in the case of gummata. At some points, however, this capsule is seen, but it is formed by the septum of the lobule, and not by newly-formed tissue. No central artery is found. The lesions are rather grouped around the bronchi, as in broncho-pneumonia. The lesion as a whole consists in several masses of caseous appearance, which are surrounded by more active zones. The caseous mass is composed of pulmonary alveoli, filled with cells, which have lost their shape and appear to be undergoing fatty degeneration. The envelope of the caseous mass is sometimes formed by a thickened interlobular septum, at other times by alveoli which are filled with leucocytes, and present thickened walls. Furthermore, we find in the thickness of the bronchi, vessels or interlobular septa, masses of young cells which reveal an inflammatory condition."

Syphilitic broncho-pneumonia has been studied in the foetus and in the new-born by Balzar and Grandhomme. They have found lesions like

those of ordinary broncho-pneumonia. The vascular and interstitial lesions, however, appeared to be most marked, but epithelial desquamation and slight exudation were also noted. Whether diffuse pulmonary syphilis begins as a broncho-pneumonia of bastard form, with parenchymatous infiltration involving the bronchus and the alveolus, or whether the process chiefly affects the arterioles, it is none the less true that the diffuse syphiloma may run a rapid or a slow course. In the former case the lesion results in dissolution of the invaded tissues. Tertiary syphilis, then, causes a kind of **phagedænicism** of the lung, similar to that of the velum palati or of the pharynx. These cases simulate acute tubercular broncho-pneumonia.

## 2. Pulmonary Syphiloma, simulating Ordinary Chronic Tuberculosis.

We must now consider the slow form of pulmonary syphilis which simulates chronic tuberculosis, with or without cavities. The following examples are typical :

In November, 1878, Fournier had a woman under his care at the Lourcine Hospital for an enormous phagedænic ulcer, which involved a large part of the foot. He ordered inunctions of mercury and iodide of potash. The patient was cachectic, and looked phthisical, so that she was thought at first to have **pulmonary tuberculosis**.

"This presumption, deduced from the external appearance," says Fournier, "found support in that the patient complained that she had coughed for several months, brought up abundant green sputum, and suffered from fever, profuse night-sweats, etc. Physical examination of the chest seemed to confirm this view, and revealed somewhat extensive dullness in front of and behind the apex of the left lung. The rest of the lung appeared free. At the same spot rough, cavernous breathing, cavernous râles, and splashing sounds were heard. In short, the general condition, the local symptoms, and the physical signs corresponded to those of pulmonary phthisis."

Although the idea of pulmonary syphilis entered Fournier's mind, this eminent authority felt obliged to hold to the most probable diagnosis—that of **pulmonary phthisis** (Koch's bacillus was not yet discovered). The later course did not agree with Fournier's opinion. The patient, "whose days appeared numbered," suddenly began to improve under specific treatment, and her strength returned, so that when she left the hospital, after a stay of four months, she was fat and well. The local lesions and the functional troubles improved at the same time as the general condition. The dyspnoea and the pain in the side disappeared, while the auscultatory sounds were reduced to slight crackling or to disseminated subcrepitant râles. When Fournier saw the patient again several months later, careful auscultation failed to show the least traces of the lesion.

Panas has published a similar case :

"A woman, thirty-two years of age, consulted me," says Panas, "for a lesion of the left eye, and for disease of the lung. She told me that the former began as a little whitish point at the inner edge of the cornea. This tumour, which was a little larger than a pin's head, grew rapidly. The eye became red, and in a fortnight vision was lost. We found the eye much enlarged. The cornea was hypervascular, opaque, and scarcely distinguishable from the conjunctiva. A yellowish swelling, like a tubercle, appeared near the inner border. The ciliary body projected like a blackish mushroom



through a perforation in the sclerotic. Perception of light was abolished. On examination of the chest, the left lung was healthy, but the right one was dull in its upper third, and auscultation revealed both behind and in front large mucous râles and gurgling, indicative of tuberculosis nearing the end of its second stage, and going on to cavity. Add to this slight hæmoptysis, continual cough, with scanty expectoration and a loss of weight of 40 pounds. In spite of this wasting, the patient appeared healthy, and had a good appetite. There was no reason to eliminate phthisis, but I was put on my guard by the existence of a *corona Veneris* upon the forehead. She had had syphilis seven years before. The chancre had passed unnoticed, but two miscarriages, two children dying in infancy, and then a general eruption, which had left enormous scars all over the body, together with the syphilide on the forehead, made the diagnosis quite clear. We therefore hoped that the ocular and pulmonary lesions were both syphilitic.

"I gave every second day an injection of peptonate of mercury (representing nearly  $\frac{1}{8}$  grain of the active principle). After the second injection the improvement was remarkable, and the progress was evident with each fresh injection. In the eye, which was a red staphylomatous shell, the sclerotic, the cornea, and the iris gradually became distinct, and the eye regained its proper shape. At the same time, the lung rapidly improved. Dullness and râles diminished, and then disappeared."

In my wards at the Hôtel-Dieu we have had quite as conclusive cases.

Some years ago I saw a case of this kind. I was called to see a young man condemned as phthisical. We had to decide if he were fit to leave for the South, in order to reside there during his few remaining months. I saw him, and as soon as I entered his room he recognized me, while I remembered that he had consulted me the year before for a large ulcerative syphilide of the arm, elbow, and forearm. He had applied ointments and blisters without the least success. Specific treatment (inunction of mercury and iodide of potash) rapidly cured this tertiary ulceration, which was of nearly a year's duration. I now found that he looked like a case of advanced phthisis. He told me that he had had a "neglected cold" eight months before. He had begun to cough and to spit, and had often brought up a little blood. He next lost his appetite, night-sweats appeared, the expectoration became muco-purulent, and the customary drugs for phthisis had effected no result.

On auscultation, I heard cavernous breathing and gurgling at the lower angle of the right scapula. This site, which is an unusual one for a tubercular cavity, made me reflect; and, warned by the tertiary ulceration which I had cured the preceding year, I suspected syphilis of the lung, and ordered inunction of mercury and iodide of potash, the dose of which was raised to 160 grains daily. In a few weeks the change was complete. The patient recovered, and the physical signs disappeared. The cavity in the lung filled up, the expectoration diminished, and the daily progress was evident.

The preceding cases give an idea of pulmonary syphiloma of slow course **simulating ordinary chronic tuberculosis**. This form has an insidious onset, and fever is practically absent. At first it simulates bronchitis, pulmonary congestion, or ill-defined broncho-pneumonia. The appetite remains good, the patient does not waste, the cough is frequent, and the expectoration has no special characters. Later, we find other symptoms, such as pain, dyspnoea, which may be severe and is worse at night, purulent expectoration, night-sweats, loss of appetite, and wasting. Later still, hectic fever, gurgling, and bronchial or cavernous breathing appear, according to the degree of the lesion (induration, softening, cavity). Gummatous ex-

pectoration and syphilitic vomica have been spoken of, but I think they are exceptional. Hæmoptysis is rare and small in amount. The patient only brings up some blood-stained sputum in most cases, yet in a case of Lancereaux much blood was expectorated, and one of my patients brought up two tumblerfuls of blood.

Chronic syphiloma, like the acute form, often occupies the middle part of the right lung outside the hilum. The sounds on auscultation are most marked either at the spine of the scapula or in front in the third and fourth intercostal spaces. To this rule there are numerous exceptions (Fournier and Gübler's cases).

Though the patient with pulmonary syphilis does not become phthisical so soon as with tuberculosis, and though his health may remain good for some time, while he coughs up elastic fibres or fragments of a gumma, Bazin's idea that the syphilitic patient is always a *caverneux bien portant* must not, however, be exaggerated.

Whatever be the mode of onset of this syphilitic pneumopathy, even when the patient has for some time remained in good health, it is rare for cure to supervene without treatment. Sooner or later functional troubles appear, the sputum becomes nummular, fever commences, night-sweats are profuse, wasting makes progress, the nails are curved, and, in default of treatment, the patient dies in consumption.

What are the causes of this syphilitic phthisis? We can readily understand how gummata in the lung soften and leave cavities. We know, too, that a syphilitic patient may become cachectic from the simultaneous development of specific lesions or of amyloid degeneration in other tissues (liver, kidney, spleen, larynx); but how can pulmonary syphilis cause a patient to become phthisical? There is nothing in the course of tertiary syphilis in other organs to determine hectic fever and consumption, with night-sweats and clubbed fingers. Microbiological discoveries seem to simplify the problem, and I think that in the patient with pulmonary syphilis hectic fever is not the direct result of syphilis, but of secondary infections, whose origin is readily grasped. In the lung, syphilis causes ulcers and cavities in which micro-organisms swarm. Some of these microbes find in the cavities a soil favourable to infections and toxic products.

The **diagnosis** of chronic pulmonary syphiloma is surrounded by the greatest difficulties, because no single sign, by its presence or by its absence, can make it certain that the mischief is syphilitic. The presence of Koch's bacillus in the sputum solves the difficulty, but tubercle and syphiloma may occur together in the same lung, and in this case the functional troubles, the physical signs, and the bacteriological examination all conspire to obscure the diagnosis.



Unless a happy chance should point to the development of a gumma, an osteoperiostitis, or a tertiary eruption, in whatever region it may be, or unless characteristic scars were present, the nature of the pneumopathy would often pass unnoticed. In one of my patients in the Saint-Christophe Ward tertiary glossitis put us on the track of the diagnosis ; in one of Gbler's patients the nature of the pulmonary lesion was suspected, thanks to an exostosis of the tibia. In the so-called phthisical patient to whom I was called the tertiary ulceration of the arm revealed the diagnosis ; the **corona Veneris** caused Panas to think that his patient's cavity was of syphilitic origin. As a general rule, in spite of the marked frequency of tuberculosis over pulmonary syphilis, the personal and family history should always be scrutinized, and if some stigmata of syphilis be found, specific treatment must be instituted without delay.

The pulmonary syphilis that I have just described is due to the circumscribed gumma and to the diffuse syphiloma. In acute cases events hasten on, and the patient appears to be suffering from acute tubercular lesions. In cases of slow course the lesions gradually lead to caseation, ulceration of the lung and cavity. They simulate ordinary chronic tuberculosis. To sum up, whether it be a question of circumscribed gummatous syphiloma (gummata) or of diffuse gummatous infiltration (false broncho-pneumonia), we see a process which has a tendency to ulceration, while its course may be **rapid or slow with all intermediate forms.**

### 3. Pulmonary Syphiloma, with Fibrosis or Sclero-Gummatous Lesions.

In this variety the lesion consists in hyperplasia of the connective tissue which forms the stroma of the lung. Fibro-plastic tissue is thus disseminated through the whole parenchyma. This **syphilitic cirrhosis**, which clinically assumes the form of chronic broncho-pneumonia in most cases, presents the following anatomical changes : We find in a lobe of the lung a reddish-grey, hard block, which creaks under the knife, contains ampullary or sacciform dilatations of the bronchi, and is covered by very thickened pleura, while intercostal periostitis may be also present. The surface of the lung may be furrowed by ridges, that radiate like the cicatrices seen in a syphilitic liver. We sometimes find in the deep tissues large bands of fibrous tissue having the white pearly appearance of tendons. The lesion may vary much in kind. It may be purely fibrous, without caseous nodules or evident gummata ; it may be sclero-gummatous, in which case we find in the bronchi or in the peribronchial or perilobular fibrous tissue gummata which are sometimes so small that it would be impossible to say at first sight whether they are tubercular granulations or microscopic gummata. The lesion may resemble the syphilitic pneumopathy of the new-born, which we shall describe under the name of **white pneumonia.**

The histogenesis of this process presents some interesting points. The lesion begins most often around the medium-sized bronchi at the hilum, from whence it appears to radiate into the lung tissue. The new tissue thus forms a fibrous or sometimes chondroid muff around the bronchi or around the arterioles which accompany them. It finally surrounds the alveolus and the pulmonary lobule, so that in distribution it is a form of broncho-pneumonia. The connective proliferation causes deformity of the bronchioles, which may be **dilated** or stenosed. The alveoli are pressed together, and their epithelium is often in a state of fatty degeneration.

In some cases the broncho-pulmonary lesions are accompanied by chronic **pleurisy**, with thickening of the **pleura** and **effusion**. The effusion may indeed be the chief lesion, as we shall see under Syphilitic Pleurisy.

Syphilitic cirrhosis of the lung is difficult to diagnose. Chronic broncho-pneumonia is easily diagnosed, according to certain signs presented by the patient during the past few months or years; later, if abundant foetid sputum appears, with gurgling and cavernous breathing, dilatation of the bronchi is readily recognized. The diagnosis is facilitated, furthermore, by the pulmonary antecedents and the absence of Koch's bacillus in the sputum. A physician, suspicious of the signs furnished by auscultation, will establish an exact anatomical diagnosis: chronic broncho-pneumonia, fibroid state of the lung, with or without dilatation of the bronchi. What escapes him, however, is the cause of this condition. He finds neither measles nor previous whooping-cough to explain it, and unless he discovers a specific lesion of the larynx, the skin, the tibia, etc., elsewhere, or the presence of syphilitic scars, the true nature of the lung trouble will pass unnoticed. This cirrhotic broncho-pneumonia, which usually runs a slow course, may, in exceptional cases, be acute. It is susceptible to improvement by anti-syphilitic treatment.

The sclerous syphiloma is often accompanied by **dilatation** of the **bronchi**, both in the hereditary and the acquired disease (Balzer, Grandhomme, and Grawitz). Sometimes bronchiectasis is the chief lesion. Several cases have been collected by Bourdieu. For example:

A man, fifty-eight years of age, who contracted syphilis at the age of twenty-five, came under Lancereaux for lung trouble, characterized by incessant cough and abundant expectoration. He had coughed for ten years, and brought up 8 to 10 ounces of purulent sputum in the twenty-four hours. Examination of the chest revealed at the bases rather than at the apices gurgling, which, with the other signs, led to the diagnosis of bronchial dilatation. As albuminuria, œdema of the lower limbs, and dyspnoea were present, the existence of nephritis was allowed. The patient died, and the autopsy gave the following results: The bronchi showed cylindrical dilatation; the walls were transformed into a greyish, resistant tissue, which encroached on the neighbouring parenchyma. The newly-formed tissue, which replaced the wall of the bronchus, was infiltrated by miliary gummata. Several of these were calcified, and looked like small stones, in size from a pin's head to a pea. The interbronchial tissue was resistant



and traversed by bands of fibrous tissue. The gummatous lesion in the bronchi was more marked than the fibrosis in the lung, for none of those fibrous masses which resist the knife and correspond to the description of syphilitic interstitial pneumonia were found. The liver was syphilitic and of the "tight-laced" form. The kidneys showed amyloid degeneration of syphilitic origin. Syphilis had destroyed the resistance of the bronchial walls, causing bronchiectasis.

Local or generalized **bronchiectasis** is certainly the most rare of all the anatomical forms of pulmonary syphilosis. It is always more or less associated with broncho-pneumonia, fibrosis, and pleurisy. The symptoms and the course of syphilitic bronchiectasis differ in no way from those which accompany bronchiectasis in general.

**Syphilitic adenopathies of the mediastinum** are sometimes associated with syphiloma of the lung. They exist both in the case of hereditary and of acquired syphilis. They are not frequent, but may acquire supreme importance, because they exert pressure upon the neighbouring organs, as in the following case :

A. G. was brought by her mother to the Saint-André Hospital. This child, aged ten and a half years, had suffered for three months from palpitation of the heart, giddiness with breathlessness, and violent headache at night. Painful fits of coughing had been present for nearly three months.

We learnt that the mother had aborted twice—at seven months and at eight and a half months. Before the birth of A. she had had sore throat, lost her hair, and noticed spots on her body and an eruption on the palm of her hands. The husband had similar troubles six weeks before. Examination of the child: Notched teeth, ulcerated on their anterior surface. On both sides of the neck was a chain of glands, hard, and not matted together. Chest thin and boat-shaped. Supra- and subclavicular hollows well marked. The jugular veins were dilated, and on the front of the neck was a large network of veins, which collapsed during rest, but swelled under the influence of any effort. On percussion, a band of dullness, most marked over the right sterno-clavicular articulation and behind over the three upper dorsal vertebræ, was found. Harsh, almost cavernous, breathing, with some moist râles, was heard over the right apex. The child had fits of coughing, which lasted several months, and threatened to choke her. In short, the symptoms of enlarged glands in the mediastinum were found. Profuse muco-purulent expectoration. Given the diagnosis of hereditary syphilis, the following prescription was ordered: Iodide of potash, 150 grains; perchloride of mercury, 1 grain; currant syrup, 5 ounces. Three teaspoonfuls a day. Chlorate of potassium, 45 grains daily.

Under specific treatment the appetite returned in the first week, and the headache ceased; the heart regained its normal rhythm; the interscapular dullness disappeared, and the breathing improved. Similar changes in the chain of glands. After twenty-five days' treatment there was hardly any dullness over the right sterno-clavicular articulation. The vesicular murmur reappeared under the right clavicle, and the cough was less paroxysmal, while the spasms of suffocation diminished in frequency and then disappeared. The child was examined five months later. Heart and lungs presented nothing abnormal. She was seen next year on several occasions, and remained in good health. Tracheo-bronchial adenopathy of syphilitic origin was therefore undeniable in this case.

Balzer has reported tracheo-bronchial adenopathy in a patient with acquired syphilis. The patient had, among other symptoms, stridor and sucking-in. At the

autopsy sclero-gummatous adenopathy of the tracheo-bronchial glands was found, with fibrous syphiloma involving the lung and pleura.

In a case published by Raymond the patient had, among other symptoms, stridor and sucking-in. Syphilitic adenitis of the peritracheal glands was found, with narrowing of the trachea and compression of the right recurrent nerve.

#### 4. Syphilitic Gangrene of the Lung.

**Syphilitic gangrene of the lung** exists. "In such a case," says Mauriac, "the expectoration exhales an evil odour, and may even have the foetor of gangrene. The condition is not common, but it must be noted. It is, indeed, surprising that it is so rare, for many conditions favourable to gangrene are present—*e.g.*, necrobiosis of the parenchyma, contact of the air with the products of pulmonary disintegration, and stagnation of sputum in the dilated bronchi or in the gummatous cavities."

The following case (Feulard) may be quoted :

A man, thirty-seven years of age, who contracted syphilis when twenty, was seized, after some days of malaise, with pain in the side, fever, and expectoration. Auscultation revealed blowing inspiration and râles, localized behind to the middle third of the right lung. The expectoration was blackish, and of an alliaceous odour. Gangrene of the lung was diagnosed, and as the patient was syphilitic, he was put on iodide of potash. At first improvement occurred, but a month later the patient brought up foetid sputum containing blood and pus after violent fits of coughing. Within two months a cavity formed under the right clavicle. The general condition and the signs on auscultation led Duguet to diagnose phthisis in the cachectic period. As examination of the sputum, made by Feulard and confirmed by Chantemesse, revealed no tubercle bacilli, he was again put on iodide of potash in doses of 150 grains daily, in case the pulmonary lesion was perhaps syphilitic. After this treatment improvement was manifest, yet a fresh gangrenous vomica occurred a month later. Inunction of mercury was also employed, and complete success at once followed. In three months the cavity had filled up, and six years after Feulard was satisfied that the cure was maintained.

In the course of this chapter I have cited a case of Dr. Latty's—a girl eight years old, who was suffering from hereditary syphilis, taken ill with symptoms of **gangrene of the lung**. She brought up gummatous matter in the sputum, which was the colour of wine-lees, and terribly foetid. The patient recovered, and the evacuation of the gummata was followed by enormous retraction of one side of the chest.

#### 5. Syphilitic Pneumopathy, associated with Pulmonary Tuberculosis.

Let us now consider the possible association of syphilis and tuberculosis in the lung. This condition may arise in two ways. Sometimes tuberculosis complicates syphilis, at other times syphilis complicates tuberculosis.

In the former category tuberculosis appears in a syphilitic patient. It may occur early, within a few months of infection, or late, some years after. The appearance of tuberculosis is sometimes so early that it coincides with the roseola and the first mucous patches. I have asked myself in such a case whether syphilis did not hatch the latent bacilli.



I have seen two cases of this kind, one of them with Fournier. In a strong, well-built youth, twenty years of age, hæmoptysis and early signs of pulmonary tuberculosis at the left apex appeared two months after the chancre. Jacquinet, in his thesis, has collected eight cases of early tuberculosis during the secondary stage. The patients had cutaneous or mucous syphilides, and the syphilitic infection dated only a few months or a few years back. "In these cases the pulmonary tuberculosis ran its course in a few months. Statistics would seem to show that these grave cases of tuberculosis belong to the period in which syphilis is most virulent." This assertion is possible ; let us add that it is not absolute.

Tuberculosis most often makes its appearance during the tertiary period of syphilis. In the section on Syphilis of the Larynx we have seen that an ulcerated syphilide of the larynx may determine the fixation of Koch's bacillus ; so, too, in a healthy patient exempt from any tubercular taint, tertiary syphilis of the lung may favour the fixation of the bacillus and the growth of tubercles. We can thus explain how, after a long and fruitless search for Koch's bacillus in the sputum of patients with pulmonary gumata, it may finally be met with. If in the same lung the tubercle unites, as it were, with the syphiloma, it does not mean that a hybrid, anatomical condition exists in the shape of "*scrofulate de verole*," as Ricord said.

The syphilitic and tubercular lesions arise and develop side by side, though separately, and each on its own account. The anatomical proof of this has been given by Potain, during an autopsy in which he met with disseminated tubercles around a mass of white syphilitic pneumonia. This secondary infection of a syphilitic lung by tuberculosis permits us to paraphrase the old adage of Niemeyer : "The greatest danger for a syphilitic phthisical patient is to become tubercular."

According to Landouzy, the appearance of tuberculosis in a patient with old syphilis is not so serious as its appearance during recent syphilis. "The course of events appears to me to be quite different," says Landouzy, "in a patient who has had syphilis for some twenty years, and is attacked by tuberculosis. Patients showing this chronological variety of morbid association appear to me—in a tenth of the cases, at least—to present a peculiar tuberculosis which is, from the pathological point of view, rather fibrous, and from that of evolution, slow, apyretic, and localized. Referring to this category of patients who are suffering from old syphilis and recent tuberculosis, I am in the habit of saying familiarly that they go on to the *vérolate de tuberculose*."

Hereditary syphilis has an action upon the development of tuberculosis, which may be interpreted in various ways, but which is indisputable. "Clinical observation," says Fournier, "shows that hereditary syphilis predisposes to various diseases, doubtless by reason of the relative im-

poverishment of the organism. Hence the frequency of scrofulo-tubercular affections has long been marked in children born of syphilitic stock. It is certain that the syphilitic soil is eminently propitious to the culture of the bacillus, for from statistics and contemporaneous observation which have quite confirmed the results of our predecessors on this point, it is undeniable that patients with hereditary syphilis are very subject to scrofulo-tuberculosis, notably to affections of the bones (Pott's disease, coxalgia, etc.), and to tubercular lupus. It may be asked why the syphilitic person so often gives birth to a tubercular child, whether it results from the condition of cachexia, which is not constant, or from the transmission of a specific agent, which favours the development of the other. We know, too, that syphilis is one of the constitutional diseases which produces most of the abortions, of the strumous diseases of the glands, and of degenerated scrofula—in short, most of those puny, rachitic, and weakly subjects who are an easy prey to tuberculosis."

### 6. Hereditary Syphilis of the Lung.

Let us return to the important question of hereditary syphilis, which may affect the lung, as it does other parts. It may be early or late. **Early** syphilis is no longer seen. It is met with in still-born children, or in those who have lived a few months, and has only a pathological interest. Depaul had already observed among the cutaneous and visceral changes the lesion of the lung which Virchow described later under the name of **pneumonia alba**. White pneumonia, described by Parrot in 1877, and later in 1879 by Cornil, in children who died when five, six, and seven months old, is peculiar to the syphilitic cachexia of the new-born in the same way as the flint-like liver of Gübler. The term "white pneumonia" is correct, because the tissue is white or greyish. The lesion is sometimes disseminated in a lobular form, at other times confluent in a pseudo-lobar one. The hepatized nodules are smooth, hard, and dense, creak under the knife, and sink in water. The histological lesions may be thus summed up: Thickening of the walls of the bronchioles and of the alveoli, lesions of epithelial pneumonia, with desquamation into the alveoli, while the cells themselves have undergone granulo-fatty degeneration.

**Late** hereditary syphilis of the lung is not so well known, but is far more interesting. It is less frequent than hereditary syphilis of the larynx, for in 1886, out of a total of 212 cases, Fournier, in his "*Syphilis Héréditaire Tardive*," could collect only five cases of hereditary syphilitic phthisis. It may appear some months after birth, but is most often seen from the sixth up to the twentieth year, and possibly still later. It is therefore in most cases a disease of childhood or of youth, and sometimes also of mature age.



I need not dwell on the sclero-gummatous lesions of late hereditary syphilis. They are identical with those of the acquired disease. I shall simply quote cases which prove that syphilitic lesions of the lung may develop in children born of syphilitic parents.

Fournier speaks of a child, seven years of age, who suffered from hereditary syphilis, and died by accident of an acute intercurrent malady. Three small gummata, which had given rise to no symptoms during life, were found post mortem in the lung.

Lannelongue and Lancereaux have each seen a similar case. The discovery of gummata in the lung in these cases was a post-mortem surprise.

Fournier has reported a case of Dr. Latty's given on p. 242.

I have observed the following case of hereditary syphilis of the lung :

I was called in to treat a little girl of fourteen or fifteen months, whose father I had previously attended for syphilis. She suffered in succession from ocular troubles, otitis, and periostitis of both wrists, and then went back to Russia. Two years later the father came back to Paris to bring his son to me, who, as he thought, was sure to die. He showed all the signs of cavity in one lung, and had every appearance of phthisis. Knowing the father's history, I diagnosed hereditary syphilis, ordered specific treatment, and gave hopes of an early cure. My predictions were realized, for after a few days the sweats ceased, the appetite returned, the physical signs improved, and in a few weeks the trouble had disappeared. It is possible that a physician who was ignorant of the father's history would have treated the case as tuberculosis, and this error would have cost the boy his life.

The following case of pulmonary syphilis was reported by Legrand. The disease was hereditary, as the reader will see :

A man was admitted into the Saint-Antoine Hospital with cough, hæmoptysis, and the usual signs of phthisis. We were, however, in doubt from the strange localization of the lesion to the middle part of the lung, the absence of bacilli in the sputum, and, lastly, by the presence of Hutchinson's teeth. On inquiring into his past history, we found stigmata of syphilis, the chronological connection of which was so evident as to surprise us. This man, some years previously, had suffered from gummata of the tibia, recognized by Dr. Barbe; the astonishing thing was that he had afterwards contracted an indurated chancre of the penis, the scar of which was only noticeable for sixteen months. He had had syphilis on two occasions—first, **hereditary** (the dental deformities and the gummata in the lower limbs proved this); and, secondly, **acquired** (the hard chancre of relatively recent date proved this). Iodide of potash and mercury were administered, and soon cured the lung trouble.

I do not know of a more conclusive case than the following (Dubousquet-Laborderie and Gaucher) :

A little girl, eight and a half years old, was wasted and cachectic. She coughed continually, her appetite was gone, her temperature was 102° F., and her pulse 140. Her general condition, in short, was that of a patient in the last stage of phthisis. Cavernal breathing and gurgling were heard in the right supraspinous fossa. Large mucous râles were scattered throughout the whole chest. The diagnosis was pulmonary tuberculosis in the third stage. Treatment gave no results. Fever, cough, and wasting, and signs furnished by auscultation, underwent no change. The child complained of acute pain in the middle of the sternum. Pressure was very painful, and tubercular

osteitis was at first thought of. After a few days a tumour developed in the painful region. This tumour reached the size of a small orange, and fluctuated. The skin became thin, and ulceration commenced. Puncture gave issue to a puriform, gelatinous, and shreddy fluid. The unusual course of this tumour, which was taken at first for an abscess, and the nature of its contents, gave the idea of a gumma, more especially as the child presented dental changes which had previously been considered rachitic.

As it was felt that the lesion might be syphilitic, Gibert's syrup was prescribed,  $\frac{1}{2}$  drachm morning and evening. A few days later the diagnosis of hereditary syphilis was confirmed, because the father came to Dr. Dubousquet for syphilitic onychia of the left hand and thickening of the periosteum of the right tibia. Gibert's syrup cured both these lesions in a fortnight. The girl took the syrup for a week, and was then treated with mercurial inunctions and iodide of potash in gradually increasing doses until 10 grains were taken daily. Improvement was soon manifest. The strength came back, the evening fever disappeared, the physical signs diminished rapidly, the cavity filled up, and at the end of a month only faint tubular breathing was heard at the apex. The sternal gumma, after suppurating for some time, became covered by granulations, and soon healed. In about two months the child was completely cured, and was in good health ten months after the first recognition of these symptoms.

In cases of this kind we are given a clue to the diagnosis by the usual signs of hereditary syphilis, which are as follows :

(1) Dental malformations, indentation, cup-like excavations, transverse striæ, small size of the incisor teeth ; (2) ocular lesions, diffuse interstitial keratitis ; (3) aural lesions and troubles, deafness ; (4) malformations of the tibia, swelling of the epiphysis, bosses on the shaft, flattening of the crest (Fournier).

**Treatment.**—The drugs here, as in other cases of syphilis, are mercury and iodide. They both have a marvellous action upon syphilis, but had I to choose between the two, I should give the preference to mercury. The old preparations of mercury, the proto-iodide, van Swieten's liquor, and mercurial inunctions, etc., are of service, but nothing, in my opinion, is so valuable as an injection of biniodide of mercury in aqueous solution. Each Pravaz's syringe-ful represents  $\frac{1}{16}$  grain of the active principle. Fifteen injections are given, injecting daily from  $\frac{1}{16}$  to  $\frac{1}{2}$  grain of biniodide of mercury.

The injections are then replaced by iodide of potash in a daily dose of 30 to 75 grains, according to the tolerance of the patient. Ten days to a fortnight later the injections are again given.

The results obtained vary according to the varieties of the pulmonary syphilosis. In the case of sclerous broncho-pulmonary syphiloma, with fibrous change in the lung, the bronchi, and the pleura, improvement is delayed, and complete cure is rarely obtained, because of the definite change in the tissues. If the syphiloma of the lung is still in active growth, if there is a circumscribed gumma, or an infiltrating syphiloma, with acute, subacute, or chronic broncho-pneumonia, surprising results are sometimes



obtained. The proof is found in the history of the patients given in this chapter, or in my clinical lectures at the Hôtel-Dieu. Two patients in the Saint-Christophe Ward were cured, the one of acute, the other of chronic, syphiloma of the lung. Giraudeau's patient (p. 233) was cured in a few weeks. Gübler's patient, with cavity and symptoms of phthisis, was also cured in a short while. The patient in whom Fournier and myself found rapid breaking-down of the apex of the right lung and a syphilitic testis was cured in a few weeks. The young man who appeared in the last stage of phthisis, and in whom I found an enormous cavity in the right lung, recovered with truly marvellous rapidity. By grouping these cases and recognizing how health was restored to cachectic people who presented the picture of acute phthisis or of advanced pulmonary tuberculosis, we learn the imperative necessity of making a correct diagnosis and of employing proper treatment.

The irrefutable proof that all these patients, saved by specific treatment, really had syphilitic lesions of the lung, is that they were cured of **visible syphilitic manifestations which occurred in the other parts at the same time as the lesion in the lung.** The patient whom I saw with Fournier was cured simultaneously of syphiloma of the lung and of the testis. Giraudeau's patient was cured successively of pulmonary syphiloma, of vaginal gumma, and of periostitis of the forehead. Gübler's patient was cured of pulmonary syphiloma and of a tibial exostosis. Fournier's patient was successively cured of a phagedænic ulceration of the foot and of a syphilitic cavity of the lung.

So many accumulated proofs cannot, and ought not, to leave any doubt concerning the existence of pulmonary syphiloma and the efficacy of the treatment, which is sometimes surprising. In a suspected case of pulmonary tuberculosis let us always think of the possibility of syphilis; in dealing with a patient considered as a case of incurable phthisis let us still think of syphilis; and if repeated examinations of the sputum show the absence of Koch's bacillus, let us have immediate recourse to specific treatment—let us use injections of biniodide of mercury. **It is the touchstone,** and we shall perhaps obtain an unexpected cure.

## CHAPTER V

### DISEASES OF THE PLEURA

#### I. ACUTE SERO-FIBRINOUS PLEURISY—TUBERCULAR SERO-FIBRINOUS PLEURISY

**Pleurisy** is inflammation of the pleura. According to its **situation**, pleurisy may be partial, diaphragmatic, mediastinal, or interlobar; according to the **nature** of the effusion, it may be sero-fibrinous, hæmorrhagic, or purulent. The last two varieties will be described in this chapter. In this section I shall consider only acute sero-fibrinous pleurisy of the great pleural cavity, which, I may say in advance, is nearly always tubercular. Acute pleurisy, formerly called genuine, legitimate, or a frigore, is tubercular in most cases.

**Ætiology.**—Acute pleurisy is seen at all ages; nevertheless, it is exceptional in very young children, increases in frequency about the age of five or six years, and reaches its maximum in the adult, while it becomes rarer in the elderly. **Chill** has long been considered as the most potent or almost the only cause; this form has been, therefore, called “pleurisy a frigore.” And, indeed, it appears at first sight that chill, in its different forms, should often be held guilty. It may, indeed, be a provoking agent, but every case of acute pleurisy, even when it appears to be primary, is associated with an infection which is usually tubercular. This important question will be discussed later.

**Pathological Anatomy.**—The pleura, the false membranes, the pleuritic fluid, and the lung demand consideration.

The two layers of the pleura are congested and covered by fibrinous membranes; the serosa is infiltrated with leucocytes, the bloodvessels are dilated, the lymphatics are packed with white corpuscles, and the most superficial alveoli are the seat of catarrhal pneumonia. Under the fibrinous layer the pleura presents granulations, formed of embryonic tissue and of young vessels; these new formations, welded to those of the opposite layer, form adhesions, which are rare in the acute, but common in the chronic form of pleurisy.



The **false membranes** are more or less spread out over the surface of the pleura; they are easily detached. Their surface may be smooth or mammillated. Sometimes they extend from one layer to the other, like bands, and often float in the pleural fluid. They are formed of fibrin which enmeshes white and red corpuscles and epithelial cells.

The **effusion** varies in amount; some ounces or some pints are found. The fluid is fibrinous, transparent, and citron-coloured, and, according to my observations, only commences to take a rosy or hæmorrhagic tint when it contains 5,000 to 6,000 red corpuscles per cubic millimetre. On examining the fluid at the moment when it leaves the pleura during thoracentesis, I have always counted a large number of red corpuscles per cubic millimetre. It may be said of this pleurisy that it is **histologically hæmorrhagic**. When the fluid is left in a beaker, it is more or less changed into a gelatiniform mass, according to the amount of the fibrin present. If we pass this coagulated liquid through linen which is well wrung out, we obtain a fibrinous membrane, which owes its rosy colour to the red corpuscles contained in its meshes.

The fluid has a density of 1012 to 1022. The **tension** of the fluid in the pleural cavity is rarely negative; it may reach or exceed +10 or +15. The molecular concentration and the freezing-point of these liquids are nearly the same as those of blood-serum. Chloride of sodium ingested by the patient readily passes into the exudate, and appears to accumulate there, by favouring the increase of the fluid (Achard and Loeper).

The lung subjacent to the effusion is flattened, congested, and airless. The lesions of interstitial pneumonia and carnification of the lung are only seen in old pleurisies.

**Bacteriology.**—Examination of the fluid from cases of sero-fibrinous pleurisy proves that an effusion, which is tubercular in nature, may also contain ordinary pathogenic microbes. Thus, in these cases—which, however, are very rare—the existence of the streptococcus (Lacaze), pneumococcus, staphylococcus, *Bacterium coli* (Le Damany), Eberth's bacillus (Kelsch), and also of the *Micrococcus tetragenus*, has been proved. Inoculation of animals may prove the virulence of these microbes, without allowing any conclusion to be drawn as to the prognosis and pathogenic diagnosis of the pleurisy. These bacterial associations do not appear to influence the course of tubercular sero-fibrinous pleurisy, which hardly ever becomes purulent, although the effusion may contain pyogenic microbes. These cases are so rare that Netter, Vaillard, and Lemoine admit that every sero-fibrinous pleurisy which remains sterile should, *a priori*, be looked upon as tuberculous. We must, however, make an exception in the case of metapneumonic pleurisy, in which the pneumococcus may have disappeared at the time of puncture. Prudden wished to deduce from this fact the

general theory that microbes, whatever be their species, might play an important part in the onset of pleurisy, but would disappear when the effusion was clearly formed. A different opinion has been upheld by Ferney in France, Lecoq in Austria, and Pansini in Italy.

**Pleural Permeability.**—The study of the permeability of the pleura (Ramon, Tourlet, and Castaigne) aims at discovering whether the absorbent power of the serosa varies according to the age of an effusion, and whether it differs according to the nature of the effusion. Rénon and Latron have just carried out this research in two cases. Widal and Ravaut have proposed the use of salicylate of soda, injected in doses of 0.30 gramme, first under the skin, and then into the pleura. Salicylic acid has the advantage that it can be estimated with great precision and is more rapidly eliminated in the urine than methylene blue or iodide of potash. The two injections (subcutaneous and intrapleural) may be made at very short intervals. It is a question, in short, of a double proof by pathological physiology, and further study will be interesting.

Widal and Ravaut have drawn the following conclusions from the examination of five cases: In a case of primary tubercular pleurisy, the permeability of the pleura was diminished; in a case of pneumonic pleurisy it was altered as to the time, but not as to the quantity of salicylic acid eliminated; in three cases of pleurisy of mechanical origin, in patients with cardiac lesions or with Bright's disease, the permeability was slightly increased in two, while it was diminished in the third.

In the next section we shall discuss the cyto-diagnosis of tubercular pleurisy.

**Symptoms.**—How does acute sero-fibrinous pleurisy begin? The disease may begin with pain and fever, or may be latent. Chills and fever may open the scene. The patient is seized with dry, painful cough, and suffers on one side of the chest, most often in the region of the nipple,\* from a more or less sharp stabbing **pain in the side**, which is made worse by each attack of cough, and, indeed, by each inspiration. This pain, however, is not always acute; it may be very moderate or quite absent.

The patient has difficulty in breathing, so that the respirations are short, jerky, and incomplete, because of the pain. We cannot, however, call it true dyspnoea. The **pulmonary congestion** which so often accompanies pleurisy may also cause distress.

At this period, before any trace of effusion, the following signs are found: Percussion yields dullness, without special character and precise limits; the expansion is diminished on the diseased side, and auscultation

\* This pain in the side is not always at the level of the nipple, but may radiate in various directions. According to the most widely held opinion, it is due to neuritis of the intercostal nerves. See Peter, *Clin. Méd.*, t. i.



tion reveals either true friction sounds or a sound somewhat analogous to a fine *râle*, which may be heard at the back of the chest or in the axilla, both during inspiration and expiration. This sound is more moist and more liquid than the crepitant *râle* of pneumonia, and is also much more diffuse. Opinions differ as to the interpretation of this sound. Some consider it a friction sound, others a *râle*, due to the cortical pneumonia that often accompanies pleurisy (Trousseau); and Damoiseau has given it the name of "**friction *râle*.**" In some cases it is wanting.

The initial period lasts several days, and its approximate limit cannot be fixed. If appreciable exudation does not supervene, the pleurisy is said to be **dry**; but this dry form, which is so frequent in secondary pleurisies, or in inflammation of the chest, is **quite rare in acute pleurisy**. Laënnec doubts it; Woillez has recognized it only once in eighty-two cases; and I have very rarely met with it.

**Effusion**, then, is the rule in acute pleurisy, but it appears at an indefinite time, usually from the twelfth to the fifteenth day; I have even seen a case on the seventeenth day. Let us suppose, for the sake of description, that the disease is at the fifth or sixth day, while the effusion amounts to about a pint and a half. The indefinite signs of the onset have given place to new and definite ones. The diagnosis, which was doubtful, is now certain. The pain is better, and therefore the patient breathes more freely. On percussion, we find at the posterior and lower part of the chest **dullness**, which Piorry called "**fluid dullness**"; its upper limit does not end suddenly, but gradually shades off into resonance. Percussion of the clavicular region on the affected side gives exaggerated resonance (called "**skodaic**" by Trousseau), which disappears when the effusion becomes very abundant. Palpation shows that the thoracic vibrations are diminished or abolished behind even the effusion, and measurements taken with the cyrtometer (Woillez) reveal the enlargement of the chest.

Auscultation above the effusion, in the axilla, or at the antero-superior part of the thorax, may reveal friction sounds or a friction *râle* at different points; but these friction sounds are no longer heard over the area occupied by the fluid, because the two layers of the pleura are separated from one another.

Auscultation over the region of the effusion reveals four signs: (1) Absence of vesicular murmur; (2) presence of distant tubular breathing, which is sometimes muffled, at other times clear: it is audible only during expiration, and principally at the end of expiration; (3) quavering of words, pronounced in a high voice by the patient, called by Laënnec "**ægophony**"\*

\* *Ægophony*, *pectoriloquy*, and *bronchophony* are three physical phenomena which are characterized by the echoing of the voice; in *ægophony* the voice is bleating, while it is diffuse in *bronchophony*, and clearly articulate in *pectoriloquy*.

("the bleating of a goat"); and (4) **aphonic pectoriloquy**\*—that is to say, the clear and articulate transmission of words pronounced by the patient in a whisper (Bacelli, Guéneau de Mussy). These signs are usually most marked at the lower angle of the scapula, and are of great importance, but yet not one of them is pathognomonic of pleural effusion. They may lose their purity and become disfigured if the lung be congested and resistant beneath the effusion (pleuro-congestion).

I shall now say a few words on the correct method of percussion, and the means used in determining absence or diminution of the vocal fremitus. If percussion is to give reliable information, it must not be performed forcibly, as is too often done, for the sound then obtained destroys in a great measure the delicate shades of dullness, or of impaired resonance which indicate the presence or the absence of effusion. Care must therefore be taken to percuss as lightly and softly as possible, in order to give a true result.

I may make a similar remark as regards the disappearance of the vocal fremitus. If the patient be made to count in a high voice, the vibrations are transmitted—less strongly, it is true—on the side of the effusion; but they are still transmitted, and their presence may give rise to error. If, on the contrary, the patient be made to count in a slightly raised voice, the vibrations may still be transmitted to the healthy side, but are quite absent over the effusion.

As the effusion increases (2 to 3 pints), certain signs are modified: the ægophony becomes less clear, and rather bronchophonic in character; the soft tubular breathing on expiration takes a bronchial tone, and is also audible during inspiration; auscultation reveals the exaggerated breathing, called "puerile," on the healthy side; and the effusion, by its constant progress, **displaces** the neighbouring organs.

The displacement of organs is of value in estimating the amount of the effusion. It has much less value in effusions on the right side, because the liver is much less mobile than the heart, and is only pushed downwards when the fluid amounts to at least 3 pints. On the other hand, in effusions on the left side, displacement of the heart is a sign on which I cannot lay too much stress. The **displacement of the heart** is shown by inspection, palpation, and auscultation. It is not only the apex of the heart which is displaced, but the whole heart itself, and with the change of position it also alters its relations to the chest-wall. In order to ascertain the displacement, we must look for the **maximum point of the impulse**. My numerous observations lead me to conclude, approximately, that this maximum point reaches the left border of the sternum in effusions of 20 ounces; it reaches the right border of the sternum with an effusion of 40 ounces; and lies between the

\* Laënnec had already pointed out this phenomenon, and Bacelli has given the conclusions which may be drawn from aphonic pectoriloquy as a sign of pleural effusion.



sternum and right nipple with an effusion of 60 ounces. This is the moment at which **thoracentesis** must be performed.

When these effusions are on the left side, dullness replaces the normal tympanitic sound in Traube's space. The **semilunar**, or Traube's, **space**, is situated at the **left** base, and corresponds in part to the pleuro-parieto-diaphragmatic cul-de-sac. Its upper limit is the fifth or sixth costal cartilage in front, and the ninth or tenth ribs behind, while its lower limit is the edge of the thorax. At its middle part—that is to say, in the nipple line—the vertical height is about 4 inches, and its width about  $4\frac{1}{4}$  inches. The costal wall and its pleura, the diaphragm and its pleura, the colon and the stomach correspond anatomically to the semilunar zone; the lung only touches the upper limit (Jaccoud). In the normal state percussion of this zone gives a tympanitic note; on the other hand, in a large left effusion the diaphragm is pushed down, and dullness replaces resonance. It is true that normal resonance may also disappear, at least in its upper part, as the result of adhesions.

When the effusion is very large (5 pints and more), the skodaic note in the clavicular region disappears, the dullness becomes absolute over all the affected side, the displacement of organs reaches its maximum, the whole mediastinum is pushed back, and the obstruction in the pulmonary circulation, without doubt, leads to pulmonary thrombosis, and perhaps to sudden death, which sometimes occurs in large effusions. On auscultation, we find total absence of all normal or abnormal sounds, or else tubular breathing, which may be so loud as to resemble cavernous or even amphoric breathing (Landouzy).

**Paresis** of the thoracic muscles on the affected side is more or less marked, but constant. It is accompanied or followed by muscular atrophy, which may begin with the onset of pleurisy and may remain after it. These muscular atrophies affect the intercostal and pectoral muscles, the latissimus dorsi, the serratus magnus, etc., and it is easy to appreciate them, either at first sight or by the respiratory troubles which they cause. These muscular changes explain in part the modifications in the respiratory type, the smaller amplitude of the chest (on the affected side), its relative immobility on inspection and palpation, and the respiratory restraint which results. They also enter largely into the external deformities, as well as into those of the skeleton which accompany and survive pleurisy.

**Functional Symptoms.**—As soon as the fluid reaches a certain amount, the patient lies on the affected side, in order to give free play to the healthy lung. He experiences a sensation of distress or of heaviness, but dyspnoea is rarely seen.

Dyspnoea is not a usual symptom of pleurisy with effusion. The effusion, even when it amounts to 3 pints, causes little acceleration of the

respiration rate. I do not speak, of course, of the early pain in the side, which is often accompanied by acute dyspnœa, and I make exception if congestion of the lung is also present. In all other cases of acute, subacute, or latent pleurisy, however, dyspnœa is not marked, even with an effusion of 3 pints ; and, as we shall see in a moment, when discussing the indications for **thoracentesis**, we should be quite wrong in waiting for dyspnœa to decide the prognosis and the treatment of pleurisy.

The conclusion from this absence of dyspnœa, even when the fluid is considerable, is that dyspnœa indicates the presence of **complications**. When the respirations in pleural effusion exceed thirty, the reason is either that the fluid is very abundant, amounting to about 5 pints, or that complications are present. On careful search, we shall find that the pleurisy is secondary, and has appeared in the course of Bright's disease or cardiac mischief. Other inflammations, such as double pleurisy, bronchitis, pneumonia, pericarditis, inflammation of the chest, or congestion of the lung, will be discovered.

I must dwell for a moment on **congestion of the lung**, which so frequently accompanies pleurisy. This fact has been emphasized by Potain. Congestion of a part of the lung often accompanies pleurisy, especially in its period of formation ; as a result, the signs of effusion are somewhat modified by those due to the affected lung—ægophony is not pure, but becomes broncho-ægophony ; tubular breathing, instead of being distant and muffled, becomes more harsh ; the quality of the dullness and the shades of the thoracic vibrations are modified. When we are not familiar with these points, we may miss an existing effusion and refer the signs to congestion of the lung ; or, on the other hand, we may fancy the fluid to be abundant when it is as yet scanty. I shall consider this point again, under Diagnosis.

In acute pleurisy the **fever** is moderate and sometimes absent : the temperature does not exceed 102° F., as a rule ; the defervescence may be rapid or slow, and occurs at ill-defined times, in some cases with symptoms of **crisis**.

**Course—Duration—Termination.**—Pleurisy is a disease of surprises. Its course is insidious, and the **extreme irregularity of its habits** is not the least important point in its history.\*

I was therefore very surprised, at the Académie de Médecine, to hear that pleurisy is a **cyclical**† disease. I have only to consult our case records to prove that pleurisy is **quite the opposite of a cyclical malady**. In pleurisy we cannot foresee the succession of symptoms, the temperature curve, the time of defervescence, or the moment when the effusion appears

\* Dieulafoy : "Des Irrégularités de la Pleurisie Aiguë," *Gazette Hebdomad.*, 1878, No. 3.

† *Académie de Médecine*, April, 1892.



or is absorbed. In one case pain and fever are marked, and the effusion appears between the twelfth and the fifteenth day. In another the disease is insidious, and effusion exists for a month or six weeks, when the patient comes to the hospital with some distress, which often scarcely merits the name of dyspnœa. I have several times seen fever persist, although the effusion diminished. Binet gives a remarkable example.\* In many cases fever disappears with or without symptoms of crisis, the patient considers himself cured, and still the effusion increases. In fact, pleurisy is so irregular in its course that it presents a different appearance in each patient. It is not possible to say whether the febrile period will be of long or of short duration, whether the effusion will be abundant or scanty; at what time defervescence will occur, and whether it will coincide or not with absorption of the fluid; whether this absorption will be slow or quick; and whether, in spite of the fall of the fever, the fluid will not continue to increase. Do not these clinical considerations justify the assertion that pleurisy proceeds in **quite a different way** to cyclical diseases? We cannot, therefore, speak of a cycle in pleurisy. This question will be referred to under the Indications for Thoracentesis.

If the effusion is slow to absorb, the insufficiency of the hæmatisis and the lack of appetite often cause weakness and anæmia. In exceedingly rare cases sero-fibrinous pleurisy has ended by vomica. When pleurisy terminates favourably, the fluid is absorbed; the displaced organs recover their former position; the lung resumes its function; and on inspiration and expiration we hear a grating friction sound, or *bruit de cuir neuf*, which indicates absorption of the fluid and presence of false membranes on the layers of the pleura. This redux friction may be heard at several spots in front and in the axilla. It is sometimes so marked that it may be felt on palpation. These signs remain long after the pleurisy. Impaired resonance and weak vesicular murmur often persist months after recovery.

The immediate **prognosis** is not grave. We must, however, not forget that large effusions may determine thrombosis of the pulmonary vessels or cardiac embolism, and induce syncope, rapid asphyxia, and death. This important point will be discussed under Thoracentesis. **Convalescence** is usually protracted, and many patients, even though taken ill while in excellent health, remain weak, and for several months carry the stamp of their disease.

Pleurisy at times leaves false membranes and adhesions, which affect the functions of neighbouring organs (diaphragm, lung, and pericardium), and may ultimately give rise to encysted effusions. Another important fact as regards prognosis is that the most frank pleurisy is often the beginning of tuberculosis, which becomes evident months or years later.

\* *Archives de Médecine*, April, 1884, p. 406.

**Diagnosis.**—At first, before effusion appears, diagnosis may be difficult. Pleurisy should not be confused with lobar pneumonia, for the latter begins with a violent rigor, very high temperature, intense dyspnœa, crepitant râles, and rusty sputum. The diagnosis, however, may be difficult, for the friction râle of pleurisy somewhat resembles the râle of pneumonia. **Inter-costal neuralgia** and **hepatic colic** may simulate the pleuritic **pain in the side**, but they differ in the absence of fever and of physical signs. Febrile **pleurodynia** shows more analogy with pleurisy, and it may coincide with a partial dry pleurisy. This question has been discussed under Inflammation of the Chest.

Pulmonary congestion, massive pneumonia, and spleno-pneumonia (Grancher) have several signs in common with pleural effusion. In the case of pulmonary lesions, however, the dullness is less complete, the thoracic vibrations are less affected, the neighbouring organs are not displaced, and the aphonic pectoriloquy is less clear. In spite of all these distinctive signs, I admit that an early diagnosis is difficult in some cases.

It is also important to recognize the diseases which may be associated with pleurisy—*e.g.*, the coexistence of **pulmonary congestion** and pleurisy (pleuro-congestion), for the presence of the former modifies the signs of effusion. In consequence of the congestion, the **distribution of the fluid in the pleura is modified**. The congested lung becomes more dense, and does not allow itself to be flattened. It sinks in the fluid, raising the level, so that, without careful examination, the effusion would appear very abundant, when it may really be quite scanty. The limits of the dullness, the nature of the tubular breathing, the characters of the voice, and the frequent presence of bronchial râles are the most important points.

We must distinguish between effusion due to pleurisy and that due to hydrothorax. Hydrothorax is not rare in the course of acute or chronic nephritis, and sometimes occupies both pleuræ. It is sometimes associated with œdema of the extremities and of the lung. This is chiefly the case in acute nephritis. At other times hydrothorax is an isolated manifestation of chronic nephritis, just as œdema of the larynx or of the lung may be. The general rule is: hydrothorax due to nephritis causes more severe dyspnœa than pleuritic effusion does, because hydrothorax in Bright's disease is nearly always associated with œdema of the lung or with uræmic dyspnœa. The discovery of symptoms of "Brightism" and of albuminuria will complete the diagnosis.

It is still necessary to distinguish effusion due to pleurisy from hydrothorax consecutive to heart disease.

The diagnosis of effusions into the pleura presents other difficulties, as we shall see. For example, a patient suffers from dyspnœa. He is examined, and on one side of the chest signs of a large effusion are found. The dull-



ness is complete, or nearly so, the thoracic vibrations are diminished, and ægophony, aphonic pectoriloquy, and tubular breathing, or even cavernous and amphoric breathing, are heard. A patient who presents these signs, or some of them, may have pleurisy with effusion, just as he may have a cyst in the liver, the spleen, or the kidney, which has pushed up the diaphragm and encroached on the thoracic cavity. Another patient may have cancer of the lung, or malignant hypertrophy of the thoracic glands, etc. Vergely has clearly shown these points in the case of a patient who presented all the signs of pleural effusion and in whom an adeno-carcinoma was found. Ægophony, aphonic pectoriloquy, and blowing breathing are certainly of great value, but these signs may exist without any large effusion in the pleura. False membranes, tumours, flattening of the bronchi, and condensation of the lung tissue, may modify the normal conditions of auscultation to an extent not yet well defined, and may sometimes simulate pleural effusion. In a doubtful case the surest sign of pleural effusion on the left side is **displacement of the heart**. When the effusion is on the right side, **depression of the liver** should be taken into consideration. We shall see, when we study hydatid cysts of the liver and of the spleen, upon what signs a diagnosis must be based if these cysts simulate pleural effusion.

When pleurisy has been recognized, it is also necessary to diagnose its cause, to ascertain whether it is sero-fibrinous, hæmorrhagic, or purulent, and whether it is associated with tuberculosis which is latent or is already at work.

We cannot say without exploration whether an effusion is sero-fibrinous or hæmorrhagic. We often expect to draw off serum, and are surprised to find blood-stained fluid.

**Clinical Varieties.**—Pleurisy presents numerous clinical varieties, a fact which it is important to remember.

1. **Latent Pleurisy.**—Acute pleurisy does not always begin with the acute symptoms mentioned above. The pain is sometimes trifling, and the symptoms of onset are so slight that in hospital we frequently see people who have been at work without being able to fix the start of their illness. It is not rare to see **latent** cases in which fever, pain, and dyspnoea are absent, while the effusion reaches considerable proportions unnoticed by the patient, but these cases may be accompanied by fever of the intermittent type. It usually indicates infective changes in the fluid.

2. **Rheumatic pleurisy** may be dry, or accompanied by effusion; it may be simple or double; it appears, changes, and disappears with great mobility (see **Rheumatism**).

In many cases pleurisy is provoked by a **neighbouring** or by a remote lesion (hydatid cyst of the lung, of the liver, etc.).

**Treatment.**—In acute pleurisy two chief elements—pain and effusion

—demand relief. For pain, we employ wet-cupping, leeches, injections of morphia, antipyrin; later, if the effusion makes rapid progress, what line of treatment must be followed? Blisters have only a very limited use in the treatment of pleurisy. Many persons have three, four, and five blisters, and are exposed to the risk of cystitis, erysipelas, or of a crop of boils, without gaining the least benefit. As I believe that blisters have no real effect on the effusion, I abstain from prescribing them.

I think that other medical means, such as bleeding, purgatives, diuretics, and sudorifics, have quite a secondary place in the treatment of pleural effusions. The more I see, the more I recognize that these means are useless and sometimes harmful. We may therefore discuss the value of, and the opportunity for, surgical intervention, or **thoracentesis**.

**Thoracentesis.**—To my eminent teacher, Trousseau, belongs the honour of having clearly stated the indications and operative technique of thoracentesis. In spite of keen opposition, Trousseau so popularized this operation, which had been previously neglected, that he may justly be considered as its inventor. He only punctured the chest when the effusion threatened the patient's life, and the operation was performed with Reybard's trocar.

In 1869, however, when I designed the aspirator and applied the method of aspiration to the treatment of pleural effusions, the technique of thoracentesis was so simplified that the old method was gradually abandoned, and it must be admitted the new one was soon abused. As thoracentesis by aspiration has been universally adopted, I shall describe my published method.

**Indications.**—On the question of thoracentesis, the first point for discussion is that of the indications. Should we operate in spite of fever, or wait for defervescence? Should we employ aspiration in moderate effusions, or reserve it for large ones? How far should we consider the complications which may arise? These questions, which have been often debated and differently decided, are reduced to the following proposition, which sums up the question of opportunity: Given acute pleurisy with effusion, two cases may present themselves—in **one thoracentesis is urgent, in the other it is debatable**. When is it urgent, and when is it debatable? The discussion should proceed on these lines.

The **urgency** of thoracentesis must be based only upon the estimation of the **quantity of fluid effused**. The presence or the absence of fever or of dyspnoea are secondary considerations. We must, before all, consider the amount of the effusion. We cannot rely upon dyspnoea, for it is a faulty guide. Large effusions are sometimes associated with slight distress, and patients may have three or four pints of fluid in the pleura, and still be able to walk about.



Trousseau says that a nursing woman, who carried her child, walked from Saint-Eustache to the Necker Hospital without feeling much fatigue. Six pints were drawn off by thoracentesis. A carman of whom Andral speaks went on driving his horses, and felt but slight discomfort, although he had a very large effusion. We see, says Landouzy, people who walk several miles to hospital, and complain of stomach-ache, or of attacks of fever, although they have enormous effusions. I have often seen similar cases. I have performed thoracentesis in a student who came regularly to the hospital, and felt very little distress, in spite of an effusion amounting to 4 pints. **Dyspnœa is quite deceptive.** If we wait until dyspnœa appears before evacuating an effusion, we shall delay until the patient's life has long been in danger from the quantity of the effusion before our decision has been made.

With still more reason, thoracentesis should not be delayed until the patient has cyanosis of the face and of the fingers, as some authors would advocate. The cases of sudden and rapid death caused by large effusions have not all been published. It is a pity, for we should perhaps be less severe on thoracentesis, and should see its indications better.

Trousseau saw sudden death in the course of pleurisy on three occasions. One of his patients had **such slight** distress that thoracentesis was postponed, but next morning the patient died. Landouzy has reported a case in which dyspnœa was absent. This fact did not prevent sudden death from pleural effusion. Dujardin-Beaumetz's patient had neither dyspnœa nor cyanosis, and yet his death was sudden. Oulmont quotes the case of a man who was talking with some friends in his garden. He went to lie down, and fell dead from an effusion in the pleura.

Two patients whose histories have been reported by Binet and Legrand had no dyspnœa. One suffered so little that he refused thoracentesis, and asked that it might be put off. Two hours later the effusion proved fatal. The other patient, who was in apparently good health, also died suddenly from the same cause.

After such information, is dyspnœa a necessary indication for the urgency of thoracentesis? And we know these mishaps are not the only ones; the majority are never published. I have collected forty cases, which I brought before the Académie. How many would there be if we could make a complete list?

These cases are, for the most part, copies of one another. At every turn we see that the patient who dies suddenly has been suffering from an effusion, causing only **insignificant** dyspnœa—so insignificant, in fact, that in many cases thoracentesis has been rejected, or imprudently put off till next day.

I think that we must finally dismiss the inexact indications upon which the question of urgency in thoracentesis has been based. The duration of

the pleurisy matters little. It is not a matter of importance whether such troubles as fever or dyspnoea be present or absent. The amount of the effusion is the key of the situation.

At the risk of repeating myself on this important point, I say that dyspnoea is a false sign and a treacherous guide when we have to decide upon the urgency of thoracentesis. To wait for dyspnoea and, *a fortiori*, to wait for cyanosis of the face and of the fingers, is gratuitously to anticipate a catastrophe. If we search in the hecatomb of patients who have died suddenly because puncture was not performed in time, we shall find many examples which show clearly that a large effusion may prove fatal, even though it has caused but slight dyspnoea. It is much more likely to kill if dyspnoea be intense, while precious time is lost in applying blister after blister.

These cases of sudden or rapid death may result from various causes. Sometimes they are due to clots (thrombosis or embolism) which arise in the heart, the large vessels, or even in the lung itself. If the clot is formed in the large pulmonary veins, or in the left side of the heart, it often causes cerebral embolism, with its results—viz., apoplexy, hemiplegia, aphasia, etc. If the clot is formed in the right heart or in the pulmonary artery, it may produce rapid asphyxia and death, as in cases quoted by Paget, Smith, and Blachez. The patients suffered from left pleurisy, and died suddenly from syncope. At the autopsy the clot had originated in the trunk of the pulmonary artery, and reached into the smaller vessels.

In a certain number of cases death has supervened suddenly from syncope, the effusion being situated more often on the right side than on the left, but it is not easy to explain the reason.

Louis was therefore wrong in stating that simple pleurisy is not a cause of immediate death. "A patient may die suddenly from acute pleuritic effusion" (Trousseau). These accidents can be set down to the effusion, and therefore, I repeat, the urgency of thoracentesis must be based solely upon the amount of the effusion. But, it will be asked, at what moment does urgency arise? Is it when the effusion amounts to 4 or 5 pints? And, further, how can the quantity be estimated? How can we ascertain that it reaches 3 pints or more?

My analysis in cases of sudden death shows that death has never been caused by an effusion of less than 4 pints. Once only (Blachez) the pleura contained 3 pints of serous fluid. This exceptional case ought not to be included, and I estimate that, in a well-made adult, when the effusion reaches about 4 pints, the urgency of thoracentesis is clear.

We must consider the estimation of the amount of fluid. Upon what signs and symptoms are we to rely? Careful study of the signs of pleurisy shows that in small effusions tubular breathing is muffled and limited to



expiration; in moderate ones (2 to 3 pints) it has a bronchial tone, and is present during both inspiration and expiration; in large effusions (4 pints or more) the breathing is in some cases cavernous, but in others it is inaudible. These auscultation data, however, are not absolute—that is to say, they are insufficient for the estimation of the amount of fluid present. I would say the same concerning measurements with the cyrtometer (Woillez). This method, though excellent at times, is often faulty. We must, therefore, associate the preceding signs with the more exact information furnished by the extent of the dullness and by the displacement of organs.

Let us take, for example, a left pleurisy. When the dullness and the absence of fremitus reach behind as high as the spine of the scapula, the resonance in Traube's space has disappeared, and a dull note replaces the skodaic note in the clavicular region, and especially when the heart is displaced, so that the maximum of the apex-beat is situated at the right edge of the sternum or between the sternum and the right nipple, although at this moment the pleural cavity may not be filled to its maximum, such signs in an adult denote that the effusion is about 4 pints. Thoracentesis is then urgent. Operation is imperative, and we must not forget **that procrastination is an unfortunate formula** which costs patients their lives.

When the effusion is situated on the right side, the problem is rather more difficult, because there is no displacement of the heart. Nevertheless, percussion and examination of the vocal fremitus give similar information to that which I have just mentioned. I may add that depression of the liver, when present, is a sign of great value. The liver is only pushed down by large effusions, which I estimate at about 3 pints. The estimation of the effusion is therefore rather more difficult on the right side, but it is quite as important, for the statistics given above show that sudden death is more frequent in right than in left pleurisies.

The discussion so far has referred only to simple pleurisy. The same principles are applicable to cases with complications. The direct or indirect complications of pleurisy, such as pulmonary congestion, old adhesions, valvular lesions of the heart, or pericarditis—in short, all the lesions which impair the pulmonary circulation or narrow the field of hæmatosis—are not a contra-indication to thoracentesis. On the contrary, they demand it as soon as the effusion reaches large proportions; only the evacuation of the fluid in such a case demands precautions, which will be indicated later. The estimation of the quantity of fluid is sometimes difficult, as witness the very numerous cases in which the more or less congested lung makes the amount of fluid appear larger than it really is. These facts have been emphasized by Potain. It is really only by the most delicate differences in auscultation and percussion that we can clinically

distinguish the part due to congestion of the lung from that due to effusion. The worst that could happen in such a case would be to overestimate the quantity of fluid. But where would be the harm? Is it not better to withdraw 2 pints of fluid when the urgency is not absolute than to expose the patient to sudden death by not performing thoracentesis in time?

I have just discussed "thoracentesis of urgency." Apart from urgent thoracentesis, in which the surest guide is the quantity of fluid effused, the operation is disputable in all other cases. Some admit it, others reject it, and certain authorities, indeed, consider it harmful. Let us discuss these different opinions.

As long as the temperature is raised—that is, as long as the acute stage of pleurisy exists—it is better to wait for defervescence before deciding. If the absorption of the effusion takes place naturally, and appears to be rapid, it is useless to intervene. If, however, the effusion remains stationary, or absorption appears slow and difficult, the fluid must be drawn off. Fluid cannot remain long in the pleura with impunity. The displaced organs become fixed in their vicious positions; the flattened and adherent lung acts badly; two of the chief functions in the economy—*i.e.*, hæmotosis and circulation—are compromised, without counting the passage of the inflammation into a chronic state and the liability to purulent changes (Trousseau). Thoracentesis, performed at the right moment, may shorten the malady by some weeks, and cause the fall of the residual fever which often accompanies effusions that are slow to absorb.

**Operation.**—Up to 1869 only Trousseau's method was in use. He punctured in the sixth or seventh intercostal space, and 2 inches from the outer border of the pectoralis major—that is, in the axilla. A small incision was first made in the skin in order to prepare the way for the passage of the trocar, which was at once thrust into the chest. The tube of the trocar was armed with goldbeater's skin, which, by acting as a valve, prevented the entrance of air into the chest during inspiration. The fluid escaped from the chest first in jerks, then in drips, and during the operation the patient was generally seized with fits of coughing, which were distressing, and sometimes "violent, irrepressible, and very sharp." Trousseau considered that this cough favoured the outward flow of the fluid, and in some persons it lasted for part of the day. Let me add that towards the end of the operation the fluid was commonly coloured red by admixture with blood.

Thoracentesis thus performed, although not a very difficult operation, required some skill, and at times some boldness, on the part of the surgeon, and some resignation on that of the patient. It was, therefore, reserved for urgent cases, and performed by a restricted number of physicians.

When I invented the **aspirator**, and performed aspiration for effusions



in the pleura, the old technique was replaced by a needle-prick so trifling that hardly any mark remained on the skin after the operation, and the fluid, instead of being forced out by jerks, with distressing attacks of coughing, passed into the aspirator from the thoracic cavity unknown to the patient.

Thoracentesis was thus brought within reach of the least experienced physician. It became the most easy and the least painful of all operations, and the most junior student has to perform it in my wards.

My method is as follows :

The patient sits up in bed, with his arms held forward. I mark the exact spot upon the skin, which has previously been washed with a solution of sublimate, and insert the needle **behind** in the seventh or eighth intercostal space, in a line with the inferior angle of the scapula. This point is lower and more posterior than in the old procedure. It has the advantage of attacking the fluid in a more dependent position.

Thoracentesis should be performed with an aseptic No. 2 or No. 3 needle, and not with a larger instrument. The permeability of the needle is proved by means of a silver wire. The needle is connected with the aspirator by indiarubber tubing ; a vacuum is created in the bottle, and the puncture is then made. For this purpose, the operator with his left index-finger finds the intercostal space, so as to define the rib above with the dorsal surface of the finger and the rib below with its palmar surface. He then uses the extremity of the index-finger as a guide, and holding the aspirating needle in the right hand, penetrates the chest wall with a sure thrust. The needle is pushed in about 1 inch, the tap corresponding with the aspirator is opened, and the fluid flows through the indicator tube into the bottle. If the fluid does not appear, the needle has not been sufficiently pushed home. It is pushed on boldly, the previous vacuum indicating the exact moment at which it meets the fluid. The aspirator, once filled, is slowly emptied, and this temporary delay is beneficial to the lung, which does not lend itself to too rapid expansion. This manœuvre is repeated several times, according to the capacity of the aspirator, and the flow is stopped after 2 pints of fluid have been drawn off. The needle is withdrawn, and the operation only takes eight or ten minutes. Scarcely any trace of the puncture is to be found on the skin, and no dressing is needed.

When the operation is properly performed, the patient should suffer neither fits of coughing nor pain. If pleurisy is associated with other lesions, and the field of hæmatisation is narrowed by cardiac or by pulmonary lesions, if pleural adhesions are thought to exist, and especially if during the operation the patient complains of a feeling of tearing or pain in the interior of the chest, it is better to stop the flow, and repeat the operation next day. These precautions, however, which are rightly very necessary in pleurisy with complications, are rarely required in simple pleurisy.

What must be our line of action as regards the fluid remaining in the chest? If the effusion is very large and exceeds 4 pints, we remove 2 pints next day or the day after, and so on until the effusion is drained. If, however, the quantity of fluid has been primarily estimated at about 3 pints, and 2 pints have already been withdrawn, is it then necessary to repeat the operation? I think that it is not advisable. In effusions estimated at about 3 pints I have often removed two-thirds only, and have found that recovery went on rapidly, the **residue being more quickly absorbed after part of the fluid had been withdrawn**. I think that a repetition is only indicated when the fluid remaining in the pleura amounts to 2 pints or more.

We must not forget that in some cases the fluid, after evacuation, reforms rapidly and obstinately. We must then perform thoracentesis as often as may be necessary, carefully examining the patient for some days after the operation, as the fluid may form again insidiously, and cause sudden death. If thoracentesis is performed in time, patients should not die from pleuritic effusion.

In performing thoracentesis, the choice of the aspirator is indifferent. However, the rackwork aspirator appears to me the most convenient. The choice of the needle is important, and I recommend **exclusively** No. 2 needle, the calibre of which measures only 1.2 millimetres in diameter. Several objections have been made to this needle—that it permits but a slow flow of fluid, that it is easily blocked, and that its sharp point may injure the lung. What foundation is there for these objections?

The small bore of the needle retards the flow of fluid, but this is beneficial to the patient, for the slow flow allows the lung to expand gradually, and prevents painful fits of coughing.

The hypothesis that so fine a needle may easily be blocked is hardly ever realized, and, supposing it does happen, we need only withdraw the needle and make a fresh puncture.

Another objection to the needle is that its point may wound the lung. In order to prevent this accident, a special trocar has been invented (Castiaux).

I have never seen the lung wounded by the needle. The lung, which is compressed and pushed back by the fluid, expands slowly, and does not meet the needle quickly. Further, a simple manœuvre prevents every chance of this. We need only withdraw the needle gradually as the fluid flows out, and to make it turn, so as to bring it almost parallel with the intercostal wall.

**Consecutive Accidents.**—Congestion and œdema of the lung, albuminous expectoration, slow and sudden asphyxia, syncope, hemiplegia, apoplexy, more or less rapid death, and purulent changes in the fluid, have been seen after thoracentesis.



Aspiration has more than once been accused of these mishaps, an accusation which Reybard's trocar did not escape. Let me at once say that these accusations have no foundation.

The abuse of thoracentesis has arisen from aspiration. I know this fact, and regret it ; but as regards its abuse, there is at times a question, if not of inexperience, at least of imperfect knowledge of the technique. It is not sufficient to have in hand a needle and an aspirator ; it is also necessary to know **how to use them**.

I shall analyze, therefore, the accidents imputed to aspiration, seek their cause, and discuss their worth, hoping to prove that, with precise indications and proper technique, thoracentesis by aspiration is the most innocent of all operations.

**1. Albuminous Expectoration—Asphyxia.**—Dyspnœa and asphyxia, which I place in the first group, arise as follows : Immediately or a little while after thoracentesis, the patient, who is seized with cough and distress, brings up frothy and blood-stained albuminous sputum, and the fine râles of pulmonary œdema are heard on auscultation. In mild cases the symptoms rapidly improve, but in other cases the complication is very formidable. The cough is paroxysmal, the dyspnœa increases, and the patient brings up as much as 3 or 4 pints of expectoration, which in the receiver is divisible into several layers, the upper frothy and yellowish, the lower more dense and albuminous. The intensity of the dyspnœa, the duration and the quantity of the expectoration, are variable, and the patient only regains his normal condition at the end of twelve or of twenty-four hours. Lastly, in some exceptional cases (I know only of six) the results have been fatal, and asphyxia has come on so rapidly after thoracentesis that patients have died in a few minutes.

Since 1853 these cases of dyspnœa have been studied and variously interpreted. How can we explain the albuminous expectoration and the asphyxia ? No wound of the lung has been found post mortem, and supposing that puncture of this organ may have allowed the fluid to pass into the bronchi, it would also have allowed air to pass from the bronchi into the pleura, causing symptoms of hydro-pneumothorax. Wounds or spontaneous perforation of the lung, therefore, cannot be blamed. We must look to **acute œdema of the lung** as the cause of albuminous expectoration, dyspnœa, and asphyxia (Hérard) ; but the **cause of the acute œdema** still remains to be explained.

The **method of aspiration** has been blamed. It has been said that aspiration lowered the tension in the pleura, and allowed the fluid to flow out too quickly. How little foundation exists for this accusation is seen from the reports, which show that in sixteen cases of thoracentesis followed by albuminous expectoration (Terillon's thesis), the operation had been

performed twelve times with Reybard's trocar, and only four times by aspiration. In the six fatal cases, the operation was performed three times with the trocar and three times with aspiration. Aspiration, therefore, is not directly responsible, since most of the accidents have supervened apart from it.

If these accidents have nothing to do with the operative procedure, to what, then, are they due? In six cases in which operation was followed by death, five were cases of complicated pleurisy. In the first (Gombault), the opposite lung was partly fibrous and bound down by old pleuritic adhesions; in the second (Girard), the patient was suffering from acute rheumatism with double pleurisy; in the third (Béhier), tubercular broncho-pneumonia was also present on the right side; in the fourth (Dumontpallier), the patient had bronchitis and adhesions of the left lung, in addition to right pleurisy; in the fifth (Bouveret), the asphyxiated patient had old tubercular pleurisy, with adhesions and 7 pints of fluid.

Let us analyze the cases in which thoracentesis has been followed by oedema of the lung (Terrillon's thesis) and albuminous expectoration. Here also complications were present in most cases. Thus, in Cases 13 and 17 (Lasègue), the patients had aortic and mitral lesions, oedema of the lower limbs, etc.; in Case 3 (Bucquoy), hypertrophy of the heart, with mitral and aortic murmurs; in Case 15 (Lancereaux), the patient was four months pregnant, and subject to chronic bronchitis, with night-sweats; in Case 7 (Marotte), the patient was suffering from pleurisy as well as from pulmonary tuberculosis.

I find that the accidents due to thoracentesis, when pleurisy was not associated with any complication, have been caused by withdrawing **rapidly and at one sitting too large a quantity of fluid**—10 pints in Case 6 (Marotte), 7 pints (Worms), 5 pints in Case 18 (Faussillon), and 4 pints in other cases.

The benign, grave, or fatal cases of pulmonary oedema and albuminous expectoration have, therefore, always been associated either with complications or with the immediate withdrawal of too large a quantity of fluid, and most often with these two causes combined. These accidents, then, are not directly referable to exaggerated lowering of the pleural tension produced by aspiration, since the same accidents supervene with Reybard's trocar when the tension is the **same inside and outside** the chest.

When, however, aspiration is **badly performed**, trouble follows; but it is not a question of the **quality** of the vacuum, but of the **quantity**. Accidents do not occur because 2 pints of liquid have been withdrawn with a proper vacuum, but because 4 or 5 pints have been drawn **at once** with an incomplete vacuum or **with none at all**. It is not aspiration which is at fault, **but the way in which it is done**. A patient has had 6 or 7 pints



of fluid in his chest for five or six weeks. The heart and the pulmonary vessels are displaced, the lung is flattened, the circulation is hampered. These organs are suddenly deprived of 6 pints of fluid which has for a long time impeded their functions, and the blood at once rushes into the pulmonary vessels, the air into the alveoli, and yet we are astonished that accidents supervene. What astonishes me is that they do not occur more often. It is not aspiration, nor a too perfect vacuum, which must be blamed for these accidents. It is aspiration prolonged **without measure**, or the employment of **too large a trocar**. It is, in short, the ill-advised method, which, without holding to principles that should never be departed from, allows a large effusion to be drawn off too rapidly at one sitting. I repeat again that the secret of preventing accidents consists in using a No. 2 needle and in limiting the quantity of fluid withdrawn at a sitting to 2 pints.

I have always carried out this plan, and in the 180 cases which served as a basis for my discussion at the Académie de Médecine, we find that the patients suffered no mishap. Pulmonary congestion, albuminous expectoration, and threatening asphyxia were never seen.

We may, then, draw the general conclusion that thoracentesis, properly done, never causes trouble; while thoracentesis, if imprudently rejected or postponed, exposes every patient suffering from a large effusion to the risk of sudden death.

**2. Early or Late Syncope.**—In some cases patients die from syncope one or two days after operation. Analysis of these cases proves that the accidents resulted from various causes which were in every case independent of thoracentesis, and included clots in the heart or the pulmonary vein, phlebitis, and thrombosis, and gangrene of the pleura (Besnier).

**3. Purulent Changes.**—The accusation that serous fluid may become purulent is certainly one of the gravest accusations which has been brought against thoracentesis. This accusation has been brought up during the last discussion at the Académie, but it has no foundation. If we puncture in the early stage of pleurisy, and do not examine the fluid carefully, we may conclude the effusion is benign, because the fluid is clear and citron-coloured; then, if a fresh puncture is made later, thoracentesis is wrongly accused of having caused infection, because we forget that we have punctured at **two different stages of the disease**, and that thoracentesis has nothing to do with this change.

A similar remark holds good with regard to the **pathogenic agents** in the fluid of sero-fibrinous pleurisy. Fluid containing the pneumococcus or the staphylococcus may, on the first puncture, be sero-fibrinous, and may become purulent from the nature of the disease, and not from thoracentesis.

The question of this purulent change in pleuritic effusion following

thoracentesis appears clear, in my opinion. Aseptic thoracentesis cannot cause sero-fibrinous fluid to become purulent, and if the change has occurred, it is not the operation which should be blamed, but the **operator**.

## II. HOW TO TELL IF AN ACUTE SERO-FIBRINOUS PLEURISY IS OR IS NOT TUBERCULAR—CYTO-DIAGNOSIS —SERO-DIAGNOSIS.

**Discussion.**—As we have studied the symptoms, course, and treatment of acute pleurisy, let us now consider the tubercular nature of these cases. We are often consulted by patients who have been attacked while in good health by acute sero-fibrinous pleurisy. The case appears innocent, and it has the characters of primary pleurisy. The conditions under which it has developed appear to stamp it as pleurisy *a frigore*, and yet, learning from experience, we doubt the benignity of the disease, and ask ourselves whether the condition is not really tubercular—a fact which greatly modifies the prognosis.

How can we solve this problem, which sometimes is very difficult? Are we in a position to say that the case is tuberculous or not? We must now discuss this question.\*

When I commenced my medical studies, acute pleurisies were classed as primary and secondary. The former class was said to result from chill, whence the name “pleurisy *a frigore*.” It was called idiopathic, meaning by this that it was not secondary to any other pathological process. It was also called “frank pleurisy,” which excluded the idea of any original taint. This primary form served as the general descriptive type of acute pleurisy.

In opposition to this primary form, writers described secondary pleurisies which supervened in the course of some other disease, such as pneumonia, rheumatism, Bright’s disease, etc., or were consecutive to neighbouring lesions of the thoracic and abdominal organs. In this incongruous group pleurisy due to tuberculosis occupied the chief place, but primary pleuro-tuberculosis was not yet well known, and tubercular pleurisy was chiefly considered as a complication of phthisis.

The question of tubercular pleurisy has been gradually elucidated, and it is evident that all cases do not resemble one another. The first category includes cases supervening in phthisis when lesions exist in the lung and bacilli are present in the sputum. Pleurisy appearing under these conditions is almost certainly tuberculous; but this fact is of minor importance in the present discussion, since we know in advance that the patient is tubercular.

\* Dieulafoy, *Clinique Médicale de l’Hôtel-Dieu*, 1905, leçons 1 et 2.



In the second category let us place cases **supposed** to be tubercular. The lung appears free, it is true, yet the patient is suspected of tuberculosis. One is of tubercular stock; another has previously had obstinate bronchitis, hæmoptysis, fistula in ano, or so-called rheumatism, which is only tubercular pseudo-rheumatism (Poncet). Some patients in their infancy have had suppurating glands in the neck, which have left scars; coxalgia, which has left slight lameness; or adenoid hypertrophy, with larval tuberculosis of the tonsils.\*

In short, the cases belonging to this category have been preceded at a more or less distant date by previous tubercular lesions. Sometimes they follow a prodromal phase that is of indefinite duration, and is characterized by loss of strength and wasting. Clinically, I repeat, these cases are suspicious of tuberculosis.

Quite different are the cases of tubercular pleurisy which now require notice. We find no trace of tuberculosis, either in the past or in the present. The patient has been attacked with pleurisy while in good health, either without appreciable cause or following a chill, just as a patient is seized with tonsillitis or with coryza. The onset and course of the disease, the appearance of effusion, the results of thoracentesis (if it has been performed), and convalescence itself, recall the picture of so-called pleurisy *a frigore*. Appearances, however, are often deceptive, for we shall find that the man who recovers from pleurisy suffers some years later from more or less advanced pulmonary tuberculosis. The pleurisy from which he recovered was the initial manifestation of the tubercular infection which has developed later. Another patient who has been completely cured of pleurisy dies six months or a year later from tubercular meningitis. These cases, moreover, are not isolated. On the contrary, they are frequent, and have thrown doubt upon the existence of pleurisy *a frigore*.

Landouzy in 1884 deprived pleurisy *a frigore* of its ancient privileges. "Every pleurisy," says he, "which does not stand the test is tubercular, although the patient may be robust." Kelsch and Vaillard sustained the accusation, and the course of events fitted in so well in this respect that pleurisy *a frigore* has lost part of its importance.

Landouzy's opinion, however, aroused sharp protests, and facts in opposition to his opinion were sought for. Cases of acute pleurisy *a frigore* were published. It was said they had no connection with tuberculosis, since they recovered without sequelæ. Partisans arranged themselves into camps, and pleurisy *a frigore* continued to be admitted by many physicians.

We have to see on which side the truth lies. Let us carefully examine

\* Dieulafoy, "Tuberculose Larvée des Trois Amygdales" (*Académie de Médecine*, séance du 30 Avril, 1895; et "Manuel de Pathologie Interne," t. ii., p. 197).

the evidence, for it is important in prognosis to know whether pleurisy is tubercular or not.

Let us first consider the cases of sudden death in patients suffering from pleurisy *a frigore*, and see whether post-mortem examination discloses tubercular lesions.

The following case (Landouzy) might be quoted as a type of pleurisy *a frigore*.

A healthy baker went out while in a profuse sweat to buy some wine. Whilst walking he felt that he had caught a cold, but continued his work. Pain in the side, respiratory distress, and fever appeared. The pleurisy ran its course. Some months later he died suddenly, while under Landouzy's care. Post mortem, much serous fluid was found in the right pleura, and the right lung showed small foci of latent tuberculosis. This case, although apparently one of pleurisy *a frigore*, was really tubercular.

Kelsch and Vaillard have published the following cases:

A healthy trooper was taken ill with acute pleurisy. He died suddenly while talking with his comrades, just as he was going to the Val-de-Grâce Hospital. At the autopsy a large effusion was found in the right pleura, which was studded with tubercular granulations.

Another soldier, who was suffering from right pleurisy, was reading by his bed, when he suddenly fainted and died. A large right-sided effusion was found post mortem; the pleura was studded with tubercular nodules. These cases of serous pleurisy, say Kelsch and Vaillard, "supervening in strong men without any tubercular taint and having only the usual signs of pleurisy, would certainly have been considered as inflammatory if sudden death had not revealed their true nature."

In days gone by, at the Saint-Antoine Hospital, I saw the two following cases, which have been published by Binet and Legrand:

A man in good health was taken ill with right pleurisy, which he attributed to a chill. For several days we followed the spread of the effusion, which finally reached about 4 pints. Although dyspnoea was absent, we decided on thoracentesis about 11 a.m. The patient protested, and the operation was postponed till evening; but an hour later the man got up, and died from syncope before help could reach him. At the autopsy, as I wished to know the exact amount of the effusion, I performed thoracentesis on the corpse, and drew off 4 pints of yellowish fluid. Latent tuberculosis had been present; at the apex of the lung we found a cretaceous tubercle.

The second of my cases refers to a robust man who had no tubercular history. His trade exposed him to sharp changes of temperature, and especially to local chills: he carried blocks of ice on his back. The pleura and the lungs were therefore much exposed to cold. He came under my care for pleurisy. At the right side we found an effusion, estimated at about 6 pints. Two pints were drawn off by puncture on three successive days. He felt very well, but the liquid continued to reform. Some days later he got up, but had gone only a few steps when he fell down, his face cyanosed, his lips blue, and died in a few seconds. At the post-mortem, as I wished to know exactly the quantity of effusion causing his sudden death, I performed thoracentesis on the body, and drew off 71 ounces of fluid. I then discovered that the pleurisy, which appeared to be a *frigore* (contact with the ice-basket), was really tubercular; I found tuberculosis of the pleura and a tubercular nodule in the lung.

A person may apparently regain his health after acute pleurisy, but some months later he is carried off by acute phthisis or by meningitis, show-



ing the tubercular nature of the pleurisy. In 1884 I saw the following case :

A mechanic, aged twenty-eight, came under my care for pleurisy a frigore of three weeks' standing. There was nothing to make me think of tuberculosis ; it was a typical case of pleurisy. On the left side I found a large effusion, which I estimated at 4 pints. I began by drawing off 2 pints of sero-fibrinous fluid. A few days later I drew off 2 pints more. Everything went well—the patient regained his health ; but six months afterwards he came under my care with signs of meningitis, and died in a few days. At the post-mortem we found that the pleurisy was cured, only a few adhesions remaining, but tubercular meningitis and some tubercular granulations in the lung were present.

In his anatomical and experimental researches on tuberculosis of the pleura, Péron arrives at the following conclusions : “ Acute pleurisy, called ‘frank,’ is usually tubercular in nature, and is in many cases due to discrete infection of the pleura.”

Clinical medicine teaches us that many patients who are apparently suffering from primary pleurisy are really affected with tuberculosis, because, although they may be cured of pleurisy, the tubercular infection invades the lung, meninges, peritoneum, or other parts of the body some months or years later. Clinical medicine, however, also teaches us that there are other patients in whom acute pleurisy, with large effusion, may recover without leaving any tubercular taint ; the patient, after having recovered his health, lives for ten or twenty years, but yet neither he nor his descendants show any signs of tubercular infection.

Many statistics may be consulted on this subject. Fiedler, in Germany, reports 92 cases of sero-fibrinous pleurisy which he punctured : of this number, 17 died in hospital from tuberculosis, 8 died after leaving, 66 left the hospital either tubercular or suspected of tuberculosis, and 21 were in good health at least one or two years later. The statistics of Barrs and Bowditch in England, and those of Mayor and Ricochon in France, give results which are not in agreement, but still the tubercular element occupies the largest part. For some years I questioned my colleagues (Brouardel, Grancher, Vergely, Lépine, etc.), in order to learn their opinion and the result of their observations. Vergely sent me four reports, referring to patients with pleurisy who were punctured fifteen, twenty, and twenty-two years previously ; they remained in good health, as did their children. Lépine has furnished me with six cases, seen a great number of years ago, and never followed by tubercular mischief.

Since my first work on thoracentesis by aspiration, which dates back more than thirty years, I have punctured a great many cases. I have lost sight of nearly all the hospital patients, but have been able to follow up many of those treated in private, and I can quote cases of persons who suffered from acute pleurisy and recovered without any signs of tuberculosis.

I owe the following notes to Dr. Lamarre (of Saint-Germain):

The forage-store at Saint-Germain was formerly situated in the Rue d'Alsace, nearly a mile from the quarters. The square of the Château is about half-way on the road from the forage-magazine to the barracks. When Lamarre was appointed Assistant Physician to the Saint-Germain Hospital, the outer walls of the trenches of the castle had just been razed to the height of 3 feet. This wall became a natural lounge for the soldiers who carried forage on their backs. They used to come from the store, and, while in a sweat, they leant their backs against the wall, so as to rest their load on the top. Suddenly, however, an epidemic of acute pleurisy *a frigore* broke out in the regiment, while no cases were seen among the civil population. The mischief always affected the right side. The men made good recoveries, with or without aspiration, according to circumstances.

They were clearly, says Lamarre, cases of pleurisy *a frigore*. The angle of the Château square where the soldiers rested was sheltered except from the cold north-east wind, which lashed the right side of their chests. At the request of Lamarre and of the Surgeon-Major of the regiment, the Colonel forbade the men to stop at this dangerous spot, and no more cases of pleurisy occurred.

The regiments on guard duty, however, change every six months, and fresh cases of pleurisy, due to the same cause, occurred in the regiment which had just marched in. It was again necessary to prohibit the men on fatigue duty from stopping and resting against the wall. This experiment (for this fact has the value of an experiment) occurred several times, with the same regularity.

By confining ourselves to clinical facts we see, then, that the cure of acute pleurisy, without any residue of tuberculosis, is not so rare. These facts furnish an argument for those who defend acute pleurisy *a frigore*: "You see clearly," they say, "that these cases of pleurisy are not tubercular." Moreover, they add: "Since pleurisy *a frigore* exists in animals, why should it not exist in man?" Trasbot, in an interesting paper, has shown that pleurisy *a frigore* is common in horses, dogs, and sheep, and may have nothing to do with tuberculosis. In support of this opinion, the following facts have been quoted: In 1871 a line regiment of Cuirassiers who had just been supplied with clipped horses were picketed in the open, often without blankets, and in a few weeks thirty cases of pleurisy, nearly all fatal, occurred among the animals. This fact was so striking that the military authorities at once prohibited the purchase of all clipped horses.

Duvieusart saw 100 cases of pleurisy, with 60 deaths, in a flock of 400 sheep which had just been shorn during a very cold February. These animals were not tubercular, and the pleuritic fluid, injected into guinea-pigs, never caused tuberculosis.

Rousseau saw several healthy dogs attacked by pleurisy, after having in the depth of winter followed a stag in a pond for almost an hour. In all these cases, adds Trasbot, there was no question of tuberculosis.

I quote Trasbot word for word: "The three domestic species—horse, dog, and sheep—in which pleurisy is most often met with are precisely those in which tuberculosis is most rare. . . . Thus, the facts drawn from extensive clinical observation in different species of animals are in



formal contradiction to the idea that pleurisy may in these species be a form of tuberculosis. This proposition, which is derived from clinical data, is also absolutely confirmed by experiment. The injection of the fluid from sero-fibrinous pleurisy in horses or in dogs has never caused tuberculosis in guinea-pigs or rabbits."

It appears undeniable, then, that simple pleurisy *a frigore* is frequent in animals. Veterinary medicine shows the power of chills in causing pleurisy among animals. But let us limit our study to human medicine, and state the question afresh: Does there exist in man true pleurisy *a frigore* which is not tubercular? and if it exists, by what means can it be distinguished from the tubercular form?

**Laboratory Researches.**—To answer this question, an appeal has been made to the multiple resources of the laboratory.

**Inoculations.**—It was thought that the introduction of pleuritic fluid into the peritoneum of guinea-pigs would furnish important information, as the inoculation would transmit experimental tuberculosis to the animal if the fluid were tubercular. The method of inoculation certainly gives valuable information, but in a fairly large number of cases it leaves the tubercular or non-tubercular nature of pleurisy in doubt; its value is absolute when the result is positive, but a negative result does not prove that pleurisy may not be tubercular. All observers are agreed that tubercular pleurisy may give negative results after inoculation of pleural fluid. A negative result is easily understood, because the fluid may have very little virulence, and the bacilli may be so disseminated that the few organisms introduced into the peritoneum are rapidly destroyed.

Injections of tuberculin furnish information of undeniable value. According to the official figures from the clinics in Prussia, patients suffering from apparently simple but really tubercular pleurisy are nearly as sensitive to injections of tuberculin as frankly tubercular patients are. Injection with tuberculin, even if carried out according to Grasset's rules, is not always exempt from harm.

**Cultures.**—The application of culture methods to the search for the tubercle bacillus in effusions has only recently been successful. If we are to obtain positive results, a culture medium that is extremely favourable to Koch's bacilli should be employed. Glycerinated blood-agar, as used by Bezançon and Griffon, is the best medium.

The mixture of aseptic rabbit's blood with agar furnishes soil on which the microbes develop in abundance, although they will not grow on the usual media. If glycerine is previously added to the agar-agar, glycerinated blood-agar is obtained, on the surface of which it is only necessary to place the suspected liquid, whether it be pus, cerebro-spinal fluid, removed by puncture, pleuritic fluid, etc.

We may use, in place of ordinary culture-tubes, Erlenmeyer's flasks, at the bottom of which a layer of the mixture of the blood and glycerine agar is allowed to settle.

The tube is carefully sealed and placed in the oven at 37° C. After three or four weeks, colonies, which increase in number and have the following characters, are seen to appear. At first the colonies are smaller than a pin's head, but they soon become larger, growing in prominent mulberry-shaped masses of a chocolate colour. Under the microscope, preparations show bacilli, isolated or in clumps, which are usually of a twisted form. The number visible to the naked eye is proportionate to the amount of fluid sown, and especially to the richness of this fluid in bacilli.

The results obtained by this procedure are not constant, but it is an excellent control measure, often used in our clinic.

**Sero-Diagnosis.**—What may we expect from sero-diagnosis? Let me first remind the reader that it is the sero-diagnosis of tuberculosis. Speaking generally, sero-diagnosis supposes two factors; whether the case be one of typhoid, pneumonic, or tubercular infection we need: on the one hand, a homogeneous culture in a fluid medium, in which the microbes are separated one from another; and, on the other hand, serum from the infected patient, which, on addition to the culture, produces agglutination of the microbes.

In 1898 Arloing tried to find a sero-diagnosis for tuberculosis, comparable with Widal's method in typhoid fever. The problem was solved when he obtained homogeneous cultures of Koch's bacillus in a fluid medium. In order to obtain the conditions most favourable to success, we must employ a culture of tubercle bacilli twelve days old. A small quantity of the culture is placed in a very small tube. This culture is mixed with blood-serum from the finger of a patient who is thought to be suffering from some tubercular lesion. The mixture is so made that it represents one part of serum to five parts of culture, or one part of serum to ten parts of culture, etc. The tube is then shaken, to favour mixing, and the effect is watched. If the result is positive, agglutination occurs after an interval of one to five hours. The upper layers of the mixture become clear, while flakes accumulate at the lower part of the tube and give to this layer a muddy aspect, contrasting with the limpid nature of the upper layers. Microscopic examination will confirm the result of the agglutination, and the Koch's bacilli appear in masses instead of being isolated.

Sero-diagnosis is applicable in tubercular pleurisy. Courmont found that a positive reaction may be obtained by mixing cultures of Koch's bacilli either with blood-serum or with pleuritic fluid in dilutions of 1 to 20, 1 to 10, or 1 to 5.

The agglutinating power of the blood is not always equal to that of the



serum ; it may be more or less marked, and may exist while that of the serum is absent, or *vice versa*.

Courmont in the following table has summarized the results of positive and negative reactions with pleuritic fluid and with blood-serum :

|                                     |                                                |                                                                  |
|-------------------------------------|------------------------------------------------|------------------------------------------------------------------|
| In 31 cases with<br>pleuritic fluid | Positive reactions, 23 cases<br>(74 per cent.) | 1 case, 1 in 20<br>6 cases, 1 in 10<br>16 cases, 1 in 5          |
|                                     | Negative reaction                              | 8 cases, 1 in 5 (26 per cent.).                                  |
| In 22 cases with<br>blood-serum     | Positive reactions, 18 cases<br>(81 per cent.) | 3 cases, 1 in 20<br>9 cases, 1 in 15, 1 in 10<br>6 cases, 1 in 5 |
|                                     | Negative reactions                             | 4 cases (19 per cent.)                                           |

The results obtained at the Hôtel-Dieu confirm the value of sero-diagnosis in tubercular pleurisy ; we must, nevertheless, remember that the procedure is sometimes at fault.

**Cyto-Diagnosis.**—Cyto-diagnosis is based on the examination of the cellular elements found in the fluid of pleurisy and of serous effusions in general. A few words of explanation are necessary. Certain cells respond by an offensive and defensive reaction against the attack of the pathogenic agents, but the same cells do not always react to different provoking agents. The polynuclear neutrophiles, or microphages (Metchnikoff), engulf the streptococcus or the pneumococcus. The large mononuclear cells, or macrophages, have a more powerful action ; they absorb, and sometimes succeed in destroying, the tubercle bacillus ; they readily digest large cells, such as red corpuscles and polynuclears. It was therefore natural to suppose that the presence of a particular cell in the fluid would indicate the nature of the pathogenic agent. From this idea cyto-diagnosis has arisen.

In 1900, under the name of "Cyto-diagnosis," Widal and Ravaut first published their valuable work, of which the chief features are as follows :

In a case of pleurisy we desire to know the cellular elements in the fluid. For this purpose we draw off, with a sterile syringe, some pleuritic fluid, and centrifugalize it. A sediment forms at the bottom of the tube ; we pour off the fluid, so as to leave in the tube only a little liquid, which forms a cloudy emulsion with the cellular débris. A drop of this emulsion is placed on a slide and stained with thionin, eosin-hæmatin, or with Erlich's tri-acid mixture.

On microscopical examination, we see cells of various kinds—red corpuscles, polynuclear leucocytes, large mononuclear cells, lymphocytes, isolated endothelial cells, and endothelial plaques. These elements, however, do not exist indifferently in all cases. A slide does not at the same time show numbers of polynuclears, lymphocytes, and endothelial cells.

Except for the red corpuscles, which exist in most pleuritic fluids, one specimen will contain chiefly lymphocytes, while the polynuclear and the endothelial cells are absent, or in very small numbers. Another specimen of fluid contains almost nothing but polynuclear cells, while the lymphocytes and endothelial cells are absent, or very few in number. Lastly, a third specimen contains chiefly endothelial cells and plaques—the lymphocytes and polynuclear cells are absent, or few in number.

The preponderance of one or other variety of cell in the pleuritic fluid constitutes the **cellular formula** for this fluid, and leads to the cyto-diagnosis. From this point of view, Widal and Ravaut have described three kinds of pleurisy, each with its own cyto-diagnosis.

The **first** variety comprises effusions in cardiac disease, Bright's disease, and cancer, as well as those due to irritation or to compression by neighbouring organs. No infective agents are present, and consequently we find no phagocytosis, but only the processes of transudation and desquamation; the condition is a kind of congestive oedema. To use an old expression, it is here less a case of **exudate** than of **transudate**; it is by transudation that the liquid carries away the endothelial cells from the serosa into the fluid. Accordingly, these so-called mechanical effusions have a special formula; the fluid, as a rule, contains neither lymphocytes nor polynuclear cells (at any rate, in the first stage). Endothelial cells from the serosa are almost exclusively met with.

These cells are very large in comparison with the size of the red corpuscles and the leucocytes. They may be isolated or agglomerated, bilobed, trilobed, or fused in large endothelial plaques with polycyclical edges. After staining with eosin-hæmatin, the nucleus is seen to be much darker than the protoplasm. The outline of the cells is almost circular; in the endothelial plaques, however, the outline of the cells disappears at the points where the protoplasm is fused. These plaques, which vary in size and in number, are characteristic of mechanical effusions. Widal and Ravaut met with them in twelve cases. The post-mortem examination of three patients with this variety of pleurisy, and the negative results of the intraperitoneal inoculations in guinea-pigs with pleuritic fluid from seven similar cases prove that tuberculosis is not in evidence. These endothelial masses are not only characteristic of mechanical pleurisy, but their presence in the fluid excludes the hypothesis of tuberculosis. They are not found in the fluid in recent cases of tubercular pleurisy, no doubt, "because the tubercular neomembrane prevents the patchy desquamation of the endothelium." Similar observations have been made in my wards: the fluid in every case of tubercular pleurisy, though very rich in lymphocytes, did not contain endothelial plaques. A similar assertion may be made, especially with regard to the first stages of mechanical effusions,



but later the lymphocytes may abound. The presence of the plaques suffices, nevertheless, to specify the diagnosis.

In the **second variety** of pleurisy the fluid has quite a different cellular formula. This variety includes acute infective pleurisies. The pathogenic agents, which include the streptococcus, the pneumococcus, the *Bacillus typhosus*, etc., after causing cellular reactions of attack and defence, may no longer be present, but the phagocytes or polynuclear leucocytes exist in abundance. We also meet with large mononuclear cells, which may be large leucocytes, or may be derived from the serous membrane; while we may also recognize the presence of endothelial cells, which are isolated or have remained absolutely normal.

With regard to these cases, Widal and Ravaut give the following information: In three out of seven cases of pleurisy in typhoid fever, the relative abundance of large polynuclear leucocytes characterized the formula of the effusion. In a case of sero-fibrinous streptococcal pleurisy, there were only neutrophile polynuclears, with deformed nuclei. In sero-fibrinous pneumococcal pleurisy, the formula gives rather the impression of attack and defence. This formula is characterized by the presence of red corpuscles and of a few lymphocytes, but especially by the abundance of the polynuclears and by the presence of a greater or less number of large mononuclear cells, some of which are really macrophages and engulf the polynuclears. It is quite exceptional to meet with two or three endothelial cells fused together.

Some odourless fluid was withdrawn by exploratory puncture from one of my patients who had pleurisy on the right side. The fluid was examined by Apert, and contained only polynuclears; neither lymphocytes nor endothelial plaques were met with. The absence of lymphocytes excluded acute tubercular pleurisy; the absence of endothelial plaques put mechanical pleurisy out of court. A few days later turbid foul-smelling liquid, like dirty water, was withdrawn. The fluid, which was rich in polynuclears at the first puncture, now contained only a few cells in the shape of granular masses, which did not stain well; they were dead leucocytes in process of granulo-fatty degeneration. Aerobic and anaerobic cultures of the liquid revealed a varied microbic flora. In aerobic cultures the *Staphylococcus albus* appeared. In anaerobic cultures, colonies in the form of whitish points, composed of a small micrococcus *en masse*, appeared. The leucocytic formula of this foetid pleurisy was the same as in other infectious varieties. In short, polynuclear and mononuclear elements characterize acute infective pleurisies; lymphocytes, when met with, are less numerous, and it is exceptional to find endothelial masses, with two or three nuclei. Fig. 14 represents the cellular formula. We see several polynuclear leucocytes and one large uninuclear cell.

We now come to the **third** variety, which is the most important of all, and corresponds best to acute pleurisy *a frigore*. On microscopic examination of the pleuritic fluid, we see that the cellular formula is characterized "by the almost exclusive presence of **lymphocytes**, which are confluent and mixed with a relatively large number of red corpuscles. At times we perceive here and there mononuclear cells, as well as lymphocytes, which at first sight seem to be the only leucocytes in the specimen." The polynuclears, when met with, are not numerous; they are perhaps the result of secondary infection. The endothelial cells must be very rare, because in seventeen cases Widal and Ravaut never met with them.

I have examined the cellular formula of these cases, employing preparations made with fluid from seven patients in the Saint-Christophe and

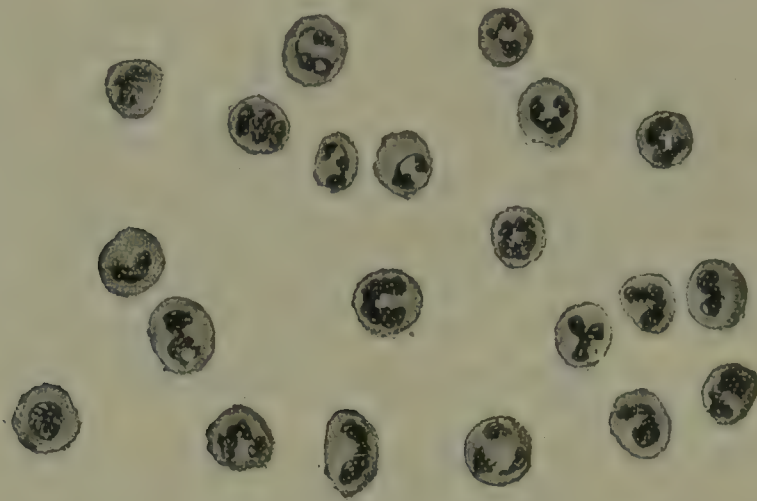


FIG. 14.—POLYNUCLEAR LEUCOCYTES.

Sainte-Jeanne wards. Numerous lymphocytes and some red corpuscles are met with; neither polynuclears nor endothelial plaques are seen. This description corresponds to that given by Widal and Ravaut.

This cellular formula, or pleural lymphocytosis with red corpuscles, indicates that the case is tubercular. Histological examination of the pleura and inoculation of the peritoneum in guinea-pigs with the pleuritic fluid confirm their tubercular nature. The following case was related to me by Widal, and is a striking example:

A youth was taken ill with the symptoms of pleurisy *a frigore*—*i.e.*, repeated rigors, stitch in the right side, and cough, but no expectoration. On the ninth day he was admitted under Widal, with the signs of abundant effusion on the right side. Thoracentesis was performed, and 4 pints of yellow liquid were withdrawn. The liquid showed a typical lymphocytic formula, indicating pleuro-tuberculosis. On the following day the temperature varied between 102° and 104° F. Four days after the puncture the patient suddenly sat up, choked, and died in a few seconds.

Post mortem: The left lung looked like an infarct engorged with blood; this condition seemed to depend upon an extensive pulmonary embolism. Tubercles were not



present in either lung. The pleural cavity contained 3 pints of sero-fibrinous fluid. The pleura was much thickened. No tubercles were visible to the naked eye, but histological sections showed everywhere tubercular tissue. Many giant cells were seen in the fibrous tissue of the inflamed serous membrane. Fig. 15 represents these lesions. In this section of the thickened pleura tissue is infiltrated with numerous cells. We see a giant cell, containing numerous nuclei, arranged like a crown at the periphery of the cell.

The entire pleura was really a tubercular membrane studded with giant cells, but the lung was unaffected. The condition was primary tuberculosis of the pleura. The fluid produced tuberculosis in guinea-pigs.

This case sums up the whole question ; it would formerly have been considered as a case of simple pleurisy *a frigore*, when it was really tubercular. The cytoscopic examination of the fluid was undertaken, but lymphocytes alone were found ; the diagnosis of tubercular pleurisy was verified by the

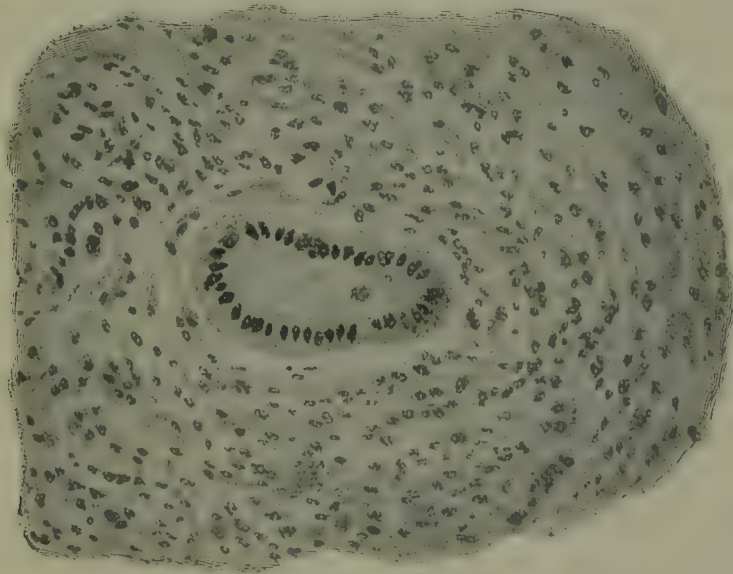


FIG. 15.—SECTION OF PLEURA.

histological examination of the pleura and by the results of inoculation of guinea-pigs with the pleuritic fluid.

We have, therefore, several methods of ascertaining the tubercular nature of acute pleurisy, but I much prefer cyto-diagnosis, because it is simple and expeditious.

In acute pleurisy (the patient being in good health) abundance of lymphocytes and absence of endothelial plaques from the fluid point to tubercular mischief. Cytoscopic examination of the fluid is as necessary as bacteriological analysis of the sputum in a doubtful case of pulmonary tuberculosis. In every case of pleurisy cyto-diagnosis is necessary. Every report in which cyto-diagnosis is wanting is incomplete. In my wards this fact is never neglected.

We must not be satisfied even though the diagnosis is clinically evident, because surprises may happen. Pleurisy, considered to be tubercular

because it has supervened in the course of phthisis, may not be tubercular ; pleurisy, considered influenzal because it has occurred in the course of influenza, may be tubercular ; the influenzal infection has here favoured the development of pleurisy. Cases of traumatic pleurisy, which might have been referred to the trauma alone, are really tubercular, the injury having awakened the latent germs. I have so far considered tubercular pleurisy, which is acute or of recent date. The leucocytic formula of the fluid may be quite different in a case of tubercular hydropneumothorax, which results from gross tubercular lesions in the lung. Under such conditions the lymphocytosis is not the chief point : " old deformed polynuclears with much divided nuclei are seen, as well as cells with vesicles in their protoplasm, and sometimes also amorphous masses, which appear to be derived from the endothelium." The diagnosis in this variety depends chiefly on clinical research.

Nattan-Larrier has devised an ingenious method, which consists in the injection of  $\frac{1}{2}$  c.c. of pleuritic fluid into the mamma of a female guinea-pig which is suckling. The mamma acts as a living culture medium. The bacilli begin to appear in the milk after five to ten days. When we wish to examine the milk, the animal is put on its back, and the mammary gland is squeezed with the fingers of the left hand ; a drop of milk exudes from the nipple. The drop is spread out over a slide in a thin layer. The films are fixed with alcohol, stained with Ziehl's fuchsin, and decolourized with 30 per cent. nitric acid ; the ground substance is stained with Kühn's blue.

We see even as early as the fifth day that some bacilli are engulfed by the macrophages, or are isolated between the leucocytes. A few days later the bacilli are more numerous, and their recognition is more easy. This method has been extended to other fluids (peritoneal effusion, cerebro-spinal fluid, etc.). It has been used in my wards at the Hôtel-Dieu, and I have proved the correctness of the results. By this method the daily search for bacilli allows us to follow the course of the inoculation, and reveals tuberculosis as soon as it begins to develop in the gland. We also see that the glands become larger in a few days and that the inguinal glands swell ; the mamma is therefore the starting-point of widespread lesions.

Let us return to the origin of our discussion. The original question was : Are we in a position to know whether acute sero-fibrinous pleurisy is or is not tubercular ? The answer is an affirmative one.

Henceforth it will be possible for us to estimate correctly the so-called " essential " pleurisy. We shall see from the first that most cases which answer to the description of so-called frank pleurisy a frigore are really tubercular ; cyto-diagnosis stamps them, and lymphocytosis unmasks their origin.

We shall see, further, that there are infectious agents capable of causing acute pleurisies which have nothing to do with tuberculosis. An individual suffers from sero-fibrinous pleurisy with effusion, as he might from pneu-



monia; the pneumococcus is the provoking agent. Another person has acute pleurisy with effusion; here the streptococcus is the pathogenic agent. In a third person, who also has acute pleurisy with effusion, we find foetid or non-foetid infection of the pleura by aerobic, or by anaerobic, germs, etc. This group of acute pleurisies, which I might further enlarge, has nothing to do with tuberculosis. Bacteriology has classed them according to their pathogenic agents; cyto-diagnosis includes them in the same cellular formula; they are cases of pleurisy with polynuclear and mononuclear cells.

The pathogenic diagnosis of pleurisies which were formerly indefinite is now so clearly elucidated that we may ask what place can be assigned in medicine to "essential" pleurisy, in which cold was considered the only cause. Ought this variety of pleurisy to surrender its place in our nosology for ever, after having occupied the chief place? Should it be definitely cut out, or should a place still be reserved for it? The future will tell us.

**Curability of Tubercular Pleurisy.**—Let us now approach another side of the question. How can we explain the fact that acute pleurisy, called a frigore, but recognized as tubercular, may recover without leaving any traces? This very curability was the chief argument against tuberculosis. Our reply is that if these cases recover it does not say that they may not be tubercular. The disease is sometimes limited to the pleura (primary pleuro-tuberculosis), the lung is free, and the rest of the organism is healthy; as the serosa is well armed for defence and the virulence is feeble, it is not surprising that pleurisy may recover without the infection of other parts.

Further, recovery is not special to tuberculosis of the pleura; other sero-membranes have the same privilege. Tuberculosis of synovial membranes, from simple arthritis to tubercular pseudo-rheumatism, is cured fairly often. Tuberculosis of the pericardium (Rendu) belongs to the same category.

Cases of recovery from tubercular peritonitis with ascites are no longer reckoned. Cases of ascites, formerly catalogued under the terms "essential," or "a frigore," are certainly cases of tubercular peritonitis, which are attenuated and readily curable. Some recover under medical treatment without the help of surgical intervention, some after puncture, with or without consecutive injections, while others are cured by laparotomy. The cure of one of these cases formerly caused much stir. On March 20, 1840, at a time when no one dared to touch the peritoneum, my uncle, Paul Dieulafoy, of Toulouse, was bold enough to inject tincture of iodine into the peritoneal cavity after removal of the fluid. This operation was successful, and since then other cases have been reported.

I have often recognized this curability in simultaneous tuberculosis of the pleura and the peritoneum.

Tubercular pleurisy is therefore perfectly curable; indeed, it may be said that it is sometimes quite benign in nature. Péron, who studied this question

from the anatomical and experimental point of view, comes to the same conclusion: "In tubercular sero-fibrinous pleurisy, which assumes the clinical bearing of acute tuberculosis, the infection is at its minimum; the reaction of the organism is considerable."

Griffon, in collaboration with Bezançon, has just verified this idea experimentally by measuring the degree of virulence of the pleuritic fluid in several patients under my care. Their researches show that the effusion in frank pleurisy which tuberculizes the guinea-pig, a most sensitive animal, is generally benign in a more resisting animal, such as the rabbit. Only one of my patients had pleuritic fluid of sufficient virulence to cause experimental tuberculosis in an inoculated rabbit. The affection was of long duration, with high fever, and this patient has come back to my wards suffering from pulmonary tuberculosis, which is secondary to the pleurisy.

The curability of tubercular pleurisy suggests some reflections. When we speak of cure in tubercular pleurisy, are not our thoughts limited to the pleura? What will happen later to the lungs after the pleurisy is cured? Is the patient destined to become tubercular? This is the whole point of the question. The answer is somewhat difficult. It is said, with good reason, that definite cure without sequelæ results when the disease is limited to the pleura and the lung is free. This statement is true, but how can we say that the lung is absolutely free in an individual who is suffering from primary pleuro-tuberculosis? Do we not know, as Potain has taught, that acute pleurisy is often accompanied by an inflammatory condition of the lung? What is the nature of the pleuro-congestion? A small focus of tuberculosis may exist in the lung without showing any symptom, and yet this latent focus may become the origin of tuberculosis. Rapidly fatal miliary tuberculosis of the lung may appear to be primary, though it is really consecutive (the autopsy shows this) to a small tubercular focus that was insidious in its growth.

The same arguments apply to tubercular pleurisy. An individual while in good health is taken ill with acute pleurisy. Cyto-diagnosis shows that the disease is tubercular, and everything points to primary pleuro-tuberculosis; the most minute examination, according to Grancher's scheme, reveals no lesions in the lung, and yet the apparently primary mischief in the pleura may well be consecutive to a small focus in the lung which has infected the pleura. We have the proof of this in the case quoted above.

Landouzy's patient while in good health was seized with apparently primary tubercular pleurisy; he died suddenly, and the autopsy revealed a small focus in the lung. My two patients who died suddenly had both been taken ill with apparently primary pleurisy. At the autopsy we found a small focus in the lung, which had given rise to infection of the pleura.

These cases prove that true primary pleuro-tuberculosis, associated with no pre-existing lesions in the lung, and secondary pleuro-tuberculosis, set up



by a small latent focus in the lung, may both assume the symptoms of so-called frank pleurisy. In many cases it is not possible to distinguish them; clinically, they may show no differences, and cyto-diagnosis includes them in the same cellular formula.

These two varieties of tubercular pleurisy, however, are not comparable as regards prognosis; one is less grave than the other. In the primary form the lesion in the pleura may recover without producing general infection of the lung, or of the other organs. If the lung is already affected, although the lesion is small, the prognosis is not so good, for we have to cure tuberculosis, both of the pleura and of the lung. The prognosis in acute cases of long duration is evidently uncertain.

**Treatment.**—I would refer the reader to the preceding section; I have, however, some remarks to add. Acute pleuro-tuberculosis is generally accompanied by much effusion. Perhaps this effusion is a mode of defence; perhaps the lung which is compressed by the fluid has less tendency to be infected from the pleura. If this hypothesis be true, it would be better not to perform thoracentesis too hastily; but yet, on the other hand, we know how dangerous it is to allow too much fluid to accumulate in the pleura: sudden death may be the consequence, whatever be the theory employed to explain it. It is therefore necessary to perform thoracentesis in good time.

There is another question which is also of importance. In the case of acute tubercular pleurisy the fluid may reform rapidly, even when it appears to have been drained away by thoracentesis. I have found that this rapid and obstinate reproduction of fluid is much less marked in acute infective pleurisies that are not tubercular. I have often in tubercular cases had occasion to draw off 4 or 5 pints of sero-fibrinous fluid by two or three successive punctures. The effusion seemed to cease for the moment, and the patient was considered cured; but yet the fluid reformed without fever, dyspnœa, or pain, and in a few days amounted to 3 pints or more. The patient must be kept under observation, even if the acute phase appear to be ended; and we must not forget that fluid may reform rapidly after puncture, and cause sudden death, if we be not forewarned.

I have just given my recommendations in the acute phase of pleurisy, but treatment does not stop there. The patient is convalescent, but the tubercular lesion lies hidden, though health is apparently regained. What will happen in this case? Will it be cured without leaving any sequelæ, or may it not rather be the first stage of tubercular infection, which will later attack the lung or the other organs? We know nothing of this, but we do know that the patient has tuberculosis, and we should place him under the best therapeutic and hygienic conditions.

An individual who is convalescent from acute pleurisy should for a long while take care of himself, even though he be considered as cured of active

disease. Tubercular infection lies in wait for him. Years must pass before he can be considered free from all risk of tuberculosis.

Special attention must be paid to hygiene. The patient should avoid all causes of over-fatigue. Nourishment should be substantial and varied; food and drink which excite the appetite should be chosen. As regards residence, preference should be given to high altitudes, and life should be passed in the open air. All kinds of exercise are permissible, provided they are never carried to excess.

As regards tonic and constitutional remedies, cod-liver oil should be given in increasing doses—*e.g.*, 2 to 4 ounces daily—if it is well tolerated; many patients will swallow a tumblerful of cod-liver oil before meals. In order to render it less disagreeable, it may be cooled by placing the glass in ice.

In patients who do not take cod-liver oil well we should recommend fatty foods, such as cream or bread and butter. Oysters, caviare, sardines in oil, tunny fish, smoked fish, and meat should form part of the diet. Raw meat and meat-juice are of benefit in cases of tuberculosis (Richet and Héricourt, Josias and Roux). Injections of cacodylate of soda should also be given.

### III. HÆMORRHAGIC PLEURISY.

**General Considerations.**—For many years while I was occupied with the histological examination of fluid from acute pleurisies, I had seen that my specimens contained some thousands of red corpuscles per cubic millimetre.

With 1,500, 2,000, and 3,000 red corpuscles per cubic millimetre the colour of the fluid was not sensibly altered; the colour only becomes rosy when the fluid contains 5,000 to 6,000 red corpuscles per cubic millimetre. I have called these pleurisies **histologically hæmorrhagic**, in order to differentiate them from true hæmorrhagic pleurisies, which are quite distinct. Fluid which is very rich in red cells may remain histologically hæmorrhagic without becoming hæmorrhagic in the true sense of the word.

In this section I shall leave out hæmorrhage into the pleura from injury, and shall only take count of hæmorrhagic pleurisy from the medical aspect.

It is customary to include various morbid conditions under the term “hæmorrhagic pleurisy.” Hæmorrhagic effusions into the pleura which are consecutive to tubercular or to cancerous lesions are the most frequent. These effusions are sometimes only symptomatic, and develop as a complication in the course of cancer, or of pleuro-pulmonary tuberculosis; at other times they attract attention from the first, and appear as the prodromata of hidden tubercular or cancerous lesions. In some cases hæmorrhagic effusions into the pleura appear independent of tuberculosis or cancer; they seem to be simple hæmatomata of the pleura. This simple hæmatoma, however, must be extremely rare, and the more I study the question the more



I believe that the hæmatoma is only a benign or curable hæmorrhagic tubercular pleurisy.

We do not see therefore one, but several kinds of hæmorrhagic pleurisy. The fluid is reddish or blackish, and contains fibrin, hæmatin, red corpuscles, and dissolved elements; the composition depends upon the nature of the pleurisy and on the abundance and the age of the fluid.

I may say in advance that the hæmorrhagic nature of the pleural fluid usually comes as a surprise; thoracentesis is performed, and the fluid is found to be hæmorrhagic. It is practically impossible to affirm before thoracentesis that pleurisy is hæmorrhagic. What are the signs and symptoms which would lead to such a diagnosis? In the great majority of cases hæmorrhagic pleurisy is just like the sero-fibrinous form; I see no distinctive signs between them: the course may in both cases be acute, sub-acute, or latent. On palpation the same modifications of the vocal fremitus; on percussion, the same character of the dullness; on auscultation, the same tubular breathing and ægophony, as well as aphonic pectoriloquy which has been given as a distinctive sign between sero-fibrinous and purulent or hæmorrhagic effusions. I have found aphonic pectoriloquy in most of my cases, and it was very clearly marked in a case of hæmorrhagic pleurisy described by Jaccoud, and hence I repeat the hæmorrhagic nature of the fluid is a surprise. We perform thoracentesis, thinking to draw off sero-fibrinous fluid from the pleura, and we are often astonished to find it hæmorrhagic.

Under some conditions hæmorrhagic pleurisy may simulate empyema; the general symptoms which lead to this error in diagnosis are due to the tubercular or to the cancerous lesions which have set up pleurisy. The patient is feeble, has an earthy colour, and shows œdema of the lower limbs and of the chest-wall; thoracentesis is performed with the idea that pus will result, but here again we are much astonished to withdraw blood-stained fluid. We make this mistake because we are too accustomed to consider œdema of the chest-wall as a sign of suppuration; it is, indeed, a valuable sign, but it is not limited to purulent effusions. It is also met with in hæmorrhagic and even in some sero-fibrinous effusions.

Hæmorrhagic pleurisy may at times be suspected beforehand—*e.g.*, when the trouble develops in a cancerous patient. Whether the cancer be primary or secondary, we may prophesy in such a case that the effusion is perhaps hæmorrhagic. I say perhaps, for effusion of cancerous origin is sero-fibrinous in at least one-third of the cases.

In short, the diagnosis of the hæmorrhagic nature of the fluid rests upon no certain sign; its existence may be suspected and reservations made as to the qualities of an effusion which shows unusual characters, but it is impossible to affirm the hæmorrhagic nature. After these few remarks it will be

evident that the study of hæmorrhagic pleurisy is surrounded by difficulties ; therefore, in order to facilitate the description, I shall divide them into four groups :

**First Group.**—These cases supervene in the course of hepatic cirrhosis and of Bright's disease, or appear as a pleural hæmorrhage in the course of scurvy and the eruptive fevers. In this group I shall also place hæmorrhage from the opening of an aortic aneurysm, or from the rupture of an atheromatous aorta. This group, then, contains the most dissimilar varieties.

**Second Group.**—This group comprises the tubercular pleurisy. Three varieties must be distinguished. In the first variety the condition forms part of an **acute** tuberculosis ; in the second variety pleurisy supervenes in the course of ordinary **chronic** tuberculosis ; in the third variety hæmorrhagic pleurisy appears as the first symptom of tuberculosis : it is the result of **local or primary tuberculosis** of the pleura.

**Third Group.**—To this category belong **cancerous** pleurisy, whether the cancer be primary or secondary.

**Fourth Group.**—Simple hæmatoma of the pleura forms the fourth group.

### First Group.

**Description.**—The most dissimilar effusions are found in this group. Does cirrhosis of the liver deserve the place assigned to it in the pathogenesis of hæmorrhagic pleurisy ? I think not. In Moutard-Martin's remarkable work two cases of hæmorrhagic pleurisy are, in my opinion, wrongly considered as dependent on cirrhosis of the liver. One of them is taken from Laënnec's famous memoir, in which the lesions of atrophic cirrhosis were first described. A patient with atrophic cirrhosis had also hæmorrhagic pleurisy on the left side. Laënnec, however, did not say that the pleurisy resulted from the cirrhosis ; I am more inclined to believe that the pleurisy was tubercular in nature, for at the autopsy "the deep layer of the pleura contained innumerable greyish tubercles." The other case which has also been considered as dependent on cirrhosis of the liver may, I think, have been due to independent lesions of the pleura ; for if we look up the details of the autopsy we shall agree that it is difficult to admit atrophic cirrhosis in a liver of "normal size, which showed remarkable friability, and broke up on pressure with the finger into a pulp."

I do not deny, of course, the hæmorrhagic form of pleurisy in the course of hepatic cirrhosis, for I have seen several cases ; but I think that it is rare as opposed to the sero-fibrinous form, which is fairly common.

I also regard hæmorrhagic pleurisy associated with Bright's disease as exceptional, though Bright's disease predisposes on the one hand to effusion, and on the other to hæmorrhage.



In pleuro-pulmonary inflammations of infectious origin (influenzal pleuro-pneumonia, typhoid fever), the fluid is sometimes hæmorrhagic.

In the hæmorrhagic forms of the eruptive fevers hæmorrhagic effusion is sometimes met with, but it is here a case of hæmorrhage into the pleura rather than that of an inflammatory condition, properly speaking.

Hæmorrhagic effusion may also result from opening of an aortic aneurysm, or from the rupture of an atheromatous aorta. Several cases have been published; the following case is given by Ribail:

A man, thirty-five years of age, suffering from palpitation, breathlessness, and angina pectoris, came into the Beaujon Hospital, under Gombault. The diagnosis of aortic aneurysm, with aortic insufficiency, was made. A month later the patient felt a sharp pain on the left side. Pleural effusion was recognized, and punctures gave issue to 12 ounces of bloody fluid on the first, 16 ounces on the second and third occasions. The patient died suddenly from angina pectoris. Post mortem, the left pleura was found covered by a clot, which was continuous with the clot in an aortic aneurysm.

### Second Group.

**Description.**—This group includes hæmorrhagic pleurisies of **tubercular nature**. I shall divide them into three varieties. The first variety is associated with acute granular tuberculosis, or with acute tubercular broncho-pneumonia. The lesions in the pleura and in the lung appear together; the general symptoms are usually very marked: fever is acute, temperature is very high, dyspnœa is severe and continuous, or sometimes paroxysmal. The estimation of the quantity of fluid is very difficult, because the signs of pleurisy are distorted by the subjacent lesions in the lung.

The dyspnœa is sometimes so violent and the quantity of fluid appears so large that thoracentesis is performed; 1 or 2 pints of hæmorrhagic fluid are withdrawn, but practically no relief follows, because the dyspnœa, like all the other symptoms, is due rather to the lung trouble than to the effusion.

The effusion, however, either from its early appearance or its abundance, sometimes appears to be the chief lesion. The patient experiences some relief after the evacuation of the fluid, and may even ask for a second or a third operation; but the severity of the general symptoms, the elevation of the temperature, the persistent or rapid reappearance of dyspnœa after evacuation of the fluid, the wasting and the signs found on auscultation, prove that the effusion is associated with acute tuberculosis of the lung and pleura. The sputum must be examined for bacilli. The prognosis is nearly always fatal in these forms.

In the second variety, hæmorrhagic pleurisy is associated with chronic phthisis. The pathogenic diagnosis is very simple; the patient presents the symptoms of pulmonary tuberculosis and of pleurisy. Fever, pain, and dyspnœa may be absent; the mischief ends after one or more punctures, because it has been merely an incident in the course of tuberculosis.

In the third variety—and I draw special attention to this point—pleurisy appears as the initial symptom of tuberculosis. It is the result of primary tuberculosis of the pleura. Tuberculosis may commence in the pleura, just as it may in the synovial membrane, testis, prostate, eye, skin, pericardium, etc.; remain localized for a lengthy period, and recover without becoming general.

As we have seen in the section on sero-fibrinous pleurisy, it often happens that pleurisy is met with in an individual who recovers, but shows signs of pulmonary tuberculosis some months or years later. In this case pleurisy, though simple in appearance, was only the result of tuberculosis, which showed itself by effusion, and then became generalized throughout the lung.

Hæmorrhagic pleurisy, therefore, may result from local or from initial tuberculosis of the pleura; and just as persons have hæmoptysis long before other signs of tuberculosis, so others have hæmorrhagic pleurisy as the first symptom, and, if I may use the expression, these people **“reject their hæmoptysis into their pleura.”** These cases may present all the signs of sero-fibrinous pleurisy, and the hæmorrhagic nature of the fluid is only recognized on puncture; thoracentesis is performed once, twice, three, or four times, the fluid is drawn off, the pleurisy cured, and the case thought to be one of simple hæmatoma of the pleura; but yet signs of pulmonary tuberculosis appear a few months later, and show the error in diagnosis.

These considerations show that the pathogenic diagnosis of this variety may be fairly easy or very difficult. It is easy if the patient has signs of acute or of chronic pulmonary tuberculosis; if the pleurisy arises during apparently good health, the diagnosis cannot be settled either by the quality or by the quantity of the fluid, or by the course of the pleurisy, which may be acute, subacute, or latent. In such a case the various methods of laboratory research given above must be employed.

In a patient under my care for diabetes and hæmorrhagic pleurisy, the lymphocytosis demonstrated the tubercular nature of the pleurisy.

This form of pleurisy, when accompanied by fever, becomes much more serious, and the gravity arises from the lesions in the lung. Nevertheless, the condition may recover perfectly after one or several punctures. I have published cases, and Lereboullet has quoted others. The patient is sometimes definitely cured, in which case it is probable that the hæmorrhagic pleurisy was the result of local tuberculosis of the pleura; at other times the patient, after recovering from pleurisy, subsequently develops tuberculosis in the lung.

**Pathological Anatomy.**—The lesions show some peculiarities. Sometimes the lesion is found at the same time in the lung, in the pleura, beneath the pleura, or in the false membranes; at other times it is limited to the



pleura or the false membranes. The walls of the vessels show coagulation necrosis, and Kelsch thinks that the hæmorrhage is due to this change. Numerous vessels are obstructed by hyaline thrombi; the vessel walls are no longer distinct, and are surrounded by fibroid networks.

The newly-formed membranes are generally stratified and rich in vessels, friable if young, thick and firm if old. These membranes are composed of granulation tissue, and of deeper layers that are made up of lymphatic cells, connective bundles, and fibrous tissue (Malassez).

### Third Group.

**Description.**—Pleurisy in the course of pleuro-pulmonary cancer is not always hæmorrhagic; the fluid is sero-fibrinous in at least one-third of the cases, and this fact must carefully be borne in mind, for it would be wrong to reject the hypothesis of cancer because the effusion was sero-fibrinous. The hæmorrhagic form alone, however, must now occupy our attention. Hæmorrhagic pleurisy in cancer may arise quite suddenly, like acute pleurisy, or have an insidious onset, so that the patient finds some difficulty in fixing the date. These different varieties are found, moreover, in sero-fibrinous, hæmorrhagic, purulent, tubercular, or cancerous pleurisy.

From the clinical point of view I shall divide hæmorrhagic pleurisy in cancer into two varieties.

In the **first variety**, pleurisy appears in an individual who has obvious cancer. We find in one patient cancer of the stomach, œsophagus, intestines, rectum, omentum, liver, kidney, bladder, prostate, testis, eye, skin, or of one of the vertebræ; in another patient we see cancer of the uterus or of the breast. Cough, thoracic pain, continuous or paroxysmal dyspnoea and currant-jelly expectoration, appear during the course of these cancers. Pleural effusion is then discovered, and thoracentesis gives vent to hæmorrhagic fluid. In such a case the pathogenic diagnosis is clear—viz., secondary cancer of the lung and of the pleura; and it may be stated that the hæmorrhagic pleurisy is of cancerous origin.

In some cases we do not witness the evolution of the cancerous lesions, but the patient shows traces of a more or less recent **scar**, resulting from an operation for epithelioma of the nose or of the lip, for cancer of the breast or of the testis, or for osteo-sarcoma. Pleurisy then appears and thoracentesis yields hæmorrhagic fluid. The lung and the pleura have evidently been attacked by **secondary cancer**.

The pathogenic diagnosis, however, is not always so simple. In the cases which constitute the **second variety**, hæmorrhagic pleurisy is not preceded by otherwise appreciable cancerous lesions. **Primary** cancer may affect the pleura and only give rise to symptoms of pleurisy, which may be acute or insidious in its onset. The pathogenic diagnosis is sometimes difficult.

If hæmorrhagic pleurisy, consecutive to mediastino-pulmonary cancer, were always accompanied by special symptoms, such as dysphagia, aphonia, œdema of the arm or of the face, and well-marked collateral circulation, which are so common in tumours of the mediastinum, and if the patient suffering from pleurisy showed supraclavicular glands, currant-jelly expectoration, and violent attacks of dyspnœa, which are seen in cancer of the lung, the pathogenic diagnosis of the pleurisy would be signally simplified; there are cases in which nothing leads us to suppose the existence of cancer of the mediastinum or of the lung. There are also cases in which cancer of the pleura is primary, or associated with early cancer of the lung, which may pass unnoticed; the pleural effusion is then the chief feature, and we find hæmorrhagic pleurisy which presents much difficulty as to its origin.

The following signs and symptoms helped me to make a diagnosis in a case of hæmorrhagic pleurisy, consecutive to primary cancer of the lung, in a man twenty-two years old:

**Pain** constitutes an important symptom; it is frequent, sometimes sharp, persistent, unlike the "stitch in the side" of common pleurisy. It may be worse at the base of the thorax, and radiate to the shoulder, the arms, and the wrists, so that patients believe themselves to be suffering from rheumatism. Acuteness and radiation of the pain are fairly frequent symptoms in pleuro-pulmonary cancer. Neuralgia of the brachial plexus was the chief symptom in one of Béhier's cases. One of Lancereaux's patients complained of "a sharp pain in the left side of the neck, and in the shoulder on the same side," and later swelling of the joints of the left arm supervened. In several of my cases I have noted pains in the joints, so that I have asked myself whether pseudo-rheumatism may not be one of the manifestations of cancer. These pains are not found in hæmorrhagic tubercular pleurisy.

**Dyspnœa** is one of the usual symptoms of cancerous pleurisy. It may be continual or paroxysmal, and is relieved by thoracentesis, but the relief is only of short duration. This dyspnœa is found in most of the cases of cancerous pleurisy, and I have seen it cause terrible agony on three occasions; it depends chiefly upon the cancerous lesions in the mediastinum and the lung. Similar dyspnœa is not found in chronic tubercular hæmorrhagic pleurisy. Some cases of hæmorrhagic pleurisy, associated with acute tuberculosis of the lung and of the pleura, may be accompanied by acute dyspnœa; in these cases, however, the fever is high; this does not happen in cancerous pleurisy.

**Permanent displacement of the heart** is seen in cancer of the left pleura; the factors which account for this displacement are: first, the fluid effused into the pleural cavity, and next, the growth in the pleura and in



the lung, which also takes some part, though not, as a rule, to such an extent as does the pleuritic effusion. Further, the heart sounds are clearly audible all over the chest, as though they were transmitted to the ear by solidified lung, which is a good conductor of sound.

**Acceleration of the pulse** has often been observed; and the pulse-rate may rise to 100, or even to 120, although fever is absent. It has been asked whether this acceleration may not be due to the pressure of the growth on the pneumogastric nerve. It is interesting to note, that in one of my patients, who had continuous tachycardia, I found a nodule of cancer of the size of a hemp-seed in the interventricular septum of the heart.

The **nature of the fluid**, obtained by thoracentesis, may furnish useful evidence in favour of cancer. The colour of the fluid is often brownish or blackish. It is noteworthy that the effusion contains very little fibrin; on the other hand, fibrin is present in considerable amount in most cases of pleuritic effusion, due to tubercular disease of the pleura. Fraenkel, Quincke, and others have observed polymorphous epithelial cells with a large nucleus and vacuoles in the fluid obtained in cancer of the pleura; these cells may be isolated or may be grouped *en masse*. These points are well shown in Figs. 16 and 17 (page 293); in Fig. 16 we see that the cells are polymorphous and isolated, Fig. 17 shows the cells aggregated *en masse*.

Bard, who has done some very careful work upon the nature of the various effusions in diseases of the pleura, has laid stress upon the characters of the serum after centrifugalization. He finds that, in cases of cancer of the pleura, the serum, obtained by thoracentesis, remains coloured by the hæmoglobin. This colouration of the fluid means that the red corpuscles undergo hæmolysis, and that their hæmoglobin is dissolved in the serum. This hæmolytic action, which can be readily recognized with the naked eye, is rendered quite certain by the addition of tincture of guaiacum and turpentine (the laked serum takes on a blue colour with this reagent). He has also found that the serum remains colourless in hæmorrhagic effusions, due to other conditions, because hæmolysis does not occur. These researches have not been confirmed by all writers.

In some cases, the pleuritic fluid consists of almost pure blood. When thoracentesis is performed, the fluid obtained closely resembles arterial or venous blood; such a finding naturally awakens a feeling of alarm. In one of my patients (this case will be found with all details in a thesis published by one of my assistants, M. Vergely) the puncture gave exit to a fluid having the appearance of arterial blood. The appearance of the fluid led me to believe, at first sight, that the condition present might be one of intra-pleural hæmatoma, following upon the rupture of an atheromatous or

of a dilated aorta. These intra-pleural hæmorrhages may occur as the result of any malignant tumour: lymphosarcoma, colloid cancer, epithelioma, endothelial cancer, etc. In these cases the blood remains liquid, and coagulation does not occur.

The **rapid and persistent reproduction** of hæmorrhagic fluid after thoracentesis is common to tuberculosis and to cancer; it is, however, much more marked in the case of cancer. Thus, in one of my cases thoracentesis was performed thirty-three times in five months, and 44 pints of hæmorrhagic fluid withdrawn. In one of Desnos' cases thoracentesis was performed thirty times in six months, in a woman with cancer of the pleura, and 80 pints of hæmorrhagic fluid were withdrawn. I have, however, seen cases in which the fluid of pleuro-pulmonary cancer may dry up after a few punctures; and, on the other hand, I have seen cases of tubercular hæmorrhagic pleurisy in which the fluid formed with such obstinacy that six, ten, and fifteen punctures were necessary. It would, therefore, be wrong to base an absolute opinion upon the drying up, or upon the obstinacy of the hæmorrhagic fluid, in order to banish the idea of cancer.

The youth of the patient is no argument against the hypothesis of cancerous pleurisy, for this lesion has been found in patients who were only eighteen years, twenty-four years, twenty-three years, twenty-two years, and ten years of age respectively. One of my patients was only twenty years of age, and a young woman whom I saw on October 19, 1910, was but thirty-two years of age. She was in perfect health when primary cancer of the pleura with hæmorrhagic effusion developed. Age, therefore, supplied only a very vague indication as to the diagnosis of the cause.

In many cases, especially at the commencement of the pleurisy, the diagnosis of the cancerous lesion can be affirmed only by the cytological examination of the fluid.

Many years ago Fraenkel and Quinke found polymorphous epithelial cells, with a large nucleus and vacuoles in the fluid; the cells occurred alone or in masses. Nattan-Larrier, from his study of cases of cancer of the pleura in my wards, has worked out the cytological formula of these effusions. Besides the red corpuscles, two different types are found—(1) Large thick masses of cells, with polycyclical outlines; the cells, which are arranged in several planes, vary in size, and show indistinct outlines with refracting vacuolar protoplasm and irregular unequally stained nuclei. (2) Isolated cancer cells, which are irregular in size and are larger than leucocytes or endothelial cells; they have an ovoid or irregular form, clearly defined outlines, refracting protoplasm, studded with basophile granules and clear vacuoles, and multiple nuclei irregularly disposed and unevenly stained. Polynuclear eosinophiles are never met with in the fluid of those hæmorrhagic pleurisies.



The following figures are taken from my lecture on hæmorrhagic cancerous pleurisy.

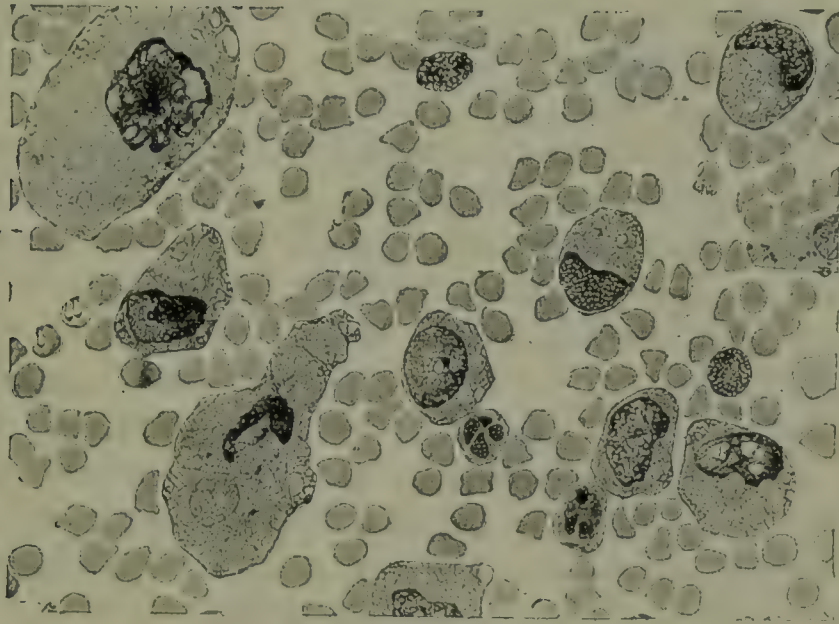


FIG. 16.

Fig. 16 shows the sediment of the blood-stained fluid from a woman who was attacked by cancer of the pleura while in excellent health. Morphologically, most of the cells do not correspond either to the elements of the blood (large mononuclear or polynuclear leucocytes) or to the endothelial cells. They are metatypical and polymorphous; ovoid, polygonal or racket-shaped. The protoplasm is studded with vacuoles; the nucleus is, in some cases, represented only by a few angular granules.

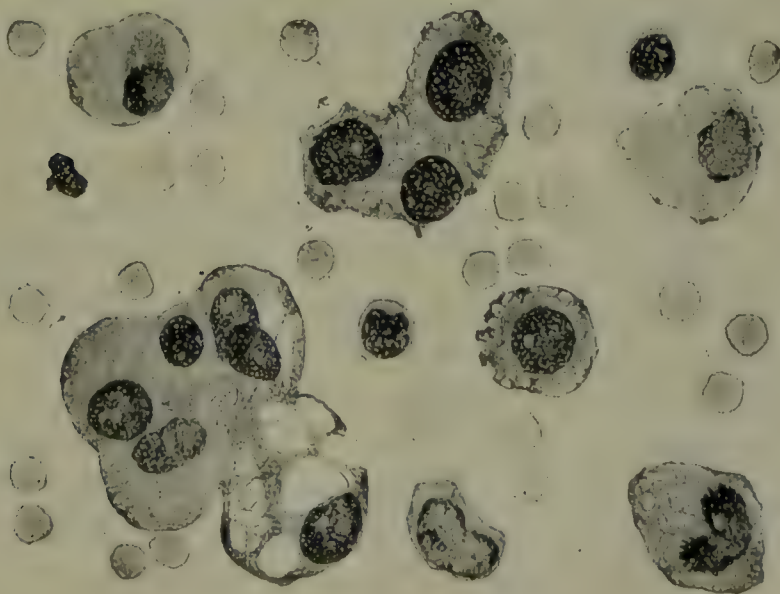


FIG. 17.

Fig. 17 shows masses and cells; the former are thick and made up of large cells of unequal size. The protoplasm is stained a rose-violet colour, and is studded with vacuoles. The nuclei, which are rich in chromatin, vary in size and shape. The isolated cells present the same appearance as those aggregated *en masse*.

Let me repeat in brief what I have said previously. In some cases the clinical diagnosis of cancer of the pleura with hæmorrhagic effusion is easy, *i.e.* when a patient with hæmorrhagic effusion in the pleura has cancer in another region. The diagnosis is easy when hæmorrhagic pleurisy appears after a more or less recent operation for cancer in some other part. On the other hand, the clinical diagnosis of cancer of the pleura is not possible if the patient has, while in excellent health, been taken ill with hæmorrhagic pleurisy. We are, then, led to suspect simple hæmatoma of the pleura or primary pleuro-tuberculosis. We are loth to mention cancer—and yet the cytological examination of the hæmorrhagic effusion shows that the condition is cancer; without such an examination the diagnosis of the cause would have long remained in doubt.

I saw a case of this kind in a woman thirty-two years of age; she was in excellent health, when pleurisy of an insidious kind appeared. Thoracentesis had given issue to hæmorrhagic fluid. Clinically we should not have dared to diagnose cancer of the pleura, and yet the cytological examination proved the presence of cancer cells. The patient died in six months, after being tapped repeatedly.

#### Fourth Group.

**Description.**—Under the term “**pleural hæmatoma**” we must include cases of hæmorrhagic pleurisy, which are not due to tuberculosis or to cancer. The anatomical process is in some points comparable to chronic hydrocele and to pachymeningitis; the condition is a hæmorrhagic pachy-pleuritis.

In some cases the hæmatoma, according to Wintrich, is said to be genuine pleurisy, in which the intensity of the initial inflammation determines hæmorrhage into the pleura. A typical case of **simple hæmatoma** may be thus described: A patient has hæmorrhagic pleurisy. The symptoms sometimes resemble those of simple pleurisy, but at other times the appearance of the patient and the general symptoms may lead us to fear tuberculosis or cancer of the pleura. Thoracentesis is performed, and hæmorrhagic fluid drawn off. In simple hæmatoma the liquid is fibrinous, and the tendency to reaccumulation is slight. The condition sometimes yields to a single puncture, and we are rarely obliged to aspirate more than two or three



times. The relief is notable, and the general condition shows progressive improvement.

The diagnosis of simple hæmatoma must not be hastily made, even though the conditions have yielded to thoracentesis. In some cases, as I have already said, hæmorrhagic pleurisy is the only indication of local or primary tuberculosis of the pleura. Hæmorrhagic pleurisy may recover after thoracentesis, when a favourable prognosis holds good; on the other hand, a pleural hæmatoma is thought to have been cured, and the error is recognized some months later, when undoubted signs of tuberculosis appear.

The more I see, the more I believe in the rarity of pleural hæmatoma; the condition is nearly always tubercular. In my wards at the Necker Hospital I had a patient with every sign of pleural hæmatoma. The fluid, which dried up after a single puncture, did not cause inoculation tuberculosis in guinea-pigs. The patient, who had never had any sign of pulmonary tuberculosis, left the hospital in good health. If I had lost sight of him, I should have thought that it was a case of simple hæmatoma, but he came back to me a year later with pulmonary tuberculosis. The so-called pleural hæmatoma was therefore hæmorrhagic tubercular pleurisy.

### Treatment of Hæmorrhagic Pleurisies.

The **treatment** is very simple. Revulsives, blisters, and other medical means, such as diuretics and purgatives, etc., give here no more benefit than they do in sero-fibrinous pleurisy.

What line of treatment must be adopted in a case of hæmorrhagic pleurisy? One treatment alone is rational—viz., aspiration of the fluid. Pleurotomy, customary in empyema, should not be employed in these cases.

The rules for aspiration laid down under simple pleurisy are in every way applicable to hæmorrhagic effusions. The pleura is punctured with a No. 2 or No. 3 needle, care being taken never to withdraw more than 2 pints of fluid at one sitting. In this way we can avoid fits of coughing, attacks of dyspnoea, intrathoracic pain, and other much more serious troubles which sometimes accompany thoracentesis when too large a quantity of fluid **has wrongly been withdrawn** at one sitting. As I have discussed the important question of thoracentesis fully, further notice is needless. The evacuation of 2 pints of fluid at a sitting, which represents the **maximum** amount in simple pleurisy, is usually too high in the hæmorrhagic variety. After evacuation of 20 to 30 ounces, the patient often experiences dragging pains, and I have been several times obliged to stop the flow after 10 or 15 ounces.

The patient rarely experiences the marked relief which follows the evacuation of the fluid in simple pleurisy. This fact depends much on the condition of the pleura and of the lung, as well as of their respective lesions.

I have always seen that the relief is more marked in cases of tuberculosis than of cancer.

When the fluid reaccumulates rapidly, we are obliged to perform thoracentesis repeatedly, and the patient finally **clamours for it**, because the operation gives momentary relief to his distress.

I recently treated a lady who had been sent to me from Lisbon. She begged me to perform thoracentesis every four or five days. I succeeded in lessening her terrible dyspnœa by means of repeated punctures and injections of morphia. Thoracentesis is only to be performed when there is necessity, and only the **surplus** in the pleura should be drawn off. As the fluid is sometimes very rich in blood cells, thoracentesis is a form of bleeding, and repeated punctures cause weakness.

In certain cases of hæmorrhagic pleurisy the fluid yields after one, two, or three punctures; this happens in simple hæmatoma of the pleura, and in some cases of tubercular pleurisy. In cancerous pleurisy the fluid is reproduced with more obstinacy. In some malignant cases, however, we succeed in drying up the fluid. All hæmorrhagic pleurisies may, then, be curable. The tuberculosis is but little curable, the cancer is incurable. Under some circumstances the fluid which contained much blood at the first puncture contains less in the following ones; it loses its tint and becomes serous. These changes are seen both in tuberculosis and in cancer, as I have reported. These very important facts show us that it would be wrong to take the drying up or the decoloration of a hæmorrhagic fluid as the basis of a favourable or of an unfavourable prognosis as to the cause of the hæmorrhage. The evolution of hæmorrhagic fluids in the pleura gives us imperfect information as to the cause and the nature of the lesion which produces the hæmorrhage.

Hæmorrhagic pleurisies only become **purulent** (a change which is very **rare**) if organisms of suppuration produce secondary infection. In the contrary case, whatever be the number of the punctures, the fluid does not become purulent, either in simple hæmatoma or in tubercular and cancerous pleurisy.

I have performed more than thirty punctures on the same patient, but the hæmorrhagic fluid did not become purulent.

Thoracentesis is the only means available in cancerous pleurisy. The dyspnœa is commonly so acute that we are obliged to give several injections of morphia daily.

The commonplace treatment by tonics and constitutional remedies, such as arsenic, lecithin, and preparations of coca and kola remains.



IV. TRAUMATIC HÆMOTHORAX.

Traumatic hæmothorax demands the physician's attention, and therefore deserves notice in a text-book of medicine.\*

*Résumé of a Case.*—On September 24, 1906, man, aged twenty-two, admitted to the Hôtel-Dieu, under Le Dentu, for a stab in the back. Pain in chest and dyspnœa were severe two days later; he was therefore transferred to my care. The wound, which had healed over, was in the fifth left intercostal space, about an inch from the spine. Inspection showed slight bulging, with absence of vibrations, and dullness at the left base. Traube's space was resonant.

Soft tubular breathing on expiration, ægophony, and pectoriloquy pointed to pleural effusion. No signs of pneumothorax. Heart not displaced; cough frequent; dyspnœa somewhat marked; temperature 103° F. (this fact attracted my notice). No hæmoptysis; no blood-stained sputum. Diagnosis: traumatic hæmothorax.

Exploration in order to examine the fluid, and also to see if the pleura were infected. 20 c.c. of fluid, almost as red as pure blood, withdrawn.

During the first week in October the only change was an increase in the amount of effusion, and during the next week it was clearly abundant. The dullness now extended higher, vocal fremitus was absent over the lower two-thirds of the chest, and the heart was displaced to the right. The increase in the effusion was not due to blood, but resulted from sero-fibrinous pleurisy, as proved by punctures. On October 18 the fluid had lost its former red tint. Friction sounds above the effusion proved pleurisy following hæmothorax.

On November 1 the fluid had partially absorbed; friction sounds were present in the axillary region. The fluid, on puncture, was pale yellow and fibrinous. Patient's appetite was good, his general condition excellent, and he left for Vincennes on November 11.

He came back a week later for slight dyspnœa. There was a good deal of fluid, and on puncture 200 c.c. of sero-fibrinous liquid were drawn off. A fortnight later he went out well.

The nature of the fluid withdrawn at the different punctures was determined in my laboratory. As aerobic and anaerobic cultures on solid and fluid media showed the absolute *sterility* of the liquid, the fever clearly did not depend on infection of the pleura. At the first puncture (September 27) the fluid, which was almost blood-red, contained 2,100,000 red and 4,000 white corpuscles. The fluid was put in a tube; twenty-four hours later we found a red sediment, containing blood cells, and amounting to a fifth of the contents of the tube. The supernatant fluid was not laky, but pale yellow; it was not fibrinous, and showed no trace of coagulation. No clot had formed in this tube on December 10. We were surprised that the sediment formed one-fifth and the serum four-fifths. Did this excess of serum come from clots in the pleura? If this were so, some of the blood effused must have clotted.

Puncture, October 18: the fluid was quite different. It was no longer red, and its rosy tint showed how quickly the red cells had been absorbed. The proportion of red to white cells was only 35,000 to 6,200.

|                       |    |    |    |    | Per Cent. |
|-----------------------|----|----|----|----|-----------|
| Eosinophiles          | .. | .. | .. | .. | .. 35     |
| Lymphocytes           | .. | .. | .. | .. | .. 35     |
| Macrophages           | .. | .. | .. | .. | .. 20     |
| Cells from the pleura | .. | .. | .. | .. | .. 10     |

Nucleated red corpuscles: 1 or 2.

\* Dieulafoy, *Clinique Médicale de l'Hôtel-Dieu*, 1906, 11<sup>me</sup> et 12<sup>me</sup> leçon.

The deposit, on standing, was not red, but of a rose colour, and its amount about one-thirtieth of the total contents. Flakes of fibrin were floating in the yellow fluid.

On November 11 the fluid resembled that of an ordinary pleurisy, and contained 13,000 red to 8,500 white corpuscles. It was practically a "histologically hæmorrhagic" pleurisy. No deposit appeared on standing; a thin layer of sediment, with a rosy tint, appeared only after centrifugalization. The fluid was sero-fibrinous, and clotted in the tube.

Blood-counts showed :

|             |    | Red Corpuscles. |    | White Corpuscles. |
|-------------|----|-----------------|----|-------------------|
| November 1  | .. | .. 3,200,000    | .. | 5,000             |
| November 11 | .. | .. 3,840,000    | .. | 4,400             |
| November 22 | .. | .. 4,200,000    | .. | 6,000             |

The eosinophiles amounted to 10 per cent.

**Pathological Physiology.**—Does the blood effused in hæmothorax remain fluid, or does it form clots? Trousseau and Leblanc, from experiments on the horse, have shown that clotting occurs if blood be allowed to flow into the pleura after section of an intercostal artery. Trousseau hence concluded that similar coagulation might take place in hæmothorax in man.

This view has recently been opposed by Tuffier and Milian, who state "the bloody effusion in the pleura does not clot in cases of traumatic hæmothorax." We can decide the point by consulting the published cases of traumatic hæmothorax (C. de Géry).

**CASE 1.**—Very severe hæmothorax on the left side. Six pints of bright blood drawn off. Later, 4 pints of blood, "mixed with clots, which at times stopped the flow," drawn off by thoracentesis.

**CASE 2.**—Young woman wounded by a revolver bullet. Hæmothorax formed quickly; repeated hæmoptysis occurred; weakness was extreme, and syncope imminent. Two inches of the eleventh rib resected. Blood gushed out, and blackish clots, as large as oranges, were expelled on expiration.

**CASES 3 AND 4.**—Hæmothorax. Resection of ribs; fluid blood and clots removed.

These quotations prove that Trousseau was right. The blood may clot in the pleura, just as it does in the peritoneum, the pericardium, the meninges, and the joints. This fact has an important practical application; the clot may plug the wound in the lung. The results of Trousseau's experiments may be stated as follows :

Hæmothorax was produced by making a penetrating wound of the lung in a horse. The animal was killed, and the wound in the lung examined. "A clot of fibrin filled up the wound in the lung like a sword in its scabbard." This protecting clot sometimes formed half an hour after the injury, and on attempting to draw the clot out it was necessary to break it, because it was embedded in the interlobular cellular tissue by numberless fibrinous radicles, which broke on traction.

If the post-mortem was not performed till forty-eight or seventy-two hours after the injury, the wound was closed by a remarkable process.



The wound in the lung was inflamed, and the pleura itself was also affected to a variable extent. A plastic exudate was adherent to the serous membrane, and blended with the fibrinous nucleus of the wound, intimately adhering to it. The whole track of the injury was thus obliterated by fibrinous clot, and the edges were covered by a fibrinous disc, adherent to the pleura, to the lips of the wound, and to the plug of clot.

The effusion of blood, therefore, in traumatic hæmothorax may form clots in the pleural cavity; yet, strange to say, the same blood, when drawn off and placed in a test-glass, does not clot, or at least coagulation is imperfect or delayed. The study of cases clears up this point.

In my own case, the bloody fluid did not coagulate in the tube, which was kept in the laboratory for two months.

In Sacquépée's case the fluid from the pleura was set aside, and behaved like defibrinated blood; it did not clot, although it was kept for two months.

Patel and Leriche punctured the pleura four days after a wound, and drew off 7 ounces of blood. The fluid had not clotted by night.

In a case published by Gaultier and Français puncture was performed on the twenty-first day. Three pints of bloody fluid were drawn off. The fluid did not begin to clot till six hours later, and the clot did not retract till twelve hours later, while fresh blood from the patient's finger clotted normally at the end of half an hour.

Milian quotes a case in which the fluid was drawn off sixteen days after the injury. It clotted at the end of an hour, and the retraction of the clot expressed yellowish serum.

These examples prove that we have not yet exact knowledge as to the properties of the fluid in hæmothorax. As a rule, the fluid does not clot, though it may do so, and in the latter event coagulation is delayed.

The blood in hæmothorax remains alive, hæmatolysis does not occur, and the serum does not become laky, but remains of a straw colour. All authorities agree upon this point, as the following examples prove:

In my case, hæmatolysis was quite absent, the red corpuscles were intact, and the exuded serum kept its yellow tint for six weeks. In Tuffier and Milian's case "it was not possible to find blood cells in process of mortification. The red corpuscles and the leucocytes preserved their normal shape and their usual reactions; they took stains perfectly, and, in fact, remained alive. The serum exuded *in vitro* after retraction of the clot was yellowish, and did not show the least tinge of red. This fact shows that hæmatolysis did not occur in the effusion, and that the intact red corpuscles were destined to be absorbed." In Sacquépée's case "the serous fluid above the red sediment had a very marked yellow colour; it showed no colouring due to hæmoglobin, and was not laky; the same characters obtained in all the samples taken."

The point is therefore decided, and we may state that hæmatolysis does not occur in traumatic hæmothorax.

Cytological examination of the blood gives interesting information. When I gave the cellular formula in my case, I mentioned that the eosinophiles were as high as 35 per cent. The eosinophiles in the blood were 10 per cent., while normal blood only shows 2 to 4 per cent.

Other cases show this enormous predominance of the eosinophiles. In Sacquépée's counts they amounted to 33 per cent.

I do not wish here to discuss eosinophilia in hæmorrhagic pleurisies, and in traumatic hæmothorax. Burnet thus writes on pleural eosinophilia :

“ The affinities for stains between the eosinophiles and the red blood-corpuscles would lead us to suppose that the eosinophile is a polynuclear cell charged with red corpuscles. Klein describes a case of hæmorrhagic pleurisy, with marked eosinophilia in the exudate and in the blood. He holds that the eosinophiles are not leucocytes normally charged with hæmoglobin, but phagocytes which have ingested degenerated red corpuscles.”

Widal and Faure-Beaulieu arrive at the following conclusions upon pleural eosinophilia : “ Our cytological findings lend support to Dominici's idea that the eosinophiles may be of lymphatic origin. We conclude from our researches that the pleural eosinophilia takes place *in situ*, and that the blood eosinophilia results from the migration of cells which have their origin in the morbid focus.”

However this may be, it is certain that my patient had 35 per cent. of eosinophiles in the effusion, and 10 per cent. in his blood.

The absorption of the red corpuscles may be very rapid in hæmothorax. The colour may change from red to rose in a week or a fortnight. The number in my patient fell from 2,100,000 to 35,000 in three weeks. In Tuffier and Milian's case the red corpuscles fell from 390,000 to 22,500 in sixteen days. “ As we know by blood-counts that the number of corpuscles diminishes daily in the effusion, and as, on the other hand, we know that these red corpuscles are not destroyed, they can only be absorbed.” The method of this rapid absorption of the red corpuscles in hæmothorax has been repeatedly discussed. We can eliminate hæmatolysis because the red corpuscles remain intact, and do not lose their hæmoglobin, as the yellow tint of the serum proves. How, then, do they disappear ? Do the eosinophiles absorb the red corpuscles ? “ It is probable that the polynuclear and mononuclear cells destroy the red corpuscles, and carry away the débris. On the other hand, the endothelial cells do not remain inactive ; we often find perfectly intact corpuscles in their protoplasm, while others are swollen, pale, and surrounded by a vacuole. A certain number disappear, therefore, by intracellular digestion. These two modes, however, do not appear sufficient to produce such a rapid effect. Other unknown causes doubtless play some part ” (Sacquépée).

It is strange that the number of white cells increases, while the number of red corpuscles rapidly diminishes. In my patient the white cells rose from 4,000 to 8,500. The polynuclears first disappear ; the lymphocytes are found in the fluid for a long while.



**Symptoms.**—We may now commence our clinical study of traumatic hæmothorax. Gross injuries of the chest, the heart, and the great vessels demand surgical treatment, and are therefore foreign to our subject, which embraces only those forms of hæmothorax that are amenable to medical science.

We shall here consider the course of hæmothorax apart from complications, which will be dealt with later.

A patient has been wounded in the chest. The injury may involve all the layers of the chest-wall, or may not be visible externally (fracture of ribs); the bleeding may come from some vessel in the wall or from a wound of the lung, but yet in each case the blood very readily finds its way into the pleura, and forms a hæmothorax.

The initial symptoms vary somewhat with the nature of the wound and with the amount of bleeding. The injured man may become pale, complain of pain and distress, and lose consciousness. The injury, however, may be almost unnoticed, and the only sign may at first sight be the external bleeding.

The wound of the lung may show itself by early hæmoptysis. In some instances the patient brings up blood-stained sputum, but in others we find frothy bright red blood, which is coughed up at intervals. One of my patients, with a bullet wound, had a severe attack of hæmoptysis, which was followed by slighter attacks. In Sacquépée's case the hæmoptysis lasted for several days. The case reported by Connellsville is exceptional: the patient died from hæmoptysis in a few minutes, and it was found post mortem that a fragment of rib had pierced the lung.

It would be reasonable to suppose that a wound of the lung would always be followed, if not by abundant hæmoptysis, at least by blood-stained sputum. Our supposition does not hold good; slight or profuse hæmoptysis is far from being frequent. My patient who was stabbed in the chest did not bring up any blood. Hæmoptysis was noted only eight or ten times in forty-four cases of traumatic hæmothorax collected by de Géry. It is true that hæmothorax is not always associated with a wound of the lung; the blood in the pleura may come solely from the chest-wall, in which case the absence of hæmoptysis is readily explained.

When blood invades the pleural cavity, dullness, absence of vocal fremitus, tubular breathing, and perhaps ægophony enable us to determine the presence and the amount of the effusion. Cough, dyspnœa, weakness, and pallor vary with the severity of the case.

Hæmothorax may not be accompanied by fever, but in some cases, from the third to the fifteenth day, the temperature rises to 103° or 104° F., and remains high for some time. We may then ask whether the pleura has not been infected by the foreign body. The fever leads us to fear pneumonia or

empyema, and we are the more anxious in that the injured man has more pain and more distress while the fluid in the pleura increases.

Such an attack naturally awakens our attention, but fortunately it does not, as a rule, indicate any serious complication. When my patient had a temperature of 103° F. four days after the injury, we were not greatly surprised; cultures of the fluid remained quite sterile, and we therefore concluded that the attack of fever indicated absorption of the blood and the onset of secondary pleurisy.

We know that fever may last some time in cases of hæmothorax, quite apart from infection, and we now admit that absorption of the red corpuscles gives rise to fever, and to effusion of serous fluid in the pleura.

The same condition obtains in subarachnoid hæmorrhage, when blood passes into the ventricles of the brain and into the subarachnoid space of the spinal cord. The blood, as Prus says, may have a very irritant action upon the parts with which it comes in contact. "The recognition of Kernig's sign and the hæmatolytic reactions in subarachnoid hæmorrhage show that the extravasated red corpuscles undoubtedly cause inflammatory changes."

Further, whatever truth there may be in these hypotheses, it is certain that fever in hæmothorax may coincide with absorption of the red corpuscles, with the gradual decoloration of the hæmorrhagic effusion, with the appearance of serous fluid, and with the subsequent discovery of pleural friction sounds. The following cases prove this point:

In my patient the temperature was 105° F. on admission, and the attack was of short duration. We found soon after an increase in the pleural fluid, which coincided with the rapid decoloration of the original effusion, the fibrinous condition of the newly-formed fluid, the marked diminution in the number of red corpuscles, and the appearance of friction sounds. In Sacquépée's case the fluid is said to have remained stationary for some time, although the number of red corpuscles diminished.

"We must conclude that a large amount of serous fluid passed into the pleura, and thus compensated for the absorption of the solid part." In Tuffier and Milian's case the patient had fever, the serous effusion increased in amount, and the specimens showed progressive diminution in the red corpuscles. Friction sounds were heard later in the axilla.

Secondary pleurisy may appear early or late, but it is impossible to fix the exact moment of its onset, for the same signs (dullness, absence of fremitus, tubular breathing, ægophony) are common both to serous effusion and to hæmothorax.

Unless we are on our guard, we may wrongly suppose that more blood is being poured out into the pleura, when a serous effusion is really being formed. In order to gain information as to the relative importance of the bloody and of the serous effusion, we may take samples of the fluid, which



are placed in small tubes. On comparison of the samples, the presence of pleurisy is shown by the rapid attenuation of the red tint of the lowest layer, which coincides with the diminution in the number of red corpuscles, and by the appearance of fibrin in the serous layer. Friction sounds later make the diagnosis certain.

We may fear that the fever is due to infection of the lung, or to commencing empyema; we have, however, a simple means of deciding this point: "Make cultures of the fluid. If you find them sterile, you can eliminate any idea of infection."

**Complications.**—I have first sketched the course of an uncomplicated case of traumatic hæmothorax. The prognosis is nearly always good, and recovery results in a few weeks, without the need for surgical intervention. Let us now pass on to the complications of hæmothorax, and first consider the condition known as "hæmopneumothorax." Air may enter the pleural cavity either through a wound of the chest-wall or of the lung. This double channel of entrance might lead us to suppose that pneumothorax would be a frequent complication of chest wounds. I am of opinion, however, that this complication is somewhat rare. It was not seen in the cases quoted above, and was found in only twelve out of forty-four cases collected by De Géry. It may perchance pass unnoticed, because we can only make a summary examination in a patient who is fainting from loss of blood.

The presence of pneumothorax makes the situation worse: distress, suffocation, weakness of the pulse, and lipothymia are more marked when this complication is present. Consideration for the patient does not always allow us to make a complete examination, and we cannot try for a succussion splash; yet the tympanitic note on light percussion over the upper part of the chest is usually sufficient, especially if amphoric breathing is also present.

In some cases we may hear the air entering and leaving the chest at each respiration.

Let us now consider suppurating hæmothorax. In some patients the pleura is infected, and the fluid becomes purulent, as in the following examples:

Hæmothorax occurred on the right side from a bullet-wound (Greenleaf). A little blood was drawn off by thoracentesis. Empyema supervened; Estlander's operation was performed, and the patient finally recovered.

*Case reported by Christovitch.*—A boy was shot at close range with a revolver. He was kept quiet for three days. On the fourth day effusion, fever, and dyspnœa. On the sixth day he nearly died from suffocation. Foul-smelling bloody fluid was withdrawn on puncture in the seventh space. Resection of the fifth and sixth ribs. On incision of the pleura and the lung much pus and blood came out, the bullet and three bits of gangrenous lung being removed. Perfect recovery in two months.

Elimoff has published the following case: A man was wounded in the axilla with a shot-gun. Signs of hæmopneumothorax. Operation postponed, as patient was weak.

Some days later foul-smelling bloody fluid escaped from the wound at each fit of coughing; the wad of the charge soon came away in the secretion. Twelve days after the accident much pus escaped from the wound. Thoracotomy was proposed, but patient refused. Rapid improvement took place, and cure was complete in six weeks.

Peyrot says: A boy was shot in the second intercostal space. Hæmothorax resulted. Symptoms of infection appeared twelve days later: temperature,  $103^{\circ}$  F.; pulse-rate, 112; rapid breathing; and great collapse. As these symptoms became worse, the ribs were resected, but he died on the table. Almost 6 pints of reddish fluid, with foul-smelling gas, were let out by the incision. Examination of the fluid showed streptococci and anaerobic microbes.

Loeper related the following case to me: A boy had been stabbed in the third left space a week before. He had some distress, and complained of acute pains in the chest on the left side. Vocal fremitus was abolished on that side up to the subclavicular region. Dullness was absolute over the same area. That night the temperature rose to  $103.5^{\circ}$  F., and next night to  $104^{\circ}$  F.; it then varied between  $100^{\circ}$  and  $104^{\circ}$  F.

Normal blood was taken from the finger; clotting was rapid, and the clot retracted well. The serum, rather dark in colour, contained urobilin, and a normal amount of chlorides. Freezing-point was  $33.02^{\circ}$  F. and  $33.08^{\circ}$  F. The red corpuscles were much reduced in number; the count showed 2,650,000, with a fair number of poikilocytes. The white corpuscles were much increased in number; the count showed 17,500, 84 per cent. being polynuclears and 16 per cent. mononuclears. The patient had signs of a left effusion, but the high temperature, the severe general symptoms, and the existence of marked leucocytosis were against simple hæmothorax or sero-fibrinous effusion.

The fourth space was punctured, and fluid of a chocolate tint was drawn off. The fluid was odourless, and froze at  $32.84^{\circ}$  F. The microscope showed normal red corpuscles and numerous polynuclear cells, of which some were normal and others were in process of disintegration, indicating suppuration in the fluid. The fluid was sown on agar and on broth. The latter culture at the end of twenty-four hours showed a thin bacillus, which was mobile and present in a pure state; it appeared to be the coli bacillus. Forty-eight hours later the culture became green, as though the pyocyanus were present. The green tint later changed to a blackish-brown colour. The colonies on agar showed similar colour changes. The patient's serum agglutinated the bacilli in the fluid. The case was one of suppurating hæmothorax. Thoracotomy gave exit to brownish fluid, containing many clots. No gas. The fever at once fell, and recovery was complete in six weeks.

Infection of the pleura is therefore fairly common in traumatic hæmothorax. When fever appears in a case of hæmothorax, we must at once look for the cause. The condition may simply coincide with absorption of blood and with the onset of pleurisy; but it may also be due to infection of the pleura, which requires early operation. The diagnosis must be made promptly. Leucocytosis is in favour of pus, but cultures furnish the more reliable information.

Both in my case and in that of Sacquépée the cultures were sterile, and it was therefore clear that the pleura was not infected, in spite of the fever present. The same result obtained in the case reported by Tuffier and Milian. Inoculation on broth, on agar, and on agar with sugar (tubes of Liborius and Veillon) showed absolute sterility, although the patient had fever. In the cases of Peyrot and of Loeper, on the contrary, the hæmothorax was infected, and cultures showed the nature of the organisms.



Infection of the pleura is to be feared both in hæmothorax and in hæmopneumothorax. Every infected case should undergo operation. If the patient dies in spite of operation, he does so because the operation has been performed too late.

I have spoken of the acute forms, but infected hæmothorax may show another variety. The initial symptoms at times subside, and the patient appears on the road to recovery, but yet the infection persists in a chronic form. Peyrot tells how a patient was about to be discharged after the apparent cure of a wound in the right axilla. He had no fever, and would have been looked upon as well if he had not shown signs of a large effusion. Puncture in the eighth space showed the existence of granular pus, which was not homogeneous and had a slight colour.

In two cases reported by Ch. Nélaton the collections of pus were emptied through the bronchi long after the wound of the chest. Peyrot's second case was as follows :

A woman had been stabbed in the chest. The signs of an effusion of blood into the pleura were present. She made good progress, but several weeks later she suddenly coughed up a quantity of very foul-smelling blood. The condition again became normal. She thought that she was cured, and her health appeared excellent, when the same thing occurred some weeks later. Several recurrences took place during the next year.

Traumatic hæmothorax is often accompanied by subcutaneous emphysema, which gives the well-known feeling of downy crepitation. It commences around the wound, especially if the latter is narrow and anfractuous. It usually remains limited, and rarely extends to a distance.

Traumatic pneumonia is chiefly seen in the case of fractured ribs. It is somewhat difficult to diagnose, because the crepitant râles, the tubular breathing, and the rusty sputum are obscured by the signs of hæmothorax or of hæmopneumothorax. Pneumonia is a serious complication *per se*, and, further, it may give rise to infection of the pleura.

**Treatment.**—We may first consider cases that recover without surgical treatment. Two of our patients belong to this category.

A person has just received a penetrating wound of the chest. You are called to see him. The wound is bleeding. The patient, who is pale and distressed, is breathing badly and is coughing up blood, showing that the lung is injured. There is no fever. The pulse is quick, but is of fair strength, so that you may suppose the intrapleural bleeding to be moderate.

Do not tire the patient by useless manipulation; do not probe the wound, but be content to disinfect it, and to apply an occlusion dressing. Absolute rest in bed must be ordered. Be very careful to auscultate and to percuss the chest. If you do not find either tympanitic resonance or amphoric breathing, pneumothorax is absent. The dullness in the

dependent parts will indicate the amount of blood effused. If the patient has much pain in the chest, give an injection of morphia, which is repeated if need be. If the heart is weak, use injections of caffeine; and if the weakness increases, give injections of serum every three hours, and stimulants. A mixture of Rabel water and syrup of rhatany may be given for hæmoptysis. The diet should consist of milk, eggs, and broth. In order to ascertain whether the blood continues to flow into the pleura, be careful at your first visit to take some blood from the finger, and to count the red corpuscles. Repeat this examination every two or three hours, if the condition becomes worse. If you find, after two or three examinations, that the number falls rapidly, you may reasonably hold that the intrapleural hæmorrhage is still going on.

Let us suppose that the patient is not growing worse. A day passes, and the general condition is better. More exact examination shows an effusion, which may amount to about a pint. The situation remains practically unchanged during the next few days. It is then unnecessary to evacuate the fluid, because absorption of the effusion is sometimes very rapid; you need only await events. Recovery in traumatic hæmothorax is very frequent, even though the fluid has not been evacuated. The cases reported by Millard, Sacquépée, Tuffier, De Géry and Bergeaud are of this nature.

Events, however, do not always follow this course, and you will see patients whose condition some hours after the injury causes anxiety, because the intrapleural bleeding continues.

Hæmoptysis and pneumothorax may or may not be present, but yet the condition grows worse. The breathing is not so easy, dyspnœa follows the least movement, the pulse becomes quick, the strength fails, syncope threatens, and the effusion may amount to 3 or 4 pints.

What line of treatment is to be adopted? If it were simply a question of an effusion, which by its large amount threatened life, the problem would be quickly solved by performing thoracentesis. The matter, however, is not always so simple. The source of the bleeding may not be exhausted; the blood from the lung or from the chest-wall may be still flowing into the pleura, and the situation is most serious. The surgeon must now take up the tale, and decide the question of resecting the ribs in order to make a complete exploration of the thoracic cavity and to arrest all bleeding.

I have described two extreme cases: on the one hand, hæmothorax of moderate severity; on the other, grave hæmorrhage, in which operation is imperative. We see, however, intermediate forms in which puncture may replace thoracotomy. Thoracentesis has been performed with success on the third, fourth, sixth, eleventh, and twenty-first days respectively.



What are the indications for puncture? Are we to do it early or late? Are we to be content with drawing off part of the fluid, or must we evacuate a large quantity at one sitting?

I would first state that the expediency of thoracentesis should depend solely upon the condition of the patient. If aggravation of the symptoms coincides with signs indicating abundant effusion, and if immediate surgical intervention is not justifiable, thoracentesis is indicated, in my opinion.

As regards the quantity of fluid that should be drawn off, some authors say that we must only draw off a small amount, while others give the opposite advice. To quote examples:

A case of traumatic hæmothorax was admitted under Jaboulay. As the symptoms were alarming, he performed thoracentesis on the fourth day, and drew off only 8 ounces. The result was excellent, and he insists on the benefit of thoracentesis in such a case.

A man had hæmopneumothorax. Dyspnoea became very acute some days later. Examination of the chest revealed a large effusion. Souligoux performed thoracentesis, but only drew off 8 ounces of bloody fluid. The patient recovered.

Lejars quotes a case of traumatic hæmothorax in which 3 pints of blood-stained fluid were drawn off ten days after the injury. Recovery was uninterrupted.

In a case in which the fluid was slow to absorb, Gaultier and Français performed thoracentesis three weeks after the injury. Three pints of bloody fluid were drawn off. The patient recovered.

A man fell on the shaft of a waggon, and his chest was squeezed by the wheel. No fracture was found, and the surgeon gave a good prognosis. Dr. Jagot was called to him at 9 p.m. The patient was pale and breathless; the left side of the chest was dull, and the heart was pushed over to the right. Jagot diagnosed traumatic hæmothorax, and drew off 6 pints of bright blood by thoracentesis. Relief followed, but the fluid reformed, and 4 pints of chocolate fluid, mixed with clots, were drawn off. Recovery now followed.

These examples prove that recovery followed whether much or little fluid was drawn off. For my part, I do not see the necessity for drawing off at one sitting several pints of fluid; I should be afraid of starting fresh hæmorrhage, or of causing a fresh pneumothorax. I would rather withdraw a pint of fluid, and then repeat the procedure, according to the result obtained. We must not lose sight of the cases in which surgical intervention is imperative; delay or indecision may cost the patient his life.

## V. PURULENT PLEURISIES OF THE GENERAL PLEURAL CAVITY.

**Discussion.**—Writers formerly spoke of “purulent pleurisy,” but we should really speak of “purulent pleurisies.” Bacteriology has completely modified certain questions in medicine, and purulent pleurisy is among the number. I shall now describe purulent pleurisies of the general pleural cavity, and later discuss the interlobar, mediastinal, and diaphragmatic varieties.

It is not possible to write a set chapter on purulent pleurisies, because each variety necessitates a special description, according as it is due to the pneumococcus, to the streptococcus, to tuberculosis, to appendicitis, etc. Each of these types will be described in turn, and we shall see how they differ from one another; each type owes its special characters and its degree of gravity to a special pathogenic agent, and this new view of the question is of much interest. Nevertheless, there are some general considerations which may find their place here.

**Pathological Anatomy.**—The older the mischief, the more are the lesions marked. At the onset the fluid is turbid and sero-purulent, or sometimes purulent; the false membranes are not thick. Later, however, the lesions are well marked, false membranes spread over both layers of the pleura, and may be  $\frac{1}{3}$  inch thick, especially on the parietal pleura. The adhesions between the two layers of the serosa divide up the cavity, and form pockets of pus; they extend their harmful action still farther, fix the ribs by restraining their action, and contribute to deformity of the chest-wall, which becomes flattened and retracted in the lateral and posterior region.

The **fluid** may amount to 4, 6 or 8 pints. The lung, though pushed back and flattened, is but little changed if the pleurisy is recent; in old-standing cases, the lung is reduced to a kind of stump of the size of the fist, and is indurated, fibrous (Brouardel), compressed against the costo-vertebral groove, surrounded by false membranes, and consequently reduced to a condition that renders it incapable of regaining its normal size and functions. The skeleton of the thorax is affected; the periosteum is adherent to the pleural membranes, and the ribs are the seat of osteitis. The pus may make its exit by various channels, by the bronchi (vomica), or by an intercostal space (this is most frequent in the fifth space). The relative frequency of these perforations is thus given by Flammarion:

|                                  |    |    |    |    |    |
|----------------------------------|----|----|----|----|----|
| Pleuro-cutaneous fistulæ         | .. | .. | .. | .. | 10 |
| Pleuro-broncho-cutaneous fistulæ | .. | .. | .. | .. | 5  |
| Pleuro-bronchial fistulæ         | .. | .. | .. | .. | 3  |
| Pleuro-abdominal fistulæ         | .. | .. | .. | .. | 1  |

These statistics prove that empyemata empty themselves more often by an intercostal space than by the bronchi, and the remarkable fact is that the perforation does not occur behind at the most dependent part, but in front, at the middle of the intercostal space, in the proportion of eleven to three. Let me hasten to add that these terminations, which were fairly frequent at a time which I have called the **prehistoric period** of pleurisy, have become almost unknown since early operations for empyema have been performed.

The presence of air in the pleural cavity (pneumothorax by perforation), or the formation of gas (pneumothorax by putrefaction) in the case of putrid pleurisies, constitutes pyopneumothorax. The kidney, the liver,



and the spleen, are sometimes affected by amyloid degeneration in chronic cases of empyema.

**Common Signs.**—Some signs may be common to the various forms of empyema.

**Dullness** is absolute over the whole extent of the effusion, or alternates with resonant zones if the pleurisy is partial or encysted. Sometimes, by reason of adhesions, the pus accumulates at the base of the thorax, depresses the diaphragm at the expense of the abdominal organs, which are pushed down, and the encysted pleurisy at the base then simulates a tumour of the liver, the spleen, or the kidney.

The **vocal fremitus** is abolished or diminished over the effusion, but often persists at the site of the adhesions. The thorax on the affected side undergoes a transient bulging, which is followed by retraction and flattening of the lower intercostal spaces; this flattening is seen in spite of the presence of the fluid, unless it be very abundant. In the latter case the spaces are dilated, and the thorax forms a large curve. The **deformity** in time becomes very marked, especially after the natural or the artificial evacuation of the effusion. The shoulder is depressed, the chest is flattened, the muscles are atrophied, and the vertebral column becomes concave towards the affected side. This deformity, which may persist indefinitely, is a natural and necessary device, destined to fill up the void caused by the partial atrophy of the lung.

**Œdema** of the chest-wall is often seen on the diseased side; it may occur early in the disease, and may be limited to the posterior border of the axilla, or may extend to the arm and hand. In long-standing cases the fine arborizations of a complementary circulation may be seen on the skin of the thorax.

The neighbouring organs are pushed back by the effusion, the **heart** is **displaced**, and the liver is pushed down according as the pus is on the left or the right side, and these vicious positions are the more durable in that they are maintained by the false membranes.

The signs on auscultation are variable, especially when the disease has lasted some time; in some cases they are negative. We may examine and find a total absence of normal and abnormal sounds; at other times we may hear bronchial, cavernous, or amphoric breathing (Landouzy).

**Ægophony**, which may exist when the onset is acute and the liquid is sero-purulent, is wanting in other cases, and aphonic pectoriloquy, which is so clear in sero-fibrinous effusions, may lose its characters (Bacelli). At points where the false membranes exist alone, without adhesions and fluid, a harsh and sawing friction sound may be heard.

In some cases on the left side we find in the region of the effusion a pulsating tumour, or expansile movements, like those of an aneurysm. **Pulsating empyema** is the term applied to this form.

The **fever** has no definite character ; it is more or less acute and intermittent ; it may be absent, or may in time take a hectic course, with evening rise and abundant sweats. The **dyspnœa** varies with the amount of the fluid, the extent of the adhesions, and the condition of the neighbouring organs (pulmonary tuberculosis, pericarditis, and broncho-pneumonia).

When pus is left to make its way through an intercostal space, the patient feels pain at a point which soon becomes more prominent than the neighbouring parts. The tumour takes several days or weeks to form ; it becomes fluctuating, invades one or two intercostal spaces, has an elongated form, and is reducible. The skin becomes thin, red, and perforated, and a **fistula** forms, through which the pus flows out. According to the nature of the fistulous tract, the external air may or may not enter the pleura, and in the former case symptoms of pneumothorax arise. The pus sometimes has a foetid odour, and after a transient improvement, which coincides with the first evacuation of the pus, symptoms of septic absorption and hectic fever may occur if appropriate treatment be not at once employed.

The pus may traverse the tissues like a congestion abscess, and point in the **lumbar region**, most often on the left side. In order to reach this spot it may follow various routes. Sometimes it takes quite a superficial route across the posterior part of the lower intercostal spaces beneath the latissimus dorsi, and in this case the progressive raising of the integument indicates the course of the collection. Sometimes it chooses a deeper route, passes through the diaphragm near the vertebral column, follows the anterior surface of the psoas magnus, or the outer border of the quadratus lumborum, and appears in the lumbar region. The lumbar tumour may eventually reach a large size ; it is oblong, fluctuating, painless, partly reducible, and sometimes shows expansile pulsations (*vide* section on Pulsating Empyema) ; it may become ulcerated when evacuation of the empyema follows pleurisy. When the pus passes into the bronchi (**pleuro-bronchial fistula**), there is a **vomica** (see Section XVI.).

The change from sero-fibrinous or hæmorrhagic fluid to pus, after thoracentesis, is now denied no longer. I may say that this change cannot occur provided the operator takes proper care. I have discussed this question in detail under thoracentesis.

Micro-organisms must be regarded as the final cause of empyema. After these general remarks we may consider the chief varieties of empyema, which owe their special characters to their pathogenic agents. This side of the question has been examined with the greatest care by Netter.



### Purulent Pleurisy due to Streptococci.

**Description.**—The **streptococcus**, which often causes suppuration in the serous membranes, is perhaps the most usual microbe when the disease occurs in adults. I say in adults, because in children the most usual microbe appears to be the pneumococcus.

The presence of the streptococcus in the pleural fluid does not always determine pus; some cases are sero-fibrinous. Pus is seen especially when the pleura is in contact with a focus rich in streptococci (focus of influenzal broncho-pneumonia, bronchial dilatation, tuberculosis, pulmonary gangrene), yet the focus may be so small that it would pass unseen post mortem without a minute examination. In other cases the focus is remote (lesions of the mediastinum, breast, puerperal infection, etc.). Sometimes purulent streptococcal pleurisy arises in the course of an infectious disease (scarlatina, typhoid fever, erysipelas, diphtheria, etc.); it may also occur as a primary infection, the point of entrance of the organism being unknown. The fluid in streptococcal pleurisy is rarely purulent from the start; it is usually first turbid, then sero-purulent, and finally purulent fluid. The turbid appearance may be so slight that a sample on superficial examination will be taken for sero-fibrinous fluid, but microscopic examination usually reveals red corpuscles and polynuclear cells. Bacteriological examination, including cultures, reveals micrococci in little chains.

When the liquid becomes purulent, the pus is not homogeneous and laudable, like that due to the pneumococcus; it separates into two layers in the glass, the upper being serous, the lower dense, with a sediment which falls to the bottom. The false membranes which cover the pleura are not so thick and firm in streptococcal as in pneumococcal pleurisy.

Endocarditis, pericarditis, meningitis, otitis, etc., are much rarer in this variety than in pneumococcal pleurisy, but suppurative meningitis is fairly common.

The **symptoms** and course show nothing special. Sometimes, however, the fever and the temperature undergo great variations, while oedema of the chest-wall is more frequent than in pneumococcal pleurisy; in some cases, too, the disease takes a rapid typhoid course, which answers to the description of septic pleurisies; often, too, the course of the disease is slow, the symptoms are moderate, the fever is not high, and the disease is almost latent. It is therefore clear that the disease may, clinically speaking, assume all forms. **Vomica** is more rare in streptococcal than in pneumococcal pleurisy. In puerperal cases the disease may have a rapid course, an excessive virulence, and a most grave prognosis; it used to appear in epidemic form when epidemics of puerperal fever were seen. These epidemics are no longer seen, or, at least, they ought not to be seen.

The **diagnosis** can only be made by bacteriological examination of the pus. The sample fluid, as I have already said, may be turbid, sero-purulent, or purulent. Bacteriological examination and cultures will indicate the micro-organism. "The appearance of chains, after staining with gentian violet, proves the streptococcus; only if the chains be long, bent, and have very round granules, we must not forget that the pneumococci in serous membranes are often disposed in little chains, though they are straighter and less bent than those of streptococci, and are composed of more elongated and less numerous elements" (Netter). We may add that the pneumococcus is encapsuled.

The **treatment** consists in thoracotomy. The operation for empyema, with or without resection of ribs, is the rational treatment for these cases. It is extremely rare indeed for the streptococcal form to recover, like pneumococcal pleurisy, after simple puncture; when this occurs the virulence of the streptococcus is markedly attenuated. I have seen a case of this nature at the Necker Hospital. It is described by my present colleague Widal.

Pleurisy had appeared as a late complication in the puerperium, and was then the only abnormal feature. Examination and cultures of the fluid showed the presence of streptococci, and inoculation showed that the pus had lost in virulence. At each fresh puncture the fluid was less rich in microbes, and the virulence diminished, so that the patient was cured by aspiration, without operation for empyema. Such a result is exceptional; the rule is early rather than late surgical intervention.

### **Purulent Pneumococcal Pleurisy.**

**Description.**—This form is rarer in the adult than streptococcal pleurisy; on the other hand, it is much more frequent in the child. The pneumococcus is usually present in a pure state, other microbes being only met with in a fourth of the cases.

The condition is commonly associated with pneumonia, but it may be independent when the pneumococcus infects the pleura on its own account. The pleura is nearly always affected in the course of pneumonia. The pleurisy is often dry, and confines itself to the production of false membranes of variable thickness, which line the pleura for a certain extent, especially at the interlobar fissures. In other cases sero-fibrinous or purulent effusion is also present. It is a remarkable fact that the pneumococcus, which does not produce pus in the lung, readily does so in the serous membranes (pleura, pericardium, meninges).

Maragliano has systematically punctured the pleura in fifty-eight cases of pneumonia; in thirty-eight of these, sero-fibrinous or fibrino-purulent effusion was found.

The pus is "laudable," according to the expression of ancient writers. It



is rich in cellular elements, and fibrin—greenish-yellow, thick, viscid and creamy, homogeneous, and usually inodorous. When it is placed in a glass, it does not separate into serous fluid and plasma, like the pus due to the streptococcus.

Bacteriological examination and cultures reveal the presence of the pneumococcus. "The pneumococci are often present in the form of long lines, which might be mistaken for chains of *Streptococcus pyogenes*. The pneumococcus, however, in these cases usually presents stained capsules that are very easy of detection, and its elements have a more marked lanceolated form than in pneumonic expectoration. **Phagocytosis** is common, especially in the benign forms in process of recovery" (Netter).

The trouble usually supervenes during the decline of pneumonia, or even during convalescence. In many cases pneumonia has ended two or three weeks before pleurisy appears. These cases have also received the name metapneumonic or postpneumonic. Metapneumonic pleurisy (Gerhardt) may be sero-fibrinous, but I am concerned here with the purulent form, which is the more frequent.

Purulent metapneumonic pleurisy may invade the great pleural cavity, but is more often confined to some part of the pleura, being interlobar, mediastinal, or diaphragmatic.

Metapneumonic pleurisy is sometimes seen in series, as if they were tributary to an epidemic.

Pneumonic pleurisy rarely begins with very acute symptoms, and the onset is more usually insidious, being unaccompanied by pain and by recrudescence of fever. **Œdema** of the chest-walls is frequent in streptococcal, but exceptional in pneumococcal, pleurisy (Netter).

In about a fourth of the cases, especially in the encysted forms, the termination is by **vomica**. It is much more frequent than in the other varieties of purulent pleurisy, and supervenes from the fifteenth to the thirtieth day. In other cases the pus is not evacuated by the bronchi, but forms a swelling in an intercostal space, or towards Scarpa's triangle. Lastly, it may become encysted, like an abscess, and be absorbed, especially in children.

Pneumococcal pleurisy is notably **less grave** than streptococcal pleurisy. It may recover by vomica or by absorption of the fluid, for the pneumococcus is not endowed with great vitality. After evacuation of the fluid, the lung quickly expands again. An attempt may be made to cure these cases, especially in children, by simple aspiration of the liquid, with or without aseptic lavage. The operation for empyema may sometimes be avoided.

In some cases metapneumonic purulent pleurisy is very grave, especially when the pneumococcus is associated with other pyogenic organisms, such as the streptococcus and the staphylococcus.

Although this holds good, it would not do to place too great confidence in the benign nature of the pneumococcus ; there are cases where, although the pneumococcus is **quite isolated**, its virulence is as great as that of the most active pyogenic germs. I treated a youth for a most severe attack of purulent pleurisy, in which the pneumococcus was the only microbe found. Such cases I have usually treated by aspiration. I performed a first and second puncture, hoping to avoid an operation for empyema ; but as the fever remained high, and the fluid reformed, with severe general symptoms, it was necessary to perform thoracotomy. I remember especially two cases of purulent pleurisy, due to the pneumococcus alone. I performed two successive punctures, without result, and, at my request, the patients were operated upon, with success.

All that I have said concerning metapneumonic purulent pleurisy applies to the primary cases which supervene without previous pneumonia.

In addition to the metapneumonic form, we also see early cases in which the effusion appears at the same time as the pneumonia. Lemoine has given them the name of "parapneumonic." We must not forget the cases of aseptic puriform pleurisy ; they must not be confused with the septic forms of pleurisy.

### Purulent Pleurisies due to Staphylococci.

"The *Staphylococcus pyogenes*, which plays such an important part in suppuration of the cellular tissue, the glands, and the bones, has only relatively a small share in the ætiology of pleural suppuration" (Netter). The staphylococci are more often associated with other microbes.

The rarity of purulent staphylococcal pleurisy does not permit us to give its history. It is, however, important to know that a staphylococcus has been found in effusions which have remained sero-fibrinous ; it has also been found in the fluid of sero-fibrinous pleurisies which have subsequently become purulent without the addition of fresh microbes.

Wounds and injuries may cause purulent pleurisy, due to the staphylococcus. The *Staphylococcus aureus* may spring from an osteo-myelitis, anthrax, or suppurative tonsillitis (Fraenkel). It chiefly invades the pleura of those who offer favourable soil, and are already victims to typhoid fever, scarlatina, Bright's disease, overwork, etc. We do not always find its point of entry into the organism, and we may ask if the microbe did not exist in a latent state, awaiting a favourable occasion for development.

### Tubercular Purulent Pleurisy.

We have already studied sero-fibrinous and hæmorrhagic tubercular pleurisy, and we may therefore turn to the purulent form. This form is due to the tubercle bacillus, without other associated microbes. If there were



other bacilli, the condition would be purulent pleurisy in a tubercular patient, but the pleurisy would not be tubercular in the true sense of the word. The tubercular form only comprises about a tenth of the cases of purulent pleurisy.

The fluid is sero-purulent, contains very little fibrin, and is dull greenish, with a powdery sediment. It has, then, none of the characters of the laudable pus of pneumococcal pleurisy. The purulent fluid, especially in old effusions, may be fatty and chyliform. Koch's bacillus may be discovered in the fluid. If the disease is not a pure infection, other pyogenic agents are found in varying numbers.

This form is rarely purulent at first; the fluid is often sero-fibrinous, but is particularly rich in red corpuscles and in lymphocytes. Later the fluid becomes turbid and sero-purulent. As the effusion reforms readily, and as puncture has at times to be repeated, the purulent change was formerly set down to thoracentesis, whereas the change is really due to the fact that the puncture has been made at different stages of the disease.

The chief point about this form of pleurisy is that it may be insidious, and pass through its phases without acute symptoms. It corresponds partly to the varieties formerly described under the name of chronic or latent purulent pleurisy, which may last for several months without notable change in the condition of the patient.

It is sometimes accompanied by perforation of the pleura (pyopneumothorax), but is rarely followed by vomica.

The diagnosis of tuberculosis, in the case of purulent pleurisy, is often difficult. I do not allude to those patients who have pulmonary tuberculosis, with or without pneumothorax, for the nature of the effusion is then evident, but I refer to purulent pleurisy due to local tuberculosis of the pleura. How is the pathogenic diagnosis to be made? The tubercle bacillus is looked for in the fluid, but is rarely found, and we must therefore have recourse to the means which I have indicated in the section on Sero-fibrinous Tubercular Pleurisy.

Inoculation of a small quantity of pus into the mammilla of a suckling guinea-pig has been performed by Nattan-Larrier in six of my cases. The bacilli pass into the milk after five to ten days, and show the tubercular nature of these cases.

Although I am of opinion that surgical intervention is necessary in other varieties of purulent pleurisy, I agree with most authors that the tubercular variety should usually be respected. Thoracotomy often gives bad results. The patient should be treated medically, and we should content ourselves by removing the excess of fluid when necessary by aspiratory punctures.

### Other Purulent Pleurisy.

Other microbes, such as *Micrococcus pyogenes tenuis* (Rosenbach), pneumo-bacillus of Friedlander, *Micrococcus tetragenus* (Netter), bacillus of typhoid fever (Rendu and De Gennes), the coli bacillus, may cause purulent pleurisy. Appendicular pleurisy will form the subject of a special section.

In many conditions several pathogenic agents are associated ; when these secondary infections are superadded they deprive the pure forms of their proper appearance—I might almost say of their specific nature. The result is multiple clinical types, without well-determined characters and course.

## VI. ASEPTIC PURIFORM EFFUSIONS INTO THE PLEURA— INTEGRITY OF THE POLYNUCLEAR CELLS.

**Puriform** effusion is not always synonymous with septic effusion, as we see clinically that turbid liquids may remain sterile throughout their whole course. Aseptic pus does not involve the grave prognosis of microbic pus ; this distinction must be carefully recognized.

Bacteriology shows the absence of micro-organisms in aseptic pus, but Widal has emphasized a special characteristic in the puriform fluid from the meninges or the pleura ; this characteristic is the integrity of the polynuclear cells. When the fluid is septic, the polynuclear cells are damaged in the fight against the microbes and their toxines, the nucleus and the protoplasm being altered. This result follows from Metchnikoff's laws of phagocytosis. In aseptic puriform fluids, on the other hand, the polynuclear cells show no change either in the nucleus or in the protoplasm.

In recent years we have seen several cases at the Hôtel-Dieu. I give a summary :

1. Woman admitted for apical pneumonia. Defervescence had set in, when a band of dullness was found at the right base. Aseptic serous fluid on puncture. Second puncture three days later, as effusion had increased ; puriform fluid drawn off. We might have thought of parapneumonic empyema, but the fluid was sterile. Numerous films showed that the polynuclear cells were unchanged. The fluid was absorbed with great rapidity, and four days later we had much difficulty in obtaining a few drops of fluid which had not become serous.

2. Man admitted for bastard pneumonia at the left base. Onset was sudden, with rigor and temperature of 105° F. Dullness, increased vocal fremitus, tubular breathing, and fine subcrepitant râles. The disease stopped short on the fifth day, and the temperature fell to normal. It rose again on the seventh day to 101° F., but the patient did not appear worse, and his breathing was not quickened. The râles at the left base disappeared, but distant tubular breathing was audible on expiration. Vocal fremitus abolished. Exploratory puncture drew off turbid fluid like that seen in acute rheumatism. The fluid was centrifugalized ; some macrophages and many intact polynuclear cells with granulations that took the triacid stain, were found. The



glycogenic reaction was very clear. Cultures were negative. The case was, therefore, one of aseptic puriform effusion.

Next day the effusion had almost disappeared, and two days later an exploratory puncture was negative.

Widal and Gougerot have found aseptic puriform effusions in the course of pneumonia, or after pulmonary infarcts of cardiac origin. In the case of pneumonia the integrity of the polynuclear cells prevents confusion between aseptic puriform effusion and septic effusion due to the pneumococcus; this fact has an important bearing on the course and the prognosis of the effusion. Thus, in one of their patients with pneumonia 4 ounces of turbid fluid were drawn off on the seventh day of the disease; the polynuclear cells were normal. The signs of effusion had disappeared by the tenth day, and two days later punctures were negative; the puriform fluid was aseptic. In another case purulent fluid, with normal polynuclear cells, was drawn off on the fourth day. On the ninth day some friction sounds were still audible, but disappeared a few days later.

The pleuritic fluid in cardiac cases is usually limpid in spite of the presence of endothelial and polynuclear cells, which indicate congestion of the subjacent lung. In asystole, however, we may find turbid fluid after the appearance of a centre of pulmonary apoplexy, which shows itself clinically by blood-stained sputum.

Aseptic puriform fluid has sometimes the yellowish colour of old pus, at other times the whitish tinge of recently formed pus. A fibrinous clot nearly always forms on standing. When the previous tinge was very opaque, the clot gives the liquid a puriform tint; when the tinge was simply whitish, the clot imprisons in its meshes nearly all the scattered cells; it then forms a milky-looking mass, which floats in clear fluid.

The homogeneous nature of the protoplasm, which is finely granular, and the clear outline of the nucleus, which is rounded and punched out, show clearly the integrity of the polynuclear cells. Staining with hæmatin-eosin, after fixation with alcohol and drying in the air, of the sediment smeared over a slide, gives the diagnosis.

The turbid appearance is due to the accumulation of polynuclears which have remained normal after passing out of the bloodvessels by diapedesis. The appearance of aseptic puriform pleurisy in cardiac cases with pulmonary apoplexy proves this congestive origin. The marked subpleural congestion which an infarct determines, causes in the bloodvessels stasis of the red corpuscles, which, being endowed with passive movement, can only pass out by effraction through stomata produced by the diapedesis of the white corpuscles. By reason of their amœboid movement the polynuclear cells pass through the capillary walls and the endothelial barrier, and fall intact into the pleural fluid, where they may collect in such numbers as to make the effusion turbid. The congestion is often so acute that it induces exudation of numerous red

corpuscles, or even rupture of capillaries ; the colour of the red corpuscles then effaces the white tinge given by the polynuclear cells when they are alone present in the liquid.

We see, in any case, that a kind of selection may be established between the walls of the capillaries and the endothelium of the pleura, and that, in a somewhat paradoxical manner, the **red** congestion of the parenchyma may end in a **white** exudate from the serosa.

Widal has thus explained the origin of these puriform effusions. The asepsis of an effusion which has developed near an infectious centre of pneumonia appears paradoxical at first sight, but it can be explained by the fact that the polynuclear cell may arise from the excessive hyperæmia of the subjacent tissues.

An aseptic effusion which develops near a patch of pneumonia is not specific, but only correlative to a pulmonary infection ; it is the result of peri-inflammatory congestion, and is only an evidence of a congestive condition, and must be carefully distinguished from the septic effusion caused by invasion of pneumococci, which damage the polynuclear cells of the defence.

We can experimentally produce aseptic white exudates similar to those which develop spontaneously in the human serous membranes. Amicrobic puriform effusions may be produced by injection of aseptic broth into the peritoneum of guinea-pigs. The turbidity of these experimental exudates is due to the accumulation of numerous polynuclear cells, which have so preserved their shape and functions that they can perform *in vitro* their usual phagocytic action. This method is used in the laboratory to collect living leucocytes.

A striking opposition is seen between the cause of aseptic puriform and purulent or sero-purulent microbic effusions. The former are as transitory as the subjacent congestion which causes them ; they disappear in a few days.

The published cases of purulent pleurisy, which has appeared in the course of pneumonia, and has been cured by simple puncture, like those cases of cerebro-spinal meningitis which seem to recover after lumbar puncture, are most often cases of aseptic suppuration in which the polynuclears are normal.

When we are studying the pus in a case of pleurisy, we must make both a bacteriological and also a cytological examination in order to ascertain the condition of the polynuclear cells. If we find intact leucocytes\* we know at once that the fluid is aseptic and the prognosis is benign.

\* The integrity of the polynuclears may be shown by the glycogenic reaction which was discovered by Ehrlich, and depends on the mahogany staining which iodine vapours cause in certain cells. It is most marked, as Loeper has shown (*Arch. de Médecine expérimentale*, Juillet, 1903), in leucocytes from cases of puriform pleurisy, and much less so in the altered cells from cases of empyema. In difficult cases the discovery of glycogenesis by the leucocytes in the exudate may be of service.



Too much attention cannot be paid to the examination of turbid effusions, which are puriform. We have no means of telling at first sight whether they are septic or aseptic. And yet this fact is the key of the situation. If a puriform effusion is aseptic, the prognosis is good—no risk of vomica exists, no need arises for surgical intervention, and rapid recovery follows.

On the other hand, if the puriform effusion is septic, it is more or less serious, and surgical intervention, instead of simple thoracentesis, is most often required.

## VII. PACHYPLEURITIS AND INEXHAUSTIBLE PLEURITIC EFFUSION.

For the purpose of this chapter, I have made use of my recent communication to the Académie de Médecine.

The pleura can, in a few days, produce again and again a large quantity of fluid. In ordinary cases of acute pleurisy it is not rare to see the effusion increase daily by several hundred grammes. Sometimes the fluid reforms so rapidly that, four or five days after thoracentesis, the pleura is again filled. I have frequently noticed this fact, and the following case, published by M. Graux, is a remarkable example thereof:—

A young married woman, who had just reached full term, was taken ill with right pleurisy, the effusion being so great that the dyspnœa threatened to terminate either by suffocation or by syncope. Thoracentesis was evidently urgent, but, as the woman was in the midst of her labour, no one dared operate, for fear of accidents. On the other hand, it was a question whether the accouchement could terminate well, for the patient's dyspnœa was such that any effort at expulsion seemed impossible. The situation was perplexing. Happily, the confinement took place in a few hours without any regrettable incident. Three days afterwards thoracentesis was performed, and slowly, at intervals, 5 litres of sero-fibrinous fluid were withdrawn. The patient was greatly relieved, and everything went well; but, by the fifth day, the pleura had filled again. Suffocation and syncope again threatened, and a second thoracentesis which yielded 5 litres of fluid, was performed. The result was similar to that obtained after the first puncture. There was a great improvement; but, in a few days, the liquid reformed, the pleura was once more full, fits of suffocation again appeared, and, for a third time, thoracentesis was performed. Four and a half litres of fluid were removed. From this date the pleuritic effusion was reproduced with such rapidity that, at more or less close intervals, it was necessary to make a series of punctures, so much so that, within two months, the woman had been tapped fifteen times, the pleura having, in so short a space of time, produced 40 litres of fluid. The pleurisy

was cured, but the subsequent appearance of pulmonary tuberculosis proved that this pleurisy was, really, tubercular in nature.

The pleura can produce, again and again, not only a very large quantity of fluid, but this pathological condition may turn into **pachypleuritis**, lasting twenty years, or even more. It seems as though the pleuritic effusion were inexhaustible, and, for this reason, I have called these cases cases of **inexhaustible pleuritic effusion**, as the following examples will prove :—

**Clinical Cases.**—On February 2, 1900, a man called Blanchet, thirty-five years of age, came to the Hôtel-Dieu to consult Brissaud. The patient who, up to that time, had enjoyed excellent health, was suffering from right pleurisy. Thoracentesis was performed, and a litre of, slightly, sanguinolent fluid was withdrawn. From this time forward, the pleuritic effusion reformed obstinately, and, during the course of the year 1900, thirteen punctures gave a total of 27 litres.\*

After the sixth puncture, the general condition had so much improved that the patient left the hospital, to go back to his business as a wine merchant. When he felt oppressed, he knew what was the matter, and came to the hospital to have a puncture made, and as soon as thoracentesis had been performed, he returned home, partook of a good meal, and went to work again.

Thinking that the case might interest me, Brissaud transferred the patient. I had noticed the existence of chronic pleurisy with an indefinite production of fluid, but I was unable to formulate anything precise as to the cause of the disease. When the effusion became troublesome, the patient came to my wards and asked for a puncture. He sat, straddle-legs, on a chair. A No. 2, or a No. 3 needle was used, and from 2 to 2½ litres of fluid were withdrawn. Immediately after the operation the patient went to breakfast, and then to business. Thus, during the year 1901, twelve tappings resulted in 32 litres of a fluid that was sometimes reddish, sometimes greenish. Our patient looked well, did not cough, and had no fever. His appetite was excellent, and all the organs were in good condition. Nothing caused us to suspect tuberculosis.

During 1902, there was no change, and during the course of the year eleven punctures were made and 27½ litres of a fluid, the colour of which varied from brown to greenish-grey, were withdrawn. The pleura was very much thickened (pachypleuritis), a fact that was rendered apparent by the sensation which was felt when pushing the needle deeply through the resisting and fibroid tissue.

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\* Punctures during the year 1900 :

|              |    |    |    |    |    |               |
|--------------|----|----|----|----|----|---------------|
| Feb. 5       | .. | .. | .. | .. | .. | 1·000 litre.  |
| Feb. 16      | .. | .. | .. | .. | .. | 1·500 litres. |
| March 3      | .. | .. | .. | .. | .. | 1·400 „       |
| March 24     | .. | .. | .. | .. | .. | 1·500 „       |
| April 28     | .. | .. | .. | .. | .. | 1·400 „       |
| May 25       | .. | .. | .. | .. | .. | 2·200 „       |
| June 12      | .. | .. | .. | .. | .. | 2·400 „       |
| July 14      | .. | .. | .. | .. | .. | 3·100 „       |
| Aug. 24      | .. | .. | .. | .. | .. | 3·250 „       |
| Sep. 19      | .. | .. | .. | .. | .. | 2·000 „       |
| Oct. 12      | .. | .. | .. | .. | .. | 2·000 „       |
| Nov. 5       | .. | .. | .. | .. | .. | 2·500 „       |
| Dec. 20      | .. | .. | .. | .. | .. | 2·650 „       |
| <hr/>        |    |    |    |    |    |               |
| 13 punctures |    |    |    |    |    | 27 litres.    |



In 1903, thoracentesis was performed twelve times and  $31\frac{1}{2}$  litres of a fluid which, almost always, had a purulent appearance were withdrawn.

In 1904, the quantity of the fluid was greater still. Thirteen punctures produced  $33\frac{1}{2}$  litres of purulent fluid, the odour of which was, at times, exceedingly foetid. It was peculiar that this pleurisy which had lasted for fifteen years had not affected the patient's health in the least. As far as strength and vigour were concerned, he could give points to his employés.

In 1905, twelve punctures were made, and  $30\frac{1}{2}$  litres of fluid were withdrawn. In 1906, ten punctures produced  $21\frac{1}{2}$  litres of fluid. In 1907, thoracentesis was performed only seven times, and the amount of fluid withdrawn did not exceed 11 litres. The fluid was always purulent and foetid. In 1908, only five punctures were made, and 10 litres of purulent fluid were withdrawn.

The improvement was still more manifest in 1909. Only four punctures were made, and only 4 litres of still purulent and foetid fluid were withdrawn. Auscultation showed that respiration was not abolished throughout the entire area of the right lung, and a radiograph, taken by M. Lacaille, confirmed the auscultation. The year 1910 commenced well. The five punctures gave 4 litres of fluid. Blanchet drew our attention to the fact that, for some time past, he could lie on his left as well as on his right side, whereas, for the past ten years, he had been able to sleep only on the side of the pleurisy. To sum up: Here was a man suffering from pachypleuritis, the fluid of which reformed with such tenacity that, in ten years, about 100 punctures had to be made, giving issue to 230 litres of fluid.\* This is more than the equivalent of a small barrel of Bordeaux.

The pleura must have been about 2 centimetres thick, judging by the depth to which the aspirating needle had to be thrust, and by the sensation of resistance experienced whilst the needle passed through the indurated tissue. In this fibroid carapace, incrustated perhaps with calcareous salts, a fluid was formed which, at times, was hæmorrhagic, at others purulent and very foetid.

The surprising thing was that the thoracic framework showed no deformity. Another surprising thing was that Blanchet kept in such excellent health. Here was a man who, in the past ten years, had undergone more than 100 operations for thoracentesis, his pleura having produced more than 230 litres of fluid. For years this fluid was purulent and foetid, and, in spite of all, the man remained robust and vigorous. He had but one symptom, want of breath, which came on when the pleural sac was too full. The sac being emptied, all went well.

**What was the nature of this strange pleurisy?** Was it aseptic, or was it virulent? Did it contain microbes, and if so, which were they? These points now demand our attention.

\* Punctures and amount of liquid withdrawn in 10 years:

|                    |    |    |    |    |              |
|--------------------|----|----|----|----|--------------|
| 1900, 13 punctures | .. | .. | .. | .. | 27.0 litres. |
| 1901, 12           | .. | .. | .. | .. | 32.0 "       |
| 1902, 11           | .. | .. | .. | .. | 27.5 "       |
| 1903, 12           | .. | .. | .. | .. | 31.5 "       |
| 1904, 13           | .. | .. | .. | .. | 33.5 "       |
| 1905, 12           | .. | .. | .. | .. | 30.0 "       |
| 1906, 10           | .. | .. | .. | .. | 21.5 "       |
| 1907, 7            | .. | .. | .. | .. | 11.0 "       |
| 1908, 5            | .. | .. | .. | .. | 10.0 "       |
| 1909, 4            | .. | .. | .. | .. | 4.0 "        |
| 1910, 5            | .. | .. | .. | .. | 4.0 "        |
| 104                |    |    |    |    | 232.0 "      |

For many years the fluid in this case had been studied by our chief laboratory assistants, Nattan-Larrier, Gaultier, Le Play, Sézary, etc. Here is the résumé of Sézary's most recent researches:

(1) Examination with the ultra-microscope.—The examination which is reproduced on the following Fig. shows corpuscles with Brownian movements, cells, and microbes.

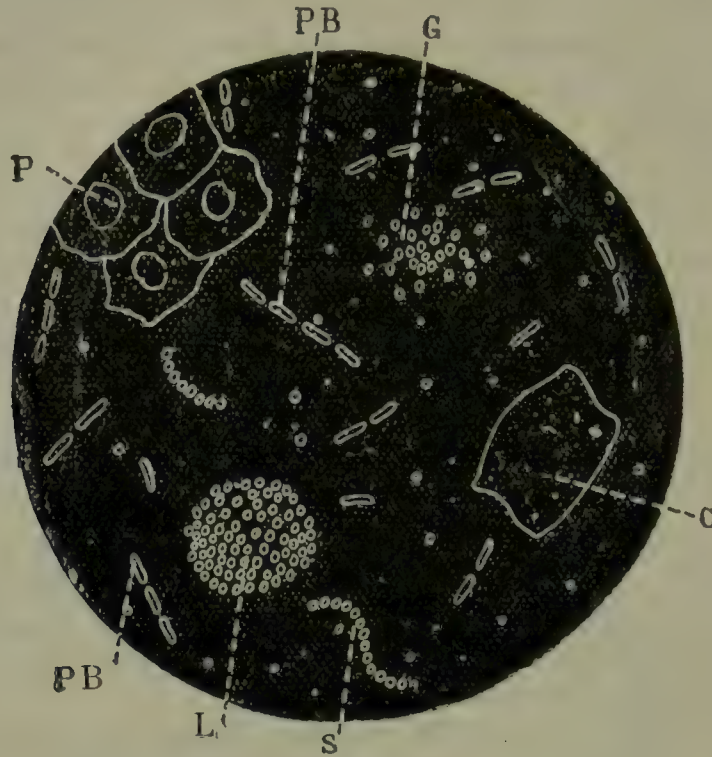


FIG. 18.

L, granular leucocyte; c, endothelial cells; P, patches of endothelial cells; s, streptococci; PB, pneumobacillus.

The cells were white corpuscles and endothelial cells. The white corpuscles (L) were crammed with refracting granulations which hid the nucleus. The endothelial cells were isolated, as at c, or united in flat patches, as at P. Their protoplasm was neither vacuolar nor granular. It sometimes contained microbes. We distinguish two varieties of microbes in the preparation. On the one hand, cocci, grouped in chains, which have the typical features of the streptococcus (s); on the other hand, bacilli of various forms, sometimes isolated, most frequently grouped in couples, or arranged in chains of three, four, and five elements (PB).

Lighted from the side, these microbes did not appear to be encapsuled, but their capsule showed up clearly in preparations stained with methylene blue.

**Cultures.**—The cultures were made on ærobic and on anærobic media. A. The ærobic cultures were made on inclined agar, in peptonized broth, and in milk. After twenty-four hours, there appeared, on the agar,



luxuriant rounded, yellowish-grey colonies, due to Friedlander's **pneumobacillus**.

Side by side with these luxuriant colonies, other punctiform ones were distinguished which were formed of typical **streptococci**. In the culture made with peptonized broth, a very marked disturbance was seen after twenty-four hours. Direct examination and fresh cultures showed only the two species previously mentioned. Inoculated milk did not coagulate.

B. Cultures in anærobic media. The culture of pus on deep glucose agar induced numerous gas bubbles in the tube. The bacilli of this culture were analogous to those which were found on aerobic agar, *i.e.* pneumobacilli. There was nothing surprising in this fact, as the pneumobacillus is aerobic and anærobic. The culture of pus in anærobic broth, covered with a layer of sterilized oil, also produced pneumobacilli.

To sum up: Aerobic and anærobic cultures made in this case demonstrated the presence of streptococci and pneumobacilli. Cases of pleurisy, due either to one or to the other of these microbes are, generally, virulent in nature. It is well known how often cases of streptococcic pleurisy are to be feared. It is also known that the pneumobacillus can produce purulent pleurisy which, clinically, greatly resembles pneumococcic pleurisy.

Why, then, in this case, did a double pleuritic infection (streptococcus and pneumobacillus) not occasion any of the complications which are so frequently present in purulent pleurisy? Why was no pyopneumothorax ever met with? Finally, how was it possible for such a case of pleurisy to evolve during such a large number of years without the patient feeling any effects of the toxi-infection.

The situation was paradoxical, and an attempt had to be made to explain it. The reason may, perhaps, be found in the **degree of virulence** of the pleurisy.

Let us examine the question.

It is well known that erysipelas, suppuration, and septicæmia are complications which, frequently, follow inoculation of a rabbit's ear by a virulent streptococcus. Now, in rabbits inoculated with this patient's pleuritic fluid, only a slight redness was visible in the ear, without any other lesion and without any other result. This proves that the virulence of the streptococcus was very much attenuated.

On the other hand, Friedlander's pneumobacillus is a microbe, the virulence of which often is very great. The mouse is, by choice, the reagent. The inoculation of cultures or of pathological exudate under the skin of the animal produces in two or three days a venous œdema at the point of inoculation. Death often results, and the microbe is found in the blood in the shape of a diplococcus or of a short and encapsuled

bacillus. The health of the mice, however, which were inoculated with the fluid from this case was in no wise affected.

These various experiments prove that the streptococcus and the pneumobacillus of this purulent pleurisy were microbes **the virulence of which was highly attenuated**. They did not affect the patient's health. We now understand why they failed to produce in the pleural cavity any process with an ulcerous or with a perforating tendency, and why they did not bring about vomica or pyopneumothorax. They were, however, by no means strangers in the work of defence which terminated in the formation of the pachypleuritis, a real carapace which surrounded the lung and which complicated the treatment in a singular manner, as we shall see in a moment.

Let us now refer to the second case of **pachypleuritis with inexhaustible pleuritic effusion**.

This was the case of a man named Baquia, fifty-nine years of age, who, twenty-one years previously, had had pleurisy on the right side. He consulted Mathieu, who ordered two successive punctures to be made. The patient felt so much better that he left the hospital. His health was, in truth, excellent. He followed his occupation without fatigue, but the pleuritic fluid reformed most persistently. He was thus obliged to visit the hospital several times a year, to get rid of this fluid. The puncture having been made, the patient returned home either on the same or on the following day.

The sero-fibrinous pleurisy lasted for a large number of years, without affecting the man's health. He had no fever, did not get thin, did not lose his strength. The only symptom which indicated that it was time for a puncture was that his difficulty in breathing increased gradually. Thus, in the space of eighteen years, thoracentesis was performed about 100 times.

Two years and a half ago, the fluid became sanguinolent. Since that date, he has returned at longer or at shorter intervals. Thoracentesis has been performed several times, and the fluid retains its hæmorrhagic tint. The pleura has become exceedingly thickened (pachypleuritis). As a proof, it is only necessary to note the depth to which the needle of the aspirator must be pushed to reach the fluid. There is, also, the sensation that the aspirating needle is traversing a very thick pleural shell. Here we have, then, a case of pleurisy lasting twenty years. For eighteen years it was sero-fibrinous, and for two and a half years it has been sanguinolent. Thoracentesis has been performed about 100 times, more than 200 litres of fluid have been withdrawn, and the system supports this strange emunctory without revolt.

On examining the patient, we were somewhat astonished to find that there was no deformity of the thoracic framework.

**What, then, was the nature of this pleurisy** which had lasted more than twenty years? Clinically, we found no sign, no symptom of tuberculosis. On the other hand, the researches of Messrs. Le Play and Sézary in the laboratory, repeated in several instances, gave no result. Cultures of the pleuritic fluid made in ærobic and in anærobic media remained sterile. Innoculations of the pleuritic fluid into the peritoneal cavity of guinea-pigs were negative. The ultra-microscope revealed no microbe in the pleuritic fluid. Intradermal injection gave no result whatever. All experiments



negatived the hypothesis of actual tuberculosis, and, on the other hand, in consequence of the absence of microbes in the fluid, it was impossible to state the nature of this pachypleuritis. To sum up: These two cases just quoted are remarkable examples of pachypleuritis with pleuritic effusion which, up to the present, is inexhaustible. The general state of health of the two men remains good, but the two cases are not comparable, seeing the nature of the fluid. In the first case, the pleurisy was purulent and foetid, its agents being the streptococcus and the pneumobacillus, but the highly attenuated virulence of these microbes explains the relative benignity of the disease. In the second case, the pleurisy was sero-fibrinous for eighteen years, and, for the last two and a half years, it has been sanguinolent. The fluid is absolutely sterile, and all attempts to discover the nature of the pleurisy have remained fruitless.

**Pathological Anatomy.**—The pleura of both patients must have been very thick. Probably it was incrustated with calcareous salts, such as are often met with during the course of pachypleuritis (Poulalion, Tuffier, Jardry and Gy). Letulle recently made a most important communication to the Académie on the pathological anatomy of pachypleuritis. We may sum up his conclusions as under:—

Every case of chronic pleurisy, characterized during life by a large, inveterate, or by a reiterated effusion, notwithstanding numerous thoracenteses, corresponds, as regards pathological anatomy, to **pachypleuritis**. Every case of pachypleuritis, the variable character of the effused fluid excepted, consists, essentially, of a diffuse, vegetating and fibroid inflammation of the two leaves of the great serous pleura. Cases of pachypleuritis fall into three categories: tubercular, infectious, or malignant. The cancerous variety results from pleuro-pulmonary cancer. In an excellent specimen of pachypleuritis, which M. Letulle was good enough to forward, it is seen that the pachypleuritis is generalized over the whole of the visceral and parietal pleura. Pulmonary collapse and the folds of the visceral pleura have been studied in detail by the author. This is his description: "Instead of remaining flat and smooth after the normal fashion, **the serous visceral membrane is folded**, whilst the subjacent pulmonary parenchyma, pushed back by the mass of the inflammatory fluid gradually effused in the pleural cavity and hindered in its inspiratory expansion, is heaped up and retracted towards the hilum of the lung (pulmonary collapse).

"All my observations have proved the constant existence of these **folds of the visceral pleura** on the surface of the parts of the lung in a state of collapse, and this is an interesting point which will shed light on the still obscure question of cases of inveterate pachypleuritis. It matters not whether the case be one of pleural tuberculosis, or of chronic vegetative

pleuritis, following pneumococcic or streptococcic infection. In all my cases, pulmonary collapse showed the same folding of the corresponding visceral pleura."

**Treatment.**—In a case of a thick and resisting pleura enfolding the lung in a rigid shell, the rational treatment appears to be to decorticate the lung, according to the method demonstrated to the Académie by M. Delorme in 1894 and again in 1907, when referring to the report on a successful case by M. Picqué.

The successful operations performed in some cases do not refer to patients such as Blanchet and Baquia. I am well aware that the treatment which these two men underwent has but little chance of ending in a cure, yet the improvement they made was clear from year to year. The fluid reappeared in smaller quantities and, by analogy, I refer to a similar case which did end in a cure.

On May 10, 1880, M. Potain, my chief, asked me to come and join him immediately at the bedside of a patient who had been sent to him from Nice. I found the patient suffering from such a violent attack of dyspnœa that he was threatened with asphyxia. The man was about fifty years of age and, for a long time, had had pleurisy on the left side, to which he had become more or less accustomed. He would not hear of an operation, and he led a fairly precarious existence, living and travelling about with his malady.

In view of the actual imminence of the danger, M. Potain requested me to perform thoracentesis on the spot. I made the puncture with a No. 3 needle and, before finding the fluid, which was deeply seated, I had to traverse such thick, indurated tissues that it was evident we had before us a case of pachypleuritis which had existed for a long time. I withdrew 2 litres of a purulent, odourless fluid.

The danger of asphyxiation was removed, but, a fortnight later, the fluid had reformed. I had to make another puncture, and, during the course of the year 1880, I performed thoracentesis six times, traversing a very thick pleural carapace on each occasion.

Thanks to this treatment, our patient felt well and, gradually, resumed his ordinary life. With such a pachypleuritis, there could be no question of an operation for empyema, and M. Potain and I were of opinion that it was best, *faute de mieux*, to continue thoracentesis as often as might be necessary.

|         |    |    |    |              |                           |
|---------|----|----|----|--------------|---------------------------|
| In 1881 | .. | .. | .. | 5 punctures. |                           |
| 1882    | .. | .. | .. | 4            | "                         |
| 1883    | .. | .. | .. | 3            | "                         |
| 1884    | .. | .. | .. | 2            | "                         |
| 1885    | .. | .. | .. | 1            | " which was the last one. |



The purulent fluid had dried up in six years, after 21 punctures; the patient had recovered and the thoracic framework was not even deformed. During the treatment, I did not wait for the effusion to assume considerable proportions before performing thoracentesis. I sought for the effusion and emptied it when it was still small. Under such circumstances, the lung may resume its functions more easily. In similar cases, it would also be useful to prescribe the respiratory gymnastics on which M. Delorme insists with so much reason.

### VIII. PULSATING EMPYEMA.

The question of **pulsating empyema** has given rise to numerous works.\* I have devoted two lectures to it, and Comby in his writings has called this disease pulsating empyema, or pulsating pleurisy. The former term appears to me preferable, for it indicates that the effusion is purulent. On careful search, however, exceptions might be found. Out of about sixty cases of pulsating effusions, three or four might be cited in which the fluid was not purulent, but, on closer scrutiny, some of these cases should be revised. The case reported by Rummo is one of hæmorrhagic pulsating pleurisy; a case reported by Comby is one of pulsating pleurisy, called serous, although the fluid on the second puncture was purulent. In one of Lépine's cases the fluid was serous, but slightly turbid. Piussan speaks of a patient in whom pulsations were discovered on auscultation of the thorax, and admits the existence of serous pleurisy; but "the diagnosis was uncertain, as thoracentesis was rejected." This case is not clear. It is evident, then, that these exceptional cases are of very little consequence: pulsating pleurisy, with serous fluid, is extremely rare; the fluid is nearly, if not always, purulent, so that the term "**pulsating empyema**" should be retained.

Another important fact is that pulsating empyema is always found on the **left** side; in sixty-nine cases, I know only one exception to this rule. We may therefore state that pulsating effusions, with very few exceptions, are purulent, and occupy the left pleura.

**Description.**—Every case has two periods—the one of purulent pleurisy, with or without pneumothorax, which lasts for weeks or months, and shows no sign of pulsation; and the other, in which pulsations occur, with or without an extrathoracic pulsating tumour.

Every left-sided empyema, whether it be tubercular, streptococcal, pneumococcal, etc., may finally pulsate; the pleurisy must, however, occupy the great pleural cavity, and be accompanied by much effusion. When the

\* Dieulafoy, *Clinique Médicale de l'Hôtel-Dieu*, 1898, p. 118.

effusion is scanty and encysted, we do not, as a rule, see pulsating empyema. In some cases the pleurisy has been preceded by pneumonia (my first patient); in others it is primary (my second patient); it is often consecutive to pneumothorax. Whether the pleurisy is primary or secondary, and its onset insidious or acute with fever and pain, there is a more or less lengthy stage, during which we find only the signs of a left effusion, with or without pneumothorax. These signs vary according to the quantity of fluid, and to the presence or the absence of pneumothorax. We find here, as in every case of effusion: dullness, diminution or abolition of vocal fremitus, absence of vesicular murmur, tubular breathing, ægophony, and sometimes also aphonic pectoriloquy and displacement of the heart (the pleurisy being on the left side). These signs are modified according to the amount of the effusion. If we add œdema of the chest-wall, which is frequent in purulent effusions, we have the picture of empyema before the appearance of pulsation.

During the first phase the general condition of the patient varies to a marked degree. In one case fever persists without remission, perspiration is abundant, appetite absent, and wasting rapid. In another case the effusion behaves like a cold abscess, without pain or reaction; fever is absent, or nearly so; the appetite remains good; the patient attends to his business, having but little distress, and only ceases doing so when the effusion is so large as to hamper the breathing.

Purulent pleurisy, as I have said above, is fairly often associated with pneumothorax. The latter may have formed insidiously, without dyspnœa or pain; or it may have been sudden, with pain and dyspnœa. However this may be, we recognize pyopneumothorax by the usual signs, including hippocratic succussion. The duration of the first stage is very variable, and the general condition of the patient depends upon many causes.

Let us come to the **second phase**. We find here the characteristic sign of **pulsation**. I must divide the description of pulsating empyema, for **an extrathoracic tumour** may or may not be present.

Let us first consider the first condition. On examining a patient with effusion, we are quite surprised, on auscultation or palpation, to find pulsations in some unusual area. Whether this area is behind, at the lower part of the chest, in front near the sternum, or at the side, we feel pulsation like that of a large aneurysm. When the hand is applied over the beating region, the pulsation shows itself by a shock, without a thrill. On auscultation, the beating is transmitted quite clearly to the ear, and is isochronous with the arterial pulse, though blowing and smacking sounds are absent. On careful inspection of the pulsating area, we generally see a movement of undulation, or of uplifting.

Many cases of pulsating empyema, **without** external tumour, have been published. I give a résumé of one of Traube's cases:



A young man admitted for left pleurisy. Costo-sternal plastron bulged ; chest-wall œdematous ; dullness complete, and heart much displaced to the right side. Further, the left side of chest, from the second to the sixth intercostal space, was raised up by pulsations that were isochronous with the carotid pulse. It was a case, therefore, of pulsating empyema, as the autopsy proved.

In this variety, I repeat, the beats of the pulsating empyema are perceptible on inspection, palpation, and auscultation, and may extend over a very large part of the thorax, but there is no tumour in the true sense of the word.

In the more common form of pulsating empyema, we find that the pus passes through an intercostal space, forms a prominence under the skin, and gives rise to **an expanded or rounded tumour**; this variety occurred in two patients at the Hôtel-Dieu.

A man, twenty-seven years of age, admitted for pneumonia of the left lung. Friction sounds were also heard. The expected crisis, however, did not occur. The pneumonic phase was succeeded by a fresh subinfrant one, with ill-defined signs. The patient was feverish, had night-sweats, and the case began to look like one of tuberculosis. The breathing was tubular, and the râles did not disappear. The idea of tubercular pneumonia was natural, and yet the expectoration contained no Koch's bacilli.

Dullness gradually became marked at the left base, and later in the left axilla. Normal respiration and vocal fremitus disappeared at these points. The patient had dyspnoea ; the heart was displaced to the right ; fluid began to collect ; and, although there was no œdema of the chest-wall, it was probable that the fluid was purulent. A new episode occurred. The patient felt sharp pain on the left side, near the sternum, which was increased by attacks of cough. Nothing could be seen, but on pressure the ribs were as painful as though affected by acute osteitis. During the next few days a swelling appeared, and formed a prominent tumour, about as large as the palm of the hand. The tumour showed expansile pulsation, and its beats were synchronous with the cardiac systole ; it might have been thought that a large aortic aneurysm had developed near the sternum. On auscultation, blowing and smacking sounds were not perceptible.

While the pulsating tumour enlarged, the effusion made progress. The whole diagnosis became clear : we had to deal with **pulsating empyema**, consecutive to metapneumonic purulent pleurisy. I punctured the tumour, and drew off homogeneous creamy pus, due to the pneumococcus. Bacteriological examination showed pneumococci, without other microbes ; cultures gave the same result, and inoculation of a mouse showed that the pus was not very virulent. The case, therefore, was pulsating empyema, with external tumour. The purulent collection in the pleura had made its way through the intercostal space, and formed a tumour at the upper part of the thorax. The ribs were resected, and some weeks later the patient was completely cured.

#### Résumé of the second case :

A man admitted to the Hôtel-Dieu for left pyopneumothorax, which contained much fluid and but little air. During the next few days the effusion increased in amount. As the fluid in the pleura increased, the succussion splash became less marked ; the dyspnoea increased ; the dullness occupied two-thirds of the chest, but œdema of the walls was absent ; and as the patient himself begged for puncture, I evacuated 2 pints of fluid. The pus was thick, greenish, homogeneous, and quite odourless ; it resembled pneumococcal pus, but microscopical examination and cultures showed the absence of the pneumococcus, and revealed the exclusive presence of the streptococcus

in small numbers. I tested the virulence of this streptococcus. A rabbit, injected with 5 centimetres, showed some reaction, but recovered completely; the streptococcus, therefore, was not virulent. Inoculation in guinea-pigs did not cause tuberculosis.

The patient improved, but the liquid gradually re-formed, and enlargement of the thorax, dullness, dyspnoea, and loss of appetite reappeared.

One morning he could not lie down on his left side because his ribs were so painful. We made him sit up in his bed, examined the left side, and, at the level of the ninth and tenth intercostal spaces found a small rounded swelling, which stood out like a hernia. It was not painful *per se*, but a deep pain, which reached back as far as the shoulder, was felt on firm compression. Our first idea was that the effusion was making its way through an intercostal space. Next day the tumour was more extensive, and reached as high as the eighth intercostal space. When it was examined by daylight, we saw slight pulsation, and also felt it on placing the hand lightly upon the surface of the tumour. There could now be no doubt as to the diagnosis: the patient was suffering from **pulsating empyema**.

During the next few days the pulsation became very clear. The tumour pulsated, but was not expansile; it was not reducible, but since pressure was very painful, we made no great attempt at reduction. I performed aspiration in the sixth space, above the tumour, and, as the pus was evacuated, the tumour diminished in size, proving that it communicated with the pleural cavity. Next day 4 more pints of fluid were drawn off; the pus was again homogeneous, greenish, and odourless. In consequence of this fresh evacuation, however, the pulsating tumour disappeared. After a few days' respite, the fluid formed again, and the pulsating tumour reappeared.

From the day of admission, I had asked myself whether surgical intervention should not be employed, because the condition was not, in my opinion, curable by simple puncture. I was afraid, too, that the great pleural cavity, which was so far but little affected, might become a centre of secondary infection. The patient, however, would not hear of an operation; the punctures were enough for him, and he would have left the hospital if I had tried to bring further pressure. I may add, also, that after mature reflection I did not feel confident as to the result of an operation, which would have been performed under bad conditions. The disease was of seven and a half months' standing; the lung would be reduced to a carnified adherent stump, surrounded by adhesions, and incapable of resuming its functions. How was it possible, even by removing a large number of ribs, for this immense cavity to fill up? and if the patient were tubercular, as appeared very probable, how would his economy stand such a drain on the system? Evidently, the wisest thing was to hold one's hand; and the patient made this decision easy, for he absolutely refused to hear of an operation. Palliative treatment was therefore resorted to, and aspiration performed every twelve or fourteen days.

As he wished to return to Venice, a final puncture was made, in order to put him in the best condition for his journey, and 4 pints of the same homogeneous greenish pus were drawn off.

In these cases, the interesting fact, from the pathogenic point of view, is that the patient had pneumothorax and pulsating empyema. In the one the empyema was of recent date, and associated with the pneumococcus; he recovered. In the other the empyema was of old date, and associated with a streptococcus of slight virulence. This man, who was probably tubercular, would have died, even if he had decided to undergo an operation.

We may now discuss the salient features of pulsating empyema. The passage of pus from the pleura into the intercostal space is sometimes



heralded by sharp pain. I am indeed surprised that this pain, which announces the exteriorization of pulsating empyema, has not attracted more attention. My second patient had very sharp pain in the seventh intercostal space before any tumour appeared externally. In my first patient the pains were still more acute, preceded the subclavicular induration by three days, and were so severe that they simulated acute osteoperiostitis. Injections of morphia and fomentations night and day were necessary to relieve these pains. The appearance of the purulent pocket shows itself, then—at least, in the cases which I have seen—by pain. The tumour does not always pulsate as soon as it forms : in the second case pulsations did not appear until the third day, and only became manifest on the fourth day in the first case.

The **pulsating tumour** is sometimes spread out, at other times rounded ; and, when once formed, may be the size of a nut, of an orange, or larger. In some cases it resembles an aneurysm, and shows expansile pulsation ; on grasping it, it feels just like a large aneurysm. The expansile movements of the tumour, though doubted by some authors, are nevertheless marked in most cases ; they were fairly clear in my second, and undeniable in my first patient. I am therefore of Bouveret's opinion : " Though an intrapleural pulsating empyema presents scarcely any lifting movements, an extrapleural one shows true expansile pulsation. It is quite comparable to the pulsation of an aneurysm. With each beat of the heart the whole surface of the tumour dilates suddenly, and beats, not only vertically, but also transversely."

In some cases the tumour is reducible, and we can squeeze the pus back into the pleural cavity. Auscultation reveals neither blowing nor smacking sounds.

The tumour may develop at different points, either behind or in front of the chest, but its usual situation is to the left of the sternum, at the level of the third and fourth space. This was the case in my first patient, and the same localization was found in a case reported by Bérard.

A young man, after an acute disease, noticed a tumour developing rapidly in the front of the chest, at the **left of the sternum**. This tumour showed movements of expansion and contraction synchronous with those of the pulse. The apex-beat was under the right clavicle. Everyone thought of an aneurysm, and awaited, with dread the moment when the thin skin would burst and give rise to fulminant hæmorrhage.

The patient, who was ignorant of his condition, impatiently awaited the rupture of " his abscess," and, wishing to hasten the work of Nature, he plunged a pin into the apex of the tumour. Pus came out in a small quantity at first, and later in abundance, when ulceration had enlarged the wound. I have seen him since, enjoying the best of health.

In this case it seems likely that the pulsating empyema was consecutive to pneumococcal pleurisy.

The tumour of pulsating empyema may reach the lumbar region, as in Courbon's case :

A young woman was admitted for tumour in the left lumbar region. It was situated just outside the quadratus lumborum, immediately below the last rib. It was elongated, fluctuating, but not painful, and was almost entirely reducible ; there was no change in the colour of the skin. The swelling had the form of an ellipse with a large axis. The noticeable feature, however, was the beats of the tumour, which were expansile and synchronous with the pulse. The whole tumour showed alternate dilatation and contraction, perceptible not only to touch, but also to sight. Palpation revealed no thrill, and auscultation gave no blowing murmur. The patient had noticed this tumour for two months ; for four years her health had been imperfect, and she had experienced pain over the left dorsal vertebræ, radiating to the left side of the thorax. Dyspnoea, palpitations, and attacks of suffocation came on as soon as the patient worked more than usual or walked upstairs.

Some months after the onset of these troubles she was in hospital at Blois for nearly a year. When Courbon saw her, her heart was pushed over to the right, and the apex-beat was beneath the xiphoid cartilage. The left side of the chest was distended, absolutely dull all over, and silent on auscultation. Courbon hesitated between an abscess of the pleura, pointing in the lumbar region, and an aneurysm of the descending aorta. He finally adopted the latter opinion. The tumour increased in size, and the pulsation became so pronounced that those who had hesitated admitted the aneurysm. The thin skin finally burst, giving exit to a large quantity of pus ; the tumour at once diminished in size, the pulsations disappeared, and the dullness on the left side diminished. It was, in short, a case of pulsating empyema.

In the following case, reported by Millard, a pulsating empyema, which simulated an aneurysm, formed a tumour in the left dorsal region.

A man, thirty-six years of age, of tubercular stock, and subject to bronchitis, was taken ill in May, 1887, with left pleurisy, complicated by pneumothorax. He made an indifferent recovery. Some months later dyspnoea and fresh signs of pneumothorax. In July, 1888, a tumour, which rapidly reached the size of an orange, appeared to the left of the vertebral column, at the level of the last rib. The swelling showed pulsations, synchronous with the pulse. His doctor diagnosed an aneurysm of the descending aorta. Millard, however, recognized the existence of pulsating empyema, forming an external tumour. He admitted the patient into his ward, and punctured the tumour, drawing off 7 pints of creamy, inodorous pus. The swelling became smaller, the pulsations at once disappeared, and the patient gained much relief. Pleurotomy did not appear to be indicated, for the disease was of fifteen months' standing, and Millard preferred to resort to intrapleural injections of tincture of iodine. After transient improvement, the situation became worse. Peyrot resected the ninth rib, and drew off about 6 pints of foetid pus. The cavity was washed out several times a day, but the patient became rapidly feeble, and succumbed to hectic fever, with colliquative diarrhoea.

I have just described two varieties of pulsating empyema. In the first the pulsations, though felt externally, remain intrathoracic, and no external tumour forms ; in the second variety the empyema projects under the skin, through an intercostal perforation, and forms a pulsating tumour, which usually appears at the left of the sternum.

Pulsating empyema may persist for a long time without spontaneous opening of the collection ; vomica has, however, been noted in some cases.



The purulent collection opens into the bronchi, and the vomica differs in no respect from that consecutive to purulent pleurisies.

**Pathogenesis.**—Such are the clinical facts. We must now discuss the pathogenesis, but I shall be brief on this question, which is not yet elucidated. I have already said that sero-fibrinous pleurisy, no matter how abundant the effusion may be, never pulsates. I have seen hundreds of cases, and studied them closely—as aspiration has particularly attracted my attention—for nearly thirty years, but I have never seen pulsation at any stage of sero-fibrinous pleurisy. Purulent pleurisy is practically the only form which shows pulsation; when it does, it is not because the fluid is purulent, but because the lesions of neighbouring organs favour the transmission of the cardiac pulsation.

According to Féréol, pneumothorax is the essential factor: “Pulsating empyema can be produced only when pneumothorax of small extent and large effusion, with displacement of the heart, are present. It is the elasticity of a compressible gas which allows this movement of expansion and contraction.” Féréol’s theory is shattered by the fact that in many cases of pulsating empyema (as in my first patient) pneumothorax may be absent. Comby gives another theory: “The airless and compressed lung plays the part of a sounding-board”; but the author declares himself “ready to abandon this theory for a better one.” Other authors—Rummo, Keppler, Traube—invoke multiple causes: intrapleural pressure, adhesions, paresis of the intercostal muscles, exaggeration of the cardiac beats, etc.; but all these theories are so open to criticism that we are not absolutely clear on the pathogenesis of pulsating empyema.

**Diagnosis.**—How can pulsating empyema be recognized and distinguished from the diseases which may simulate it? The diagnosis must be made from other pulsating tumours in the chest. When the pulsation is in the first intercostal space to the left of the sternum, the case closely simulates aneurysm of the aorta, causing an external projection; expansile pulsation, perceptible to sight and touch, exists in both cases. Certain differential signs, however, exist. In empyema the pulsating tumour appears in a few days, almost from one day to the next; *e.g.*, in my cases the tumour formed showed pulsations on the fourth day. In aortic aneurysm the tumour takes twenty times as long to appear; it requires months for the rarefying osteitis which eats away the ribs, to allow the aneurysm to project externally. In the second place, a patient suffering from pulsating empyema, whatever be the situation of the tumour, always has a large left pleural effusion, with or without pneumothorax. The signs of this large effusion, including **displacement of the heart to the right side**, are evident. In the case of aneurysm, large pleural effusion and deviation of the heart to the right are not found. Lastly, the murmurs which are heard over the

aneurysmal sac are not found in the case of pulsating empyema. It is evident, then, that though the pulsations produce some analogy between aneurysm and pulsating empyema, methodical examination will clear up the diagnosis.

**Pulsating abscesses**, which have nothing in common with pulsating empyema, may be met with in the chest. Costal or costo-sternal osteitis may give rise to an ossifluent abscess, like a shirt-button, which develops, on the one hand, beneath the tissues which it lifts up, and, on the other hand, beneath the parietal pleura which it pushes in. This subpleural collection does not involve the pleural cavity, but it may nevertheless be placed in such a position that the heart-beat is transmitted to the external collection of pus which bulges under the skin, and the tumour acquires expansile pulsation, comparable to that of empyema. We are able to distinguish these pulsating abscesses from empyema, because they are not accompanied by signs of pleural effusion. We do not find extensive dullness, absence of thoracic vibrations, deviation of the heart to the right, and the other signs of left purulent pleurisy, with or without pneumothorax.

It is not sufficient to diagnose pulsating empyema ; we must also ascertain the cause, since prognosis and treatment are equally concerned. As a general rule, the recognition of pneumothorax in a case of pulsating empyema is a sure sign of tuberculosis, excepting, of course, pneumothorax consecutive to vomica or to operation. The search for bacilli in the sputum should never be neglected ; their absence, however, does not always eliminate tuberculosis. Recourse may be made to injections of very small doses of tuberculin (Grasset's method) ; but however carefully tuberculin be used, we may sometimes see regrettable incidents, and I would advise it only in case of absolute necessity.

The prognosis of pulsating empyema is not equally serious in all cases ; the gravity chiefly depends on the cause of the disease. Pulsating empyema which supervenes in a tubercular patient, with or without pneumothorax, is the more serious, because surgical intervention leaves little to hope for. It is often, indeed, contraindicated, and we must be content with repeated punctures. On the other hand, in pulsating empyema which supervenes in an individual suffering from pneumococcal pleurisy, the prognosis is less grave, because surgical intervention gives good results.

**Treatment.**—Pulsating empyema demands surgical intervention. Repeated punctures, with or without injections of antiseptic fluid, are purely palliative ; they prolong the existence and relieve the patient, but they do not cure him. In some cases (pneumococcal pleurisy) pleurotomy, without costal resection, might perhaps effect a cure, but I should have but little confidence in it ; most often it is necessary to resort to thoracotomy, with free resection of ribs. This latter procedure saved my patient.



## IX. INTERLOBAR PLEURISY—VOMICÆ—INTERLOBAR HÆMOPTYSES.

**Interlobar pleurisy** develops, and becomes encysted between the lobes of the lung. The lungs are divided into five lobes—two in the left, three in the right lung. When the lung is examined, after removal from the thorax, we see that the surface is furrowed by fissures which penetrate deeply, as far as the hilum, and divide it into distinct lobes, which are isolated, and, as it were, suspended from the large bronchi.

On the left side there is only one interlobar fissure, which begins behind, about 3 inches below the apex, round which it passes, descending very obliquely downwards and outwards as far as the base. The left lung is thus divided into two lobes—an upper and a much larger lower one. On the right side the interlobar fissure has the same origin as that on the left, but it soon bifurcates, forming a kind of cross at its bifurcation. One branch continues an almost horizontal course, while the other passes round the lung and descends obliquely outwards as far as the base. The right lung is thus divided into three lobes, the middle lobe being the smallest and the lower one the largest.

The visceral pleura enters these fissures, and covers the corresponding surfaces of the lobes of the lung; for this reason, it has been called the interlobar pleura. When adhesions unite the edges of a fissure, and the interlobar pleura no longer communicates with the pleural cavity, we find an interlobar sac, which favours the formation of encysted pleurisy. The sac nearly always contains pus, as Laënnec has shown, and on section simulates at first sight a collection in the lung.

Interlobar pleurisy may be very extensive, and occupy the whole of an interlobar space; in such a case the neighbouring lobes are much compressed, and the encysted fluid amounts to 2 or more pints. In some cases, on the contrary, adhesions partition off the interlobar cavity, and the encysted fluid does not exceed some 6 or 8 ounces. Indeed, in a recent autopsy we happened to find several small interlobar abscesses which contained only a few drachms of pus. It is evident, then, that every intermediate form may occur between interlobar pleurisy with much effusion and small interlobar abscesses.

The topography of interlobar pleurisy is very variable. The following plates show the situation and the relation of the interlobar fissures in the normal state. The first plate shows the lungs from their posterior aspect—the left lung with its single interlobar fissure, and the right one with its bifurcated fissure.

The second plate shows the outer surfaces: the fissure of the left lung, which is almost horizontal looked at from behind, now descends obliquely;

and the fissure of the right lung, which is almost horizontal seen from behind, bifurcates, and of its two branches, viewed laterally, the one is horizontal, the other is oblique.

Interlobar pleurisy is not always localized at the back of the chest to the horizontal fissure; it sometimes occupies the axilla at the level of the oblique fissure, and may descend as far as the diaphragmatic pleura.

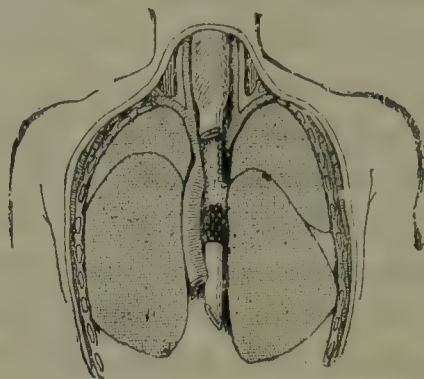


FIG. 19.—POSTERIOR VIEW OF THE LUNGS.

**Description.**—The interlobar form may be primary or secondary to pneumonia. In the latter case, if the pleurisy follows the pneumonic infection closely, or if it appears in the course of pneumonia, it is said to be parapneumonic; if it does not appear till some days or weeks after pneumonia, it is said to be post- or metapneumonic. However paradoxical the fact may appear, I think that interlobar pleurisy is more often primary than secondary. It develops on its own account—or, **at least, apparently so**—whether the infection is due to the pneumococcus or to other microbes. In none of the eight cases which I have quoted in my lectures at the Hôtel-Dieu\* did

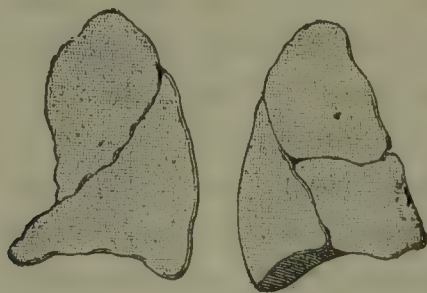


FIG. 20.—THE LOBES AND FISSURES OF THE LUNGS.

interlobar pleurisy appear to follow pneumonia, and, as far as my patients were concerned, I am quite certain that there was no pneumonia, the condition of the lung having been verified at the operation. Moreover, primary infection of the serous membrane should cause no surprise, as we know of numerous examples. Cases of primary peritonitis, pleurisy, and meningitis are at the

\* Dieulafoy, "La Pleurésie Interlobaire" (*Clinique Médicale de l'Hôtel-Dieu*, 1<sup>re</sup> et 2<sup>me</sup> leçons, 1899).



present day counted by dozens ; interlobar pleurisy is of the number, and appears to me to begin in most cases as a **primary** infection.

The present description will not, then, apply only to interlobar pleurisy preceded by pneumonia, but will especially deal with primary interlobar pleurisy. Nothing is more difficult to make out than the onset of interlobar pleurisy. Pain in the side, fever, and cough indicate that acute chest trouble is in progress ; but when we have to decide the nature and the seat of this trouble, we have no exact sign to guide us. Lobar pneumonia and pleurisy of the great pleural cavity are very different. In pneumonia, rigor, crepitant râles, tubular breathing, and rusty sputum indicate the diagnosis. In pleurisy, friction sounds, and signs of effusion—*i.e.*, dullness, disappearance of vocal fremitus, distant tubular breathing, ægophony, etc.—show us the evolution of pleurisy. If the case is one of inflammation of the chest, the **bilateral position** of the signs : bronchitic râles, scattered over both sides of the chest ; friction sounds, which bear witness to the participation of the pleura ; and tubular breathing, which shows the participation of the lung, lead us to diagnose inflammation of the chest.

The onset of interlobar pleurisy is very different. The infection in encysted pleurisy is, to a slight degree, comparable to that of a **closed cavity**, adhesions having cut off all communication with the great pleural cavity. The parts of the lung near to the infectious focus feel the effect.

The congestion of the lung shows itself by signs which we shall study, and, at the same time, masks the nascent state of the interlobar lesion.

During the first days of interlobar pleurisy examine your patient carefully to see if he has fever, cough, and pain in the side. This is quite commonplace. Percuss and auscultate the chest. You find, perhaps behind, perhaps in the axilla, over a vaguely limited region, slight dullness, râles, tubular breathing—*i.e.*, pulmonary and not pleural signs ; sometimes also a few erratic sounds. Nothing is found on the other side of the chest—no tubular breathing, no râles ; the lesion is usually **unilateral**. Being unable, at this moment, to make an exact diagnosis, you feel sure that it is a case neither of true pneumonia nor of true pleurisy, and you prescribe expectant treatment such as cupping, antipyrin, sedative draughts.

During the next few days the same uncertainty of the physical signs persists. The lesion remains unilateral ; the pain in the side is persistent ; the cough is more or less marked ; expectoration is absent or scanty ; fever acute, and dyspnoea intense. Time passes, and the diagnosis still remains in doubt. Whether the tubular breathing be more or less loud, the râles more or less extensive, the dullness more or less marked, matters little. You have always to consider the share of the lung and the share of the pleura in this obscure disease, which, for want of a better term, you name “pleuro-congestion.”

As fever and dyspnoea are present, you have some apprehension, and ask yourself if this lesion, of bastard appearance, may not be tubercular. The tenth or the twelfth day of the disease passes, and defervescence does not occur.

The picture then undergoes a change. If the fluid in the interlobar fissure reaches 8 ounces in an adult or 5 ounces in a child, percussion reveals a dull zone, **suspended, as it were, between two more resonant areas**, either towards the middle and posterior part of the chest or in the axilla. The idea of an encysted pleurisy now gains ground in your mind, and you make that diagnosis.

Anatomical researches have accurately described the normal topography of the interlobar fissures, and their relations with the intercostal spaces and thoracic wall (Rochard). The topography of the normal state is, however, no longer rigorously true in the pathological state. The situation and the direction of the fissures are modified by the fluid which has accumulated between the two lobes of the lung, and by the pushing back of these lobes.

It is, however, not exactly at the level of the interlobar fissures that the suspended zone of dullness is found. This zone may exist at different parts of the thorax. If interlobar pleurisy develops in the horizontal portion of the fissure, the zone is situated behind, at the upper third or the middle part of the chest. If interlobar pleurisy invades the oblique and descending portion of the fissure, the zone is found in the axilla, and extends as far as the anterior part of the chest.

During the first period of interlobar pleurisy the chief points are the artificial pulmonary signs, râles, tubular breathing, and dullness; when the effusion is fairly abundant, the chief sign is the existence of a dull zone, suspended between more resonant areas. The diagnosis is almost impossible in the first phase of the disease. We cannot say what the issue will be; we may think of pneumonia, pleurisy, or congestion, but we remain in doubt. For several days the chief symptoms are pain, cough, and dyspnoea; the interlobar effusion, surrounded by the lung, does not yet reveal itself by any special sign; on the contrary, the contiguous parts of the engorged lung give rise to tubular breathing and râles, which attract attention to the lung, while the abscess is hidden in the interlobar spaces. One sign, however, is of great value—viz., the limitation of a dull zone, surrounded by more resonant regions; this zone of dullness, however, is only evident when the interlobar fluid is sufficiently abundant.

In interlobar effusion we must not expect to find the classical signs of effusion into the great pleural cavity. In the latter case the fluid is in contact with the chest-wall, and therefore the physical signs reach their maximum. On the other hand, when the effusion is hidden in the interlobar cavity, the parts of the lung interposed between the effusion and the chest-wall make the signs feeble, and mask them.



The fluid in right interlobar pleurisy cannot push the liver down, but effusion on the left side, especially when the mischief invades the descending fissure, may displace the heart to the right.

Dyspnœa in cases of interlobar pleurisy is more marked than in pleurisy of the great pleural cavity. We may have 4 pints or more in the pleura, without much distress. Interlobar pleurisy, though quite limited, causes early and severe dyspnœa. The clinical picture, then, is very different between pleurisy of the great pleural cavity and interlobar pleurisy, and we should be liable to make grave mistakes if we were to suppose that the same signs are applicable to both cases.

Among the possible symptoms of interlobar pleurisy, there are two—**hæmoptysis** and **vomica**—which I wish to discuss in detail.

**Interlobar Hæmoptysis.**—**Hæmoptysis** has been noted several times in the course of interlobar pleurisy, and I saw two cases of this during the past few years.

In Prengrueber and Beurmann's case a young girl had repeated hæmoptysis, although she was not tubercular, in the course of interlobar pleurisy, for which she had been operated upon and cured. A youth whose case was reported by Letulle and Segond had **very abundant hæmoptysis**, which lasted a fortnight, although he was not tubercular. He was cured by surgical intervention. In one of Letulle's cases, a patient suffering from interlobar pleurisy was also attacked by hæmoptysis, which led to a suspicion of caseous pneumonia. The future proved that the patient was not tubercular, and surgical intervention effected a cure.

The following case has been communicated to me by Dr. Thalís :

A man, forty-five years of age, convalescent from left pneumonia, was taken ill with fever, pain in the side, and acute dyspnœa. Some days later a zone of dullness, surrounded by râles, was found at the middle and posterior part of the chest. For about three weeks the chief symptoms were fever, cough, dyspnœa, pain, and tachycardia. One morning the patient, after severe fits of coughing, brought up a quantity of pus ; he had just emptied an interlobar pleurisy. He continued every morning to fill one or two sputum-glasses with purulent expectoration, after which the cough was less severe. This condition lasted from four to five weeks, and the fever disappeared, but the tachycardia persisted. One day he brought up some bloody sputum, followed by abundant hæmoptysis. Tuberculosis was feared, but no bacilli were found, either in the purulent expectoration or in the blood coughed up. Convalescence was slow ; the expectoration only dried up after four months, yet the man completely recovered his health.

Hæmoptysis, then, is not rare in the course of interlobar pleurisy. This fact has not passed entirely unnoticed, for Pailhas has mentioned it and discussed its causes. When hæmoptysis supervenes in a case of pleurisy, with fever and purulent expectoration, we fear that the patient is tubercular. We must be on our guard against such an interpretation. The four patients whose history I have just reported recovered completely : they were not tubercular. I dwell on hæmoptysis in interlobar pleurisy because it is not sufficiently known ; we see, not simply some blood-stained sputum, but

true and sometimes abundant hæmoptysis, which may recur at varying intervals. Sometimes the spitting of blood supervenes after the vomica, as in Thalís's case; at other times it precedes or is contemporaneous with it, as in the three other patients. I think that this hæmoptysis is due to ulceration of the walls of the interlobar cavity. This process, which causes ulceration of a bronchus and vomica, may also cause ulceration of a vessel and hæmoptysis. In my little patient who was operated on by Tuffier (second case in my clinical lecture), it was easy for us to see the section of the bronchiole by which the vomica had been caused at the bottom of the interlobar cavity. If the accompanying arteriole had participated in the ulceration, hæmoptysis would have been the result. If hæmoptysis be relatively frequent in interlobar pleurisy, and almost unknown in the purulent pleurisy of the great pleural cavity, it is because the ulceration, which ends in vomica and hæmoptysis, is more active in interlobar pleurisy, which produces, as regards the pleura, a "closed cavity," with its consequences.

Moreover, whatever may be the pathogenesis of this hæmoptysis, which I propose to call **interlobar**, we must be on our guard against the conclusion that the hæmorrhage is due to tuberculosis.

**Interlobar Vomica.**—Let us now deal with an important symptom in interlobar pleurisy. I refer to the **vomica**. Generally speaking, the opening of the purulent focus is much more frequent in encysted effusions than in pleurisy of the great pleural cavity. The latter lasts months and years, without going on to vomica.

I was called to see a young girl who could neither walk quickly nor go upstairs without severe dyspnœa. Her respiration was embarrassed even during rest. The examination of the patient was neither complicated nor difficult. I at once discovered 6 or 7 pints of fluid in the left pleura.

I then learnt that she had had pleuro-pneumonia in Brazil a year before. From that time her respiration had been panting, and she had felt her heart beating on the right side; but yet she had travelled across Europe, thinking that she was "suffering from anæmia." Although I found neither fever nor cedema of the chest-wall, I thought that pleurisy of at least a year's duration must be purulent. I made several punctures. The puriform fluid, separated into two layers—the lower one thick, the upper one almost serous. The fluid was absolutely sterile, and very poor in pus cells. The pleurisy was evidently quiescent. She recovered after the fifth puncture. This large empyema, which had lasted for a year, had not gone to vomica.

I saw an American who for a long while had suffered from purulent pleurisy, and who, in spite of a large effusion, had several times crossed between France and America. After each voyage I drew off a pint of purulent fluid. I do not know what became of this patient, but I do know that purulent fluid had remained several years in the pleura without giving rise to a vomica.

Guéneau de Mussy relates the history of a patient who suffered from left pleural effusion for fifteen years. The first puncture brought away  $3\frac{1}{2}$  pints of puriform fluid, which did not contain a single leucocyte, and might have been an emulsion of fatty matter. A fortnight later  $3\frac{1}{4}$  pints of similar fluid were drawn off, and the patient experienced such relief that he went to Russia. This empyema, which had lasted for so many years, had never gone on to a vomica.



Guéneau de Mussy also quotes the case of a young man who suffered for eighteen months from empyema on the left side. The patient was punctured, and 5 pints of purulent fluid were withdrawn. Two fresh punctures were made, some weeks apart, and the improvement was such that the patient was considered cured. This empyema had not provoked a vomica, although it was of eighteen months' duration.

Some effusions of the great pleural cavity may last months and years, without going on to perforation of the lung and vomica. It is not so in encysted effusions. Whether the case be one of mediastinal, interlobar, or diaphragmatic pleurisy, encystment, after the fashion of closed cavities, favours ulceration of the walls, perforation, and vomica. Several patients have come under my care at the Hôtel-Dieu during the past few years with encysted mediastinal or interlobar pleurisy, which has usually ended in vomica. Eighteen cases of interlobar pleurisy, reported in my clinical lectures, gave rise to vomicae.

To sum up : Vomica is almost the rule in purulent interlobar pleurisy, if it is not operated on in time ; in that of the great pleural cavity it is the exception. Potain also holds this opinion.

The time at which the interlobar vomica supervenes is somewhat variable. It appeared on the eighteenth day of the disease in my second case, and before the fifteenth day in my first patient ; in other cases it was later ; but, speaking generally, the vomica of interlobar pleurisy is earlier than that of empyema of the great pleural cavity.

I have several times been able to predict the vomica twenty-four and thirty-six hours in advance, thanks to the sign which I have long since described, and which **depends on the foetor of the breath**. The fluid in interlobar pleurisy is nearly always foetid. Before the opening into the bronchi is large enough to allow free passage of the pus, a simple fissure is formed, and the foetid emanations of the cavity escape through this narrow strait. Foetor of the breath, therefore, may precede the appearance of the vomica by one or by several days. Our patient in the Saint Christophe ward had foetor of the breath and a " bad taste in the mouth " several days before his vomica ; his wife had remarked that his " breath smelt bad." The mother of the little girl in my second case told us, two days before the vomica, " that the child's breath had become faint and bad-smelling." Let us not forget this sign, which allows us to foresee the vomica, and aids us in the diagnosis of interlobar pleurisy.

The vomica does not always appear in the same manner. When the pus bursts into the bronchi, the patient is seized with fits of coughing and acute dyspnœa, and though **so far he has brought up no sputum**, he now coughs up purulent sputum, which is foetid and sometimes streaked with blood. The quantity of pus varies, according as the vomica follows empyema of the great pleural cavity or encysted pleurisy. In the former case, when

the perforation is large enough, abundant pus is brought up; the patient, indeed, appears to vomit pus—whence the word “*vomica*” (*vomere*, to vomit). He may bring up 1 or 2 pints in a few minutes. In encysted effusion which contains only a few ounces (interlobar pleurisy), the pus is not brought up in streams, but in small mouthfuls, so that the *vomica* might in some cases be taken for simple bronchial expectoration.

I cannot too strongly emphasize these **little fragmentary vomicæ which simulate bronchorrhœa**, and are much more frequent than large vomicæ in partial pleurisy. A patient tells you that for some time past he has had fits of coughing, followed by muco-purulent sputum. For want of care, you take the condition for bronchial catarrh, foetid bronchitis, or bronchorrhœa; but if you push your investigations farther, and obtain exact answers from your patient, you will learn that the expectoration in question began suddenly after severe fits of cough and dyspnœa, although on the previous days expectoration was absent. Push your inquiries farther still, and the patient will tell you that for some weeks before the onset of this foetid expectoration he had pain in the side, fever, cough, and dyspnœa; he had been obliged to keep his bed, and had been treated for an ill-defined thoracic affection. In short, he had interlobar pleurisy, and, in its course, the so-called bronchorrhœa, which was really a **fragmentary vomica**, supervened.

I am convinced that many of these interlobar abscesses thus pass unnoticed. The diagnosis of bronchitis, broncho-pneumonia, or of foetid bronchorrhœa is made, when the condition is really an interlobar abscess which opens into the bronchi. Potain has told me that he has seen an abscess, that was not as large as an egg, in the right oblique fissure, near the diaphragm. In a recent autopsy upon a case of disseminated pneumococci, we found in the interlobar fissure several small abscesses, which were distinct from one another, and only contained a few drachms of pus. I think that ulceration and perforation of such abscesses may produce ulceration of the bronchioles or of the arterioles, and give rise to hæmoptysis, the cause of which we ignore, and to small vomicæ, the origin of which remains unknown.

**Evolution of Interlobar Pleurisy.**—We see, as a rule, two phases in interlobar pleurisy which has not been operated on in time—the one which precedes, and the other which follows, the vomica. So far, we have studied the first phase; let us now see what happens when the vomica has declared itself. The cavity left by evacuation of the pus is partly filled up by the expansion of the neighbouring lung tissue; nevertheless, where there was only a dull and almost silent zone, it is not rare to perceive hollow, tubular breathing, and gurgling, which point to a cavity. In favourable cases, the evacuation of an infectious focus by vomica is followed by notable improve-



ment. The fever falls; the dyspnœa grows less; the fits of coughing diminish in intensity; the quantity of purulent sputum secreted by the walls of the interlobar cavity is more or less limited; the foetor disappears; the appetite returns; and, after some weeks or months, the condition becomes normal, the interlobar cavity cicatrizes, and the patient recovers. I have several times seen this fortunate ending in interlobar pleurisy after vomica.

In other cases, however, the spontaneous evolution is much less favourable, and multiple complications may arise. The patient has brought up a quantity of pus, but although the interlobar focus is partly emptied, remission does not occur; auto-infection continues; the fever remains high; the pulse is accelerated; sweating is abundant, loss of appetite complete, and wasting rapid. The patient who is not tubercular, nevertheless, appears to be so; broncho-pulmonary râles invade the lung, which is infected, in its turn, and the patient succumbs if a timely operation is not performed. To this category belong my first two cases. My patient at the Hôtel-Dieu continued to infect himself after his vomica; his strength decreased, the pus did not dry up, and I am convinced the man would have died if an immediate operation had not been performed. The same remark holds good in my second case: in spite of the vomica, the temperature kept up at 104° F., the pulse-rate was 140, and the respiration 44; the complexion became sallow; the infection made great strides, and, without surgical intervention, the child was certainly doomed.

Let these examples serve as a lesson; they prove that while there are cases in which the vomica is a mode of cure, there are other more frequent cases in which complications come on rapidly, in spite of the vomica, and which will have a fatal termination if an operation is not promptly decided upon.

These considerations are applicable to **gangrenous** interlobar pleurisy.

CASE 1. A young girl came under Olivier's care at the Enfants-Malades. She was taken ill a fortnight before with chills, fever, pain on the right side, and fits of coughing. Examination of the chest showed symptoms which were not localized. Twenty-five days after the onset of the disease the child's breath had a gangrenous odour. The expectoration, which was composed of greenish-brown masses, floating in dirty serous fluid, was very foul-smelling. The expectoration increased in amount, and the cough was incessant.

On examination of the chest, a dull zone, with amphoric breathing, was found between the middle and the lower lobes: it was evident that air had penetrated the empty focus. During the next few days pus was repeatedly brought up in large quantities. The foetor of the breath and of the sputum continued, the dyspnœa was very acute, the general condition became grave, and the patient died after an illness of nearly three months.

A cavity was found between the middle and lower lobes on the right side. The fluid in it was horribly foetid. The walls of this interpleural cyst were irregular, soft, and covered with putrefying débris with a gangrenous odour. A blackish slough was present. Multiple sections of the lung tissue around the cavity showed very marked fibrosis. There was no trace of tuberculosis.

**CASE 2.**—A woman, aged sixty-three, had been taken ill a month before with pain in the right side and shivering fits. Acute dyspnœa, cough, and abundant diarrhœa, accompanied the onset of the disease. As the situation did not improve in spite of treatment, she was admitted under Chauffard. She had extreme dyspnœa, the respirations were forty-five a minute, and the evening temperature was over 103° F. Examination of the thorax showed behind, on the right side, slight dullness over the lower two-thirds, abolition of the vesicular murmur, and faint tubular breathing on expiration. These signs somewhat simulated those of pleural effusion, and yet punctures in different intercostal spaces gave no result. The diagnosis of pleurisy was, therefore, abandoned, and spleno-pneumonia was thought of. Some days later cavernous breathing was heard over the middle of the right lung; interlobar pleurisy was suspected, and aspiratory puncture gave exit to 3 ounces of foul sanious pus. Bacteriological examination of the pus revealed micrococci, streptococci, and rods. Some days later foul-smelling pus was brought up. The tubular breathing, recognized at the level of the interlobar space, took an amphoric timbre. Antiseptics were injected into the focus. The infection continued its ravages, and the patient died in about two months. At the autopsy a gangrenous pocket was found in the interlobar fissure. This primary lesion was accompanied by secondary foci of suppurative broncho-pneumonia in the lower lobe of the lung. In these two cases immediate operation would probably have warded off these complications.

In another list of complications I place those of long duration which supervene long after the interlobar vomica. In some patients the improvement which follows the vomica is neither genuine nor lasting; we try several methods of treatment, and, fearing operation, we temporize. The patient continues to cough and to spit; the interlobar cavity goes on secreting and does not fill up; the fever reappears; the breathing is hampered; the patient, who intoxicates himself in small doses, has no appetite, and wastes; the fingers become clubbed, and, although he is not tubercular, he yet appears to be so.

In this picture I allude especially to a patient under my care at the Laënnec Hospital:

A man, thirty-five years of age, was admitted into Lépine's ward for left interlobar pleurisy, which was followed by vomica. For a long time he had brought up purulent and foetid expectoration, accompanied by cough and dyspnœa. He left the hospital and came under my care five months later. At this time he had fever, and brought up two spittoons full of foetid pus daily. I found a zone of dullness, which began at the spine of the left scapula, and extended obliquely from above downwards, and from within outwards, as far as the axilla. The resonance, above and below, was almost normal. Auscultation of this zone revealed the signs of cavity—viz., cavernous breathing and gurgling sounds. I had to content myself with placing a trocar in the cavity, and daily gave injections of alcohol. The patient became worse and succumbed four months later. At the autopsy I found the left interlobar fissure converted into a cavity filled with foetid pus, the walls being thickened and fibrous. The fibrosis had reached the adjacent lung tissue, and dilated bronchi were present in the fibrous and lardaceous parenchyma. No tuberculosis. This case proves that a cavity, if left to itself, in interlobar pleurisy may remain infected and cause fibrosis of the lung and bronchiectasis. Such a gloomy result would not happen to-day, as immediate surgical treatment would lead to cure.



**Diagnosis.**—Such may be the results and consequences of interlobar pleurisy if left alone ; it is necessary, however, to diagnose and to treat the condition in order to avoid the catastrophes of which I have just spoken. In my opinion the medical treatment of interlobar pleurisy is futile. Our part as physicians is to diagnose interlobar pleurisy, to follow out its course, to distinguish the cases in which it is spontaneously curable, when a vomica has occurred, to decide upon surgical intervention, and, lastly, to point out to the surgeon the exact field of operation. Our part is still considerable, for interlobar pleurisy is one of those numerous medico-surgical diseases in which the life of the patient is in our hands. It is not sufficient, during the first phase of the disease, to relieve the pain, cough, and dyspnoea by sedative draughts, by injections of morphia, or other appropriate means ; we must endeavour to make a correct diagnosis, and once the disease is diagnosed we must come to an immediate decision.

Diagnosis, however, is not easy in these cases. The disease begins with pain in the right or left side, accompanied by fever, cough, and dyspnoea ; this onset is common to pneumonia, pleurisy, broncho-pneumonia, and inflammation of the chest. The first few days pass, and the diagnosis is uncertain ; the idea of lobar pneumonia is put aside, because rusty sputum, crepitant râles, and tubular breathing are absent ; the idea of pleurisy is practically eliminated, because neither friction sounds nor the later signs of effusion are found. Percussion, however, reveals regions in which the resonance is not normal. On auscultation, râles, friction sounds, and sometimes, too, ill-defined tubular breathing are heard, and we then fall back on the elastic diagnosis of pleuro-congestion. We feel, however, that this is not the truth, and we always hope that more definite signs will permit us to localize the lesion.

During the onset of interlobar pleurisy this indecision is by no means wrong, for, as I said above, we lack the exact elements for diagnosis. The pleuritic focus, which is deeply hidden between the lobes of the lung, cannot yet reveal itself ; the adjacent lobes of the lung take their part in the congestive process, and attract our attention by causing tubular breathing, râles, and dullness. It is only about the eighth or the tenth day that the interlobar effusion becomes more abundant, and takes its share in the symptoms. Fresh signs are now seen ; the dull zone is marked off from more resonant areas. If the disease occupies the horizontal part of the fissure, it is behind, towards the upper third or middle of the lung, that we find a band of dullness, suspended, as it were, between more resonant regions. If the mischief affects the oblique portion of the fissure, we find the band of dullness in the axilla, with more resonant regions above and below it.

Theoretically, exploratory punctures should help us in the diagnosis. If aspiratory puncture in the suspected region gives exit to purulent fluid, the

diagnosis is clear. This method has often been put into practice ; in several cases it has been performed six or eight times in the same subject, but most frequently to no purpose. The conditions are quite different when it is a question of puncturing a purulent effusion in the great pleural cavity, or an interlobar effusion. In the former case the aspirating needle, after passing through the walls of the chest, at once enters the fluid without encountering the lung in its passage. It is quite different in interlobar effusion ; the collection, being fairly deeply situated, is surrounded by a more or less considerable thickness of lung, and the needle buries itself in the lung, when we expect it to enter the fluid collection. Read the cases again, and you will see that, in spite of several punctures at different levels, the focus is not found in many cases. Aspiratory punctures, therefore, in the case of interlobar pleurisy, do not give to the diagnosis the support which might be expected. Radioscopic and radiographic (Tuffier) examination is a method which ought not to be neglected, for it may yield valuable information.

In some cases the diagnosis of interlobar pleurisy can be made before the vomica ; in others it only becomes evident after the vomica. Moreover, at this time the symptoms given by auscultation change, and in place of a dull and silent zone we now find cavernous breathing, large râles, and gurgling.

I have not finished with the inherent difficulties of diagnosis in interlobar pleurisy. The following case gives much information :

A man, fifty-seven years of age, was admitted for fever, cough, and pneumonic expectoration, which was slightly foetid. He said that, after a chill, he had been taken ill two months ago with a violent rigor, chattering of the teeth, and pain in the right side. Had he had pneumonia ? I am not certain, for he had, so he said, no rusty sputum. However this may be, the disease went on, with fever, cough, pain in the chest, and expectoration. About twenty-five days after the commencement of the illness, at six o'clock in the morning, he felt something burst in his chest, and, in a few hours, brought up about 5 ounces of foul-smelling, purulent fluid. He had had a vomica. During the next few days the cough and the expectoration continued and three weeks after the vomica the patient, who was feverish and wasted, and was still bringing up 5 ounces of pus daily, came into hospital.

Examination showed the localization of the lesion. At the level of the horizontal interlobar fissure, on the right side, we found a zone of dullness, limited above and below by more resonant regions. Over the dull zone, auscultation revealed tubular breathing and râles. We had no doubt as to the diagnosis, and concluded that our patient had emptied an interlobar pleurisy by vomica. As the fever and the general ill-health persisted, I asked Cazin to open the infected cavity. The operation was performed, and we were then convinced that the purulent focus in the interlobar fissure was but a lobar abscess of the lung, situated at the upper part of the middle lobe, in contact with the interlobar pleura.

The admirable lecture of Trousseau on the diagnosis of lobar and interlobar vomicae helps us to appreciate the difficulty in diagnosis. As Trousseau says, abscesses of the lung are extremely rare, and what some



authors have taken for lobar are really interlobar abscesses. In any case, whether it be a question of vomicae consecutive to lobar abscess or interlobar pleurisy, the indications for treatment are identical.

**Treatment.**—As I have already stated, medical treatment is purely illusory. There is an infected cavity filled with fluid, which must be evacuated. In the fortunate but rare cases the vomica itself accomplishes the evacuation of this focus, and in a few weeks spontaneous recovery occurs. It is, however, preferable not to wait for the vomica, and in every case, if the fever persists, and the complications of infection continue, in spite of the vomica, we should operate; and if the operation is performed at the proper time, *secundum artem*, we shall be on the road to success. Moreover, in the eight patients whose history I have traced in my clinical lectures, six who have been operated upon recovered; the two who had not been operated upon succumbed.

## X. MEDIASTINAL PLEURISY—MEDIASTINAL SYNDROME.

**Discussion.**—Under some circumstances false membranes and adhesions shut off the pleura; the pleurisy is then said to be partial or encysted. Encysted pleurisy may occur in the great pleural cavity, which is divided up by septa; it is, however, in the interlobar, diaphragmatic, and mediastinal regions that pleurisy finds conditions most favourable to encystment.

Speaking generally, there is absolute difference between pleurisy of the great pleural cavity and encysted pleurisy. This difference depends on several causes. When pleurisy develops freely in the great pleural cavity, the liquid spreads between the chest-wall and the lung. The lung escapes by allowing itself to be depressed, thus averting the pressure, while the neighbouring organs are displaced; the symptoms are slight, and dyspnoea only becomes severe when the effusion is considerable.

The conditions in encysted pleurisy are quite different. When pleurisy develops in an interlobar space, between the contiguous surfaces of two pulmonary lobes, the latter, being in contact with a closed cavity, readily become infected, and for a time the pulmonary trouble masks the pleural mischief. Later, when the effusion has formed, it is deeply hidden, and so partly escapes our means of investigation; the symptoms, therefore, are not comparable with those of pleurisy of the great pleural cavity, in which the fluid is in direct contact with the chest-wall.

When the pleurisy is **diaphragmatic**, the picture again changes, for the relation of the diaphragm and the endings of the phrenic nerve give rise to special symptoms.

Lastly, when pleurisy is confined to the **mediastinal** region, the clinical picture depends upon the importance of the organs compressed by the

membranes and the mediastinal fluid. In order to grasp the clinical course of events, I will recall briefly the chief anatomical points.

The mediastinum is the region between the two lungs. It extends from before backwards, from the sternum to the vertebral column, and from above downwards, from the sternal notch to the diaphragm. This region, which corresponds to the median part of the thoracic cavity, may be divided into an anterior and a posterior mediastinum. This distinction, however, is fictitious, and this division into two mediastina "holds good only at the root of the lung, because above and below, between the sternum and the vertebral column, no such separation exists" (Tillaux).

We find in this region the heart and the great vessels, the trachea and the large bronchi, the œsophagus, the pneumogastric, phrenic, splanchnic and recurrent nerves, the large azygos vein, the thoracic duct, the glands, etc. All these organs are covered by a layer of pleura, which separates them from the lungs. The mediastinal pleura, like other serous membranes, is therefore formed of two layers: one of which covers the organs in the mediastinum, while the other covers the contiguous surfaces of the lungs. It is in the virtual space which exists between these two layers that the false membranes and the fluid of mediastinal pleurisy are encysted. Mediastinal pleurisy may be situated on the right or on the left side; it may be divided into several varieties, according as it affects the anterior or the posterior mediastinum, and the upper or the lower part of the mediastinum. These subdivisions are scarcely applicable clinically; in the few cases, however, which I shall relate, we shall see that we are dealing chiefly with posterior pleurisy.

What happens when effusion and false membranes accumulate between the layers of the mediastinal pleura? The membranes and the fluid form a tumour, and push back the lung and the organs of the mediastinum. The fact that the lung is pushed back matters little—it is a common condition in all pleural effusions—but if the organs of the mediastinum are displaced or compressed, the condition is very different. The heart is but little affected by mediastinal pleurisy, but this is not the case with the other organs. If the trachea is displaced or flattened, dyspnoea, stridor, and sucking-in result. In the case of the œsophagus dysphagia appears, solid foods can no longer pass, and liquids can only be swallowed with difficulty. If the great azygos vein is compressed as it receives the small azygos and seven or eight right intercostal veins, blood-stasis results, and the collateral circulation shows itself by a network of distended veins over the chest. If the pneumogastric nerve is stimulated by the presence of fluid and of pleuritic membranes, the patient is seized with fits of coughing and severe attacks of suffocation. If the recurrent nerve is hampered by the exudate, laryngeal troubles, such as hoarseness, dysphonia and spasms of the glottis, at once supervene.

I propose to call this collection of symptoms the **mediastinal syndrome**,



which points to the existence of a tumour in the posterior mediastinum. This syndrome is applicable to mediastinal pleurisy, and existed in two patients, whose history I will now sketch :

**Clinical Cases.**—A young man admitted for intense dyspnoea, with stridor and sucking-in. Inspiration was accompanied by a loud scraping noise, while the sucking-in was evident in the supra- and substernal regions.

Examination over the posterior mediastinum gave valuable information. We found pain on pressure and dullness on percussion ; we discovered that the stridor was most marked in the posterior mediastinum, from which point it spread to both lungs.

The disease had begun suddenly, five weeks before, with rigors and distress. Some days later attacks of suffocation had supervened with fits of coughing, which resembled whooping-cough ; but expectoration was absent.

The voice became raucous and dull, but not aphonic. About four weeks after the onset of the disease the patient noticed that solid food passed with difficulty ; he said that "it seemed to stop on its way down." This dysphagia, joined to the stridor, proved that the trachea and the œsophagus were displaced, or compressed by a solid or a fluid tumour in the mediastinum. This hypothesis was confirmed by the collateral circulation over the upper thoracic region, proving that the great azygos vein was compressed in the mediastinum. We had, therefore, to find out what the lesion of the mediastinum was.

It was not a glandular tumour, because enlarged glands were not present in the neck, the clavicular hollow, or the axilla ; and the patient had neither tuberculosis, syphilis, nor cancer. It was not an epithelioma of the œsophagus, compressing the trachea, because the dysphagia had been preceded by attacks of suffocation and stridor.

We could eliminate lymphadenoma, because the onset was acute and recent, while the examination of the blood was normal, the white corpuscles being in proper quantity, and the red corpuscles as high as 4,800,000. Might it be a purulent collection in the mediastinum—i.e., abscess from congestion, phlegmon, or mediastinal pleurisy ? I ought to say that I thought of mediastinal pleurisy.

On the night after admission he was seized with violent fits of coughing, and although there had previously been no expectoration, he brought up foul-smelling, purulent sputum ; and next morning I found in the sputum-glass about 2½ ounces of greenish homogeneous pus, such as is caused by the pneumococcus. Although the bacteriological examination did not reveal pneumococci, Griffon, with serum taken from the patient, obtained a positive result : agglutination of the pneumococci in chains and in masses. The pus in the posterior mediastinum was probably the result of a pneumococcal pleurisy, going on to vomica. The patient was cured six weeks later.

A woman, forty-four years of age, came under my care for obstinate cough. She had suffered for a week from attacks of suffocation and stridor, audible at a distance. The cough was paroxysmal. When the cough came on after a meal, it provoked vomiting of food ; although it lasted only a short while, the distress was extreme. The stridor, as well as the supra- and substernal sucking-in, were more marked during the crises of dyspnoea. At times the voice was raucous and muffled.

The disease appeared to have started from a chill. Cough and distress were the first signs. Stridor and attacks of dyspnoea appeared later. Dysphagia supervened, and the patient had difficulty in swallowing liquids. "Something shut up" as soon as she swallowed any food.

Breathing was audible on both sides ; no abnormal sounds. Percussion showed normal resonance. Heart and aorta normal. No fever. Temperature, 98.5° F.

Laryngoscopic examination : Obliquity of the larynx ; deviation of the trachea to the right ; swelling of the arytenoid region ; œdema of the superior vocal cords, leaving only a chink, and completely hiding the lower cords. No paralysis of the recurrent nerve.

We found here the mediastinal syndrome: pertussoid cough, stridor, attacks of suffocation, dysphagia, compression and deviation of the trachea, indicating a mediastinal tumour. The antecedents of the patient gave no information as to its nature.

An unforeseen event gave the clue. The patient had a vomica. After violent fits of cough, she began to bring up pus in small quantities. The quantity was estimated at about 6 ounces, and had no smell. Bacteriological examination showed pneumococci, but no other microbes.

The diagnosis was clear. The mediastinal syndrome was due to pneumococcal empyema, which had opened into the bronchus, after having caused the same signs as a tumour. This termination allowed us to hope for a prompt improvement. Stridor, dyspnoea, crises of suffocation, and dysphagia disappeared; the fits of coughing were less painful; the general state improved rapidly, and some weeks later the patient left the hospital in good health.

I have found some scattered cases of mediastinal pleurisy, and give a résumé of them:

**Andral's Case.**—Carman with signs of advanced phthisis. The expectoration, which had been phthisical, suddenly changed in character. He woke up with violent fits of coughing, and brought up a large quantity of purulent fluid, of a dull white colour, inodorous, and like the pus from an abscess. This expectoration continued for some time, and then the pus became greenish and very foetid. The patient wasted rapidly and died.

At the autopsy both lungs were filled with tubercular cavities. On opening the chief bronchus of the right lung, a perforation, with irregular edges, was found on the posterior aspect. The probe passed into a cavity which was full of pus, and was situated immediately behind the bronchus. This cavity was big enough to admit a large orange. The false membranes which formed its walls were in relation externally with the lung, which was pushed back, internally with the vertebral column, behind with the ribs, and in front with the afferent and efferent vessels of the lung. The patient had had a circumscribed effusion, and the posterior mediastinum formed its internal wall.

**Laënnec's Case.**—"I have sometimes," said Laënnec, "met with circumscribed cases of pleurisy in which the effusion amounted to only one or two spoonfuls, towards the apex of one lung, which was everywhere adherent. I have found similar ones between the inner surface of the lung and the mediastinum."

**Bouveret's Case.**—An elderly man had cough for a long while; he lost flesh, and presented the apparent signs of chronic phthisis. At the autopsy we found absence of any tubercular lesion. An encysted empyema was present in front of the hilum of the lung. The abscess was limited by thick fibrinous membranes; it contained about 10 ounces of pus.

**Thoinot and Griffon's Case.**—A woman, forty-five years of age, had been taken ill six days before with shivering fits and pain in the left side. Face pale; dyspnoea acute; pulse rapid; temperature, 103° F.; and much albuminuria, but no expectoration. Large râles and a double souffle were heard on the left side. Traube's space was resonant and the heart did not appear to be displaced. Exploratory puncture removed some drops of yellowish fluid, very rich in virulent pneumococci. An inoculated mouse died in twenty-four hours. In spite of treatment, the dyspnoea did not improve; the distress was extreme, the temperature reached 104° F., and the patient died on the seventh day.

At the autopsy mediastinal pleurisy was found. The collection was situated between the inner aspect of the left lung and the left wall of the mediastinum, and limited by false membranes, which were thick, fibrinous, and encysted between the two layers of the mediastinal pleura. The pus, which was not abundant, had the classical features of pneumococcal pus, and spread downwards as far as the costo-



diaphragmatic sinus, which was filled with false membranes, while it did not reach higher than the hilum of the lung. **Mediastinal pleurisy** here occupied the lower part of the mediastinal pleura. Further, the left interlobar fissure was joined together, and adhesions existed between the lower lobe and the costal pleura. The whole lower lobe was increased in size ; the cut section showed a typical areolar abscess, of a spongy appearance, the pockets of which were full of pus. The mediastinal glands were hypertrophied. Bacteriological examination showed that the pus in the pleura and the lung contained only most virulent pneumococci.

**Description.**—We may now discuss **mediastinal pleurisy**, which is a new chapter in pathology. We shall not consider pseudo-mediastinal pleurisy, which, from its situation on the left side, simulates pericarditis with large effusion (Grancher).

Mediastinal pleurisy may be primary or secondary—primary when the pneumococcal infection is present only in the mediastinal pleura, secondary when it is consecutive to pneumonia. It appeared to be primary in my two patients, as well as in Bouveret's case ; it was associated with pneumococcal suppuration in the lung in Thoinet and Griffon's case ; Andral's patient was tubercular.

Mediastinal pleurisy is unilateral, and is usually confined to the anterior, posterior, or inferior regions of the mediastinal pleura ; it may extend to other regions, such as the costo-diaphragmatic sinus, and coexist with adhesions of the interlobar fissure and adhesions of the lung to the chest-wall.

The liquid was purulent in the six cases quoted ; purulence is, indeed, common to encysted pleurisy of the mediastinum, the interlobar fissure, or the diaphragm. In three cases in which the infectious agent was looked for the pneumococcus was found. The **onset** of mediastinal pleurisy is not distinctive ; the pain in the chest, the fever, and the cough give no precise indication. When infection of the mediastinal pleura occurs, the adjacent lung is affected by the closed cavity, becoming inflamed and congested. The hidden focus is accessible neither to percussion nor to auscultation. The patient coughs, breathes with difficulty, has fever, and complains of distress ; sputum is absent or has no special feature, and we think of pneumonia or of pleurisy, but we cannot localize the lesion. Purulent fluid and false membranes accumulate in the affected segment of the pleura, and form a kind of tumour, the localization and the size of which determine the appearance of symptoms.

If the encysted collection travels toward the lung, and affects the mediastinum to only a slight extent, the signs are uncertain, and the diagnosis remains indefinite. If the pleural collection travels towards the organs of the mediastinum and pushes them back, the mediastinal syndrome then appears. Dyspnoea, attacks of oppression, sucking-in and stridor, dysphagia, whooping-cough, displacement of the larynx and the trachea, recognized on

laryngoscopic examination, vocal troubles, collateral circulation in the thorax, are the symptoms and signs which help us in diagnosis.

Let me emphasize the value of **stridor**, which occupies the chief place in lesions of the mediastinum. What must be understood clinically by this word "wheezing" (*cornage*)? It is a term employed in veterinary medicine. Some horses, whose breathing is almost normal as long as they are at rest, are seized while trotting or cantering with laboured breathing, accompanied by a rasping noise, which constitutes the "bruit de cornage" (roaring). A healthy man breathes silently both during inspiration and expiration, and we cannot, so to say, hear him breathe, because the air traverses the glottis, larynx, and trachea freely. If the air meets any obstacle to its passage, sonorous vibrations are at once produced, and the "bruit de cornage" becomes audible. The tone of this bruit varies somewhat according to circumstances; it may have a snoring, rasping, or sawing character.

The lesion may be situated in the larynx or in the trachea, so that we find laryngeal and tracheal stridor. The former stridor is far from rare; œdema of the larynx, spasm of the glottis, paralysis of the posterior crico-arytenoid muscles, and cancerous lesions of the larynx—in short, any lesion which produces stenosis of the larynx—may determine stridor. When the stridor is of laryngeal origin, the vocal troubles and examination with the laryngoscope indicate the site of the lesion.

The tracheal lesions that may cause stridor are intrinsic when they arise in the walls of the trachea (syphilitic gumma, polypoid excrescences, and stenosis), and extrinsic when they arise in the mediastinum (solid and fluid tumours); mediastinal pleurisy is of this nature.

While the injury to the organs of the mediastinum caused by pleurisy shows itself by the symptoms just enumerated, the examination of the dorsal region, which corresponds to the posterior mediastinum, furnishes valuable signs. Pressure on the upper dorsal vertebræ and the corresponding costo-vertebral grooves is painful. In the same region more marked dullness is found on the right or left of the vertebral column, according to the side affected. The stridor is most marked **at** this spot, and diminishes as the distance from the mediastinum increases. Sonorous and moist râles may be heard in the lung near the mediastinum. They indicate congestion of the lung, are present in front and behind, and are more numerous on the affected side. Radiography may give useful information, as in one of my patients.

Mediastinal pleurisy, like most encysted empyemata, tends to vomica. The vomica generally supervenes some weeks after the onset of the disease, and is scanty, as the pus, encysted in the mediastinal pleura, never reaches a large amount. The vomica of my patient at the Necker Hospital might



be put down at 6 ounces ; in my patient at the Hôtel-Dieu it did not exceed 3 ounces : it was foetid in both cases. Andral's patient had an abundant vomica, but the approximate quantity is not given.

**Diagnosis.**—The diagnosis of mediastinal pleurisy is very difficult. The signs are insufficient and indefinite until the mediastinal syndrome appears. When this syndrome appears, percussion and auscultation of the dorsal region (which corresponds to the posterior mediastinum) give information as to the localization of the lesions. We can then by these signs and symptoms, aided by radiography, arrive at the topographic diagnosis ; the knowledge that the lesion occupies the mediastinum is the first step in the diagnosis. The nature of the lesion has then to be ascertained. Is it tuberculous, cancerous, or syphilitic adenopathy ? is it a lymphadenoma, or is it an aneurysm of the aorta ? is it an abscess or pleurisy ?

As I have said, the onset and the course of the symptoms may give a clue to the diagnosis. Mediastinal adenopathies and tumours do not begin suddenly with fever : the onset is insidious, and the course slowly progressive ; moreover, the swelling in the neighbouring glands (neck, axilla) aids in the diagnosis. Mediastinal pleurisy, on the contrary, arises suddenly, like an acute febrile disease ; fever, pain, and cough mark the onset, dyspnoea is early, and vomica supervenes in at least half the cases. Furthermore, in the three cases in which laboratory researches have been made, bacteriological examination and sero-diagnosis have demonstrated the pneumococcus ; sero-diagnosis would therefore be useful before the appearance of the vomica in order to ascertain the nature of the disease.

**Treatment.**—The prognosis is not free from gravity. The disease is serious in itself, and also from the secondary infections which may reach the bronchi and the lungs. We must, then, be ready to operate if occasion arise. Thanks to the marvellous progress of surgery, the posterior mediastinum has become fairly accessible to surgical investigations. Dr. Potarca (of Bucharest), in a work entitled "*La Chirurgie Intramediastinale Postérieure*," has collected several cases of operations for purulent collections in the posterior mediastinum, phlegmon, mediastinitis, suppuration from bony or from glandular lesions, and foreign bodies. Although in the memoir in question purulent mediastinal pleurisy is not studied (apart from Zimbicki's doubtful case), it is still true that the posterior mediastinum should be opened in mediastinal pleurisy if occasion arise.

## XI. DIAPHRAGMATIC PLEURISY.

Pleurisy of the diaphragmatic portion of the pleura may be primary or secondary, and dry or accompanied by effusion, which may be fibrinous or purulent, free or encysted. The dry form is the more frequent. Cirrhosis

of the liver, peritonitis, nephritis, the puerperal state, tuberculosis, and pneumonia are the common causes of diaphragmatic pleurisy. In pelvic peritonitis the inflammation is transmitted from the peritoneum to the pleura by the lymphatic vessels, especially by those which accompany the utero-ovarian vessels, and pass along the pillars of the diaphragm. As regards metapneumonic pleurisy of the diaphragm, the considerations enumerated in the case of interlobar pleurisy remain the same.

The effusion is usually scanty, and sometimes encysted, while the lung is often the seat of acute congestion, which enters largely into the clinical picture of the disease.

The **symptoms** of diaphragmatic pleurisy vary in severity; they are sometimes moderate, but in acute cases the disease shows itself by sharp pain at the middle of the diaphragm (neuralgia of the phrenic nerve). The pain reaches as high as the shoulder, and can be provoked by compressing the insertions of the diaphragm into the tenth rib (diaphragmatic point) two fingers' breadth from the linea alba (Guéneau de Mussy), or by pressing on the phrenic nerve in its passage between the lower heads of the sternocleido-mastoid muscle. The inferior costal region is immobilized because of the paresis of the diaphragm (Andral), and the usual signs of pleurisy—viz. friction rub, dullness, tubular breathing, and ægophony—only appear when the great pleural cavity is also affected. In the grave cases—which are, moreover, rarer than Andral's description would lead us to suppose—the symptoms are acute, and recall the crises of angina pectoris; the breathing is short, and interrupted by hiccough; the voice is broken; dyspnoea is excessive; one-half of the diaphragm is immobilized, and if the other half is affected, the patient's life is in danger. Suppurative diaphragmatic pleurisy has no special symptoms, but behaves like the pleurisy which we have just described; it is encysted, like partial pleurisies, and in some cases, especially in elderly people, it remains latent, being only found at the autopsy. It ends fairly frequently by vomica. The agonizing pains of diaphragmatic pleurisy may be relieved by means of dry-cupping, fifteen or twenty leeches, or injections of morphia; antipyrin may be prescribed, and an ointment of methylate of salicylate may be applied to the painful region.

## XII. LOCULATED, AREOLAR, OR POLYMORPHOUS PLEURISY.

Pleurisy arising in the great pleural cavity may be loculated. Sometimes it occupies a large pocket, which may contain several ounces of sero-fibrinous or purulent fluid (unilocular); at other times a series of small ones, when we speak of multilocular pleurisy.

**Unilocular Pleurisy.**—I have recently had a case under my care at the



Hôtel-Dieu. The pocket was encysted in the middle portion of the right pleural cavity behind.

A woman, twenty-one years of age, had her first attack of pneumonia in 1898. The disease was situated on the left side. On May 21, 1900, pneumonia of the right apex, after a chill. Temperature was over  $104^{\circ}$  F.; acute pain in the side; tongue dry and red; vomiting and diarrhoea; condition of the patient very grave. On May 28 the temperature, which had varied from  $103^{\circ}$  F. to  $104.5^{\circ}$  F., showed some tendency to fall; the vomiting and the pain in the side became less. Two days after, pain reappeared, but was now lower down; and while auscultation revealed the existence of redux crepitation at the apex, very harsh breathing, accompanied by friction sounds and some crepitant râles, was already perceptible over the middle of the lung. From June 1 to 5, the temperature varied from  $101^{\circ}$  to  $103^{\circ}$  F., patient's condition was satisfactory, and pain in the side disappeared. On the 5th, examination showed dullness at the angle of the scapula, over an area of 2 inches in the vertical and 4 inches in the horizontal direction. This dullness was clearly limited above, where the apex had regained its resonance, while it gradually became less marked below. Vocal fremitus abolished only in the region where the dullness was absolute. Auscultation showed distant tubular breathing and very clear ægophony over the dull zone, with slight aphonic pectoriloquy. Exploratory puncture blank, 1 inch above and 2 inches below the dullness. At the focus itself a green sero-purulent fluid, rich in leucocytes, and containing numerous pneumococci, mostly in chains of from five to ten elements, as well as some diplococci, was drawn off. The pneumococcus was inoculated into a mouse, and showed itself virulent, while it grew well on the different culture media, and preserved its chain-like form on agar. From the 31st to the 7th the condition remained stationary, and the pleurisy made slow progress, diffusing in a circle, and invading the base and the apex at the same time, the dullness being propagated towards them. The pleurisy remained, however, clearly "suspended," and on June 11 punctures yielded pus neither at the apex nor at the base. In the centre of the dull zone, however, the pus was greenish, thick, creamy, and rich in pus corpuscles and fatty granules. The pneumococci in chains had become rare. On the 12th, operation for empyema was performed at the level of the angle of the scapula, with resection of a rib, and we found 10 ounces of pus, forming a focus limited above and below by false membranes.

The patient recovered. This was very clearly a case of loculated pleurisy, with a large pocket, which had developed in the great pleural cavity, and not a case of interlobar pleurisy. Moreover, in interlobar pleurisy the signs (ægophony, souffle, and pectoriloquy) have never the clearness found in the present case.

**Areolar and Polymorphous Pleurisy.**—Loculated pleurisy may be chronic from the first, or be due to acute attacks affecting the pleura, which is already partitioned off by the false membranes of former inflammation. Loculated pleurisy may be sero-fibrinous, hæmorrhagic, or purulent; indeed, it is not rare on performing thoracentesis to meet with serous fluid in some pockets and pus in others. Sometimes the loculation is simple, and the false membrane divides the effusion into two pockets only; more often the loculation is multiple, and the pockets are numerous; lastly, in some cases the pleurisy is areolar, as if the false membranes were themselves infiltrated with serous fluid.

Areolar and polymorphous pleurisy usually present the following forms: We find in a patient the signs of considerable effusion, and diagnose the

presence of 4 or 5 pints of fluid in the pleura ; puncture is performed, and we are astonished at withdrawing very little fluid, which is not in proportion to the signs present ; further, we note the persistence of the signs of effusion at the other parts of the thorax, either above or below the point of puncture. The exploratory needle shows that fluid is still present at these points, and we conclude that a membranous septum is interposed between two or more pockets of fluid. This form has been called "partitioned pleurisy."

Sometimes the fluid is identical in the different pockets ; at other times it is serous in some and hæmorrhagic or purulent in others. In this case the pleurisy is called "polymorphous" (Galliard).

The disposition of the false membranes which partition the pleura is, however, very variable. Sometimes a single horizontal, oblique, or vertical pseudo-membranous partition, which divides the pleura into two (**bilobed pleurisy**), exists ; at other times multiple and irregular adhesions divide the pleural cavity into numerous small and distinct cavities (**multilocular or areolar pleurisy**). In the latter case we often see pockets occupied by a gelatinous quivering mass that is formed of a fibrinous network impregnated with serous fluid, while the neighbouring pockets contain sero-fibrinous, sero-hæmorrhagic, or sero-purulent fluid.

The partitioning has been attributed to the false membranes of antecedent pleurisies ; relapsing pleurisy is said to be more easily partitioned than pleurisy which affects an intact serosa. In these cases the pleurisy is said to be multilocular from the start. We may also observe the following varieties: encysted serous pleurisy, followed by adjacent empyema, or encysted empyema, complicated by adjacent sero-pleurisy. The latter cases are more common, because empyema is much more often encysted than serous pleurisy.

Save in exceptional cases (Jaccoud), the diagnosis of loculated pleurisy can only be made after puncture. The attention is aroused by the insufficient quantity of fluid evacuated. Sometimes, also, the puncture may be blank, if the needle penetrate the septum. The diagnosis will be confirmed by methodical exploration of the pleural cavity, by means of an aspirator, which is provided with a long needle.

The prognosis is especially serious when encysted suppurations escape notice, because this fact prevents treatment by free incision ; we only find them at the autopsy. We must remember that polymorphous pleurisy may be symptomatic of tuberculosis or of pleuro-pulmonary cancer.

### XIII. SYPHILITIC PLEURISIES.

Early syphilitic pleurisy occurs in the secondary stage, while late syphilitic pleurisy is seen during the tertiary period.

The early form has been well described by Chantemesse and Widal, who



quote several conclusive cases. At the Hôtel-Dieu I have seen a very clear example :

A man, twenty years of age, came into the hospital with pain at the lower angle of the left scapula. Pleurisy without effusion was found. Friction sounds were heard, which extended behind and laterally as far as the axillary region. Other organs healthy. Auscultation of the lung showed no lesions, and we had to deal with apyretic, painful pleurisy of four days' duration.

We could not incriminate tuberculosis, influenza, or rheumatism. This boy, however, showed a well-marked syphilitic roseola. The syphilis was two months old. The cicatrix of a chancre was visible on the dorsal surface of the penis, and enlarged glands were present in the groins. He complained of headache and sore-throat, and we found syphilitic angina, with mucous patches. As pleurisy had appeared in a youth, who had for two months been infected with syphilis, it seemed natural to consider the pleurisy as a secondary complication of syphilis, and I placed the patient on mercurial treatment. The pleurisy remained dry, and I found no effusion. The pain and friction sounds disappeared in ten days.

Similar cases have been published, and the occurrence of syphilitic pleurisy in the secondary stage is therefore well proved. Why should we not see early syphilitic pleurisy when we see nephritis, arteritis, icterus, and bronchitis appear among the early manifestations of syphilitic infection? Early syphilitic pleurisy may be dry, or be accompanied by effusion. I do not believe that the effusion is ever so abundant as to necessitate thoracentesis. An important difference between early and late syphilitic pleurisy is that the former is not associated with pulmonary lesions, while the latter, with which I am now dealing, forms part of the broncho-pulmonary syphiloma discussed in the chapter on Syphilis of the Lung.

I have been able to find only a small number of cases of **tertiary** syphilitic pleurisy. According to Mauriac, "this pleurisy is often seen, and is accompanied by effusion." Tertiary lesions of the pleura may be placed in one of the following categories : Either the pleural lesion is an unimportant complication of the pulmonary lesion, or the effusion is abundant; the pleurisy is the chief feature, and the condition well merits the name of syphilitic pleurisy. As an epiphenomenon, mention must be made of this pleurisy in some cases of tertiary syphilis of the lung; it has been referred to by Carlier. In one, he says without further amplification, that the pleura on one side contained 300 grammes of a clear yellow fluid, and the visceral layer presented recent false membranes, which were very easily torn, while the parietal and diaphragmatic layer was also inflamed.

In Jacquin's thesis Balzer's case of syphilitic pleurisy with large effusion is described :

A man, thirty-two years of age, came to hospital with functional troubles and physical signs which led to the diagnosis of caseous pneumonia on the right side. A month later abundant effusion appeared on the right side. Pleurisy, complicating tuberculosis of the lung, was then diagnosed; but after four days the patient died, and the autopsy revealed a syphilitic liver that was studded with gummata and segmented by

scars. The right lung was also full of gummata, the largest of which just involved the pleura. These gummata did not contain a single Koch's bacillus. The lesions in the right pleura were so characteristic that I give them in detail. "In the right pleura a much more considerable effusion exists than physical examination would have led me to suppose. There are about 4 pints of turbid blood-stained serum. The parietal and visceral layers of the pleura are considerably thickened over the whole extent of the effusion. They show a continuous fibrous covering, 1 or 2 millimetres in thickness, attaining at the base of the lung a thickness of nearly 1 centimetre. This fibrous cap is surrounded by false membranes at several spots. At the points where the fibrous thickening is most considerable we find, on section of the pleura, hard yellowish caseous masses as large as a pea or as a millet-seed." This case of tertiary syphilitic pleurisy, in which the effusion amounted to 4 pints, is most conclusive.

I saw the following case :

I was called to treat a man with terrible dyspnœa, which had recurred in more or less acute attacks for the past year. Tubercular broncho-pneumonia had been diagnosed. On my first examination I found signs of an effusion, which I estimated at  $1\frac{1}{2}$  pints. Although this quantity of fluid did not explain the patient's dyspnœa, I performed thoracentesis, but drew off only 21 ounces of slightly rose-coloured fluid. The patient experienced, however, no relief. I searched, without success, for the cause of the disease, when he finally confided to me that he had had syphilis. This avowal guided my treatment. I at once gave mercury and iodide of potash in large doses, and the dyspnœa improved so rapidly that at the end of a few weeks the breathing was almost normal. At my last visits no trace of pleurisy remained, though the broncho-pulmonary lesions were slower to improve. In this case, again, the syphilitic nature of the trouble was shown by the beneficial action of specific treatment.

There exists, then, tertiary pleurisy, which is associated with syphilitic lesions of the lung, and the true means of diagnosing it is to think of syphilis.

#### XIV. APPENDICULAR PLEURISIES—PYOPNEUMOTHORAX AND SUBPHRENIC EMPYEMA.

In 1890 I read a paper before the Académie de Médecine\* on "**Appendicular Pleurisy**," a name which is generally accepted at the present day. Later, in my clinical lectures at the Hôtel-Dieu,† as I had seen how frequently appendicular subphrenic empyema precedes or accompanies infection of the pleura, I have united appendicular pleurisy and subphrenic empyema in a single description, and I shall do so in this chapter.

I think that it will be useful to give a résumé of some cases :

**Clinical Cases.**—A man, twenty-six years of age, admitted to the Hôtel-Dieu. He suffered from shortness of breath, was pale, had an anxious look, with sunken eyes, and a wretched pulse ; he appeared moribund. He complained of a pain in the right side of the chest, and was so feeble that he could not raise himself on the stretcher. On

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\* Dieulafoy, "La Pleurésie Appendiculaire" (communication à l'Académie de Médecine, séance du Mardi, 10 Avril, 1900).

† Dieulafoy, "Pleurésie Appendiculaire et Empyème Sous-phrénique" (*Clinique Médicale de l'Hôtel-Dieu*, 1903, vol. iv., 15<sup>me</sup> et 16<sup>me</sup> leçons).



the right side I found a large effusion, with absolute dullness, except at the apex, where the resonance was exaggerated.

It is not common for pleural effusion, however abundant, to show such general symptoms. In this case it was not simply dyspnoea, but adynamia, bordering on collapse, which dominated the scene. What, then, did this effusion conceal? Apert at once performed exploratory puncture, in order to ascertain the nature of this pleurisy, and drew off turbid fluid, which was of **nauseous odour** and not homogeneous.

The case was therefore one of foetid, perhaps of putrid or of gangrenous, pleurisy. There was not time to make a pathogenic diagnosis, and the imperative indication was immediate surgical intervention. Marion therefore came to see the patient, but found him in his death agony, with thready pulse and cold, cyanosed limbs. Death occurred soon afterwards.

From the information given by the family, it was possible to reconstruct the case.

He was taken ill on November 10 with abdominal pains, which increased in severity during the night, chiefly on the right side. Next morning, as he felt easier, he went to his office. In the evening he passed a motion. The next night was more comfortable, but on Sunday evening (the third day) he was seized with such severe pain on the right side of the belly "that he twisted and groaned in agony."

During the examination his wife remarked that the belly was particularly painful on the **right side**. An enema and poultices, with laudanum, were prescribed. Next day vomiting came on. Patient had fever and rigors; he was constipated, and the abdominal pains were as severe as ever. During the next few days the situation did not improve. The **hepatic region** became painful, and a blister was ordered.

Meanwhile the patient began to cough, and complained of pain in the **right side of the chest**. Pleuro-pneumonia was diagnosed. Fever returned; violent pain in the chest, with extreme dyspnoea, cold sweats, and tachycardia, supervened. On the morning of the 29th **hydropneumothorax** was found. He was then brought to the Hôtel-Dieu.

To sum up: The disease, which lasted nineteen days, was divisible into two stages: the first or abdominal stage was characterized by acute appendicitis, and treated by medical means; while the second or thoracic stage terminated with symptoms of pneumothorax and death. Let us now see what the autopsy revealed.

Thoracic cavity first examined. Intercostal space perforated under water. As soon as the pleura was opened, a nauseous odour was noticed, and bubbles of gas came up through the water, showing the presence of foetid gases in the pleura, and confirming the diagnosis of pneumothorax, made during life by the physician who had sent up the patient. The case was one of pneumothorax by putrefaction, and not by perforation.

In the pleural cavity, were 7 pints of very foul greyish fluid. The left lung, which was pushed back against the spine, was reduced to a fifth of its normal size, airless, and no longer crepitant. No adhesions bound it down to the costal wall, but its base was adherent to the diaphragm. Pus was present between the two layers of the thickened diaphragmatic pleura. No sign of perforation or of gangrene found. The pleurisy was putrid, but not gangrenous; putrefaction and formation of gas, but no mortification of the tissues, existed.

Let us now pass on to the abdominal cavity. On the left side, nothing to notice; no trace of peritonitis. On the right side, numerous adhesions from the abdominal wall to the organs, and, on tearing them through, 5 ounces of pus, quite as foetid as the pleural fluid, flowed out. The abdominal cavity was explored, pus and membranes found; some were spread out over the intestine and the liver, while others reached up behind the caecum and the colon. Posterior surface of the caecum bound down by membranes to the parietal peritoneum. The appendix was included in this mass. It was very long, of the vertical type, and passed up on the posterior surface of the caecum; it was free at its caecal origin, but higher up it was surrounded by adhesions and bathed

in pus. Its walls, which were double their proper size, were becoming gangrenous, but had not perforated. It was in this part of the appendix, which had been **transformed into a closed cavity, that the acute toxi-infection was elaborated.** The purulent track continued behind the colon, turned round the hepatic flexure, and spread out in front of the right lobe of the liver, setting up abscesses in its course.

The liver was partly hidden by perihepatitis. The diaphragm was examined for a perforation; as none was found, the infection had evidently been carried to the pleura by the lymphatic vessels.

Other organs—*i.e.*, liver, spleen, kidney, and heart—were healthy. The left lung, however, had been infected. The apex showed a splenized region as large as an egg, with several infarcts in different stages of evolution. The left pleura was healthy.

Bacteriological and experimental researches were made by Apert with the pus drawn off by aspiratory puncture, and also the peritoneal pus removed at the autopsy. The result was as follows:

Some of the micro-organisms were aerobic, while others were anaerobic. They had caused putrid pleurisy, as shown by the formation of gas within the pleura.

With guinea-pigs and rabbits, inoculation of pus into the cellular tissue and injection of pus into the vein of the ear produced abscesses and infarcts, containing the same micro-organisms that were found in the specimens of pus.

The patient had been taken ill with classical appendicitis. I do not know what the original diagnosis was, but I do know that the treatment was medical when it should have been surgical. The man would have been saved by early operation, just as we save all the patients who are operated on in good time, when **no precious time is lost in medical treatment** which is based on erroneous pathogenic conceptions. This treatment is wrong, for it appears to be doing something when it does nothing but give the toxi-infection time to strike a fatal blow. Enemata, sedatives, and rest were ordered, while infection of unusual virulence was brewing in the closed cavity of the appendix. Aerobic and anaerobic microbes then made their way through the walls of the appendix, and provoked the formation of purulent tracks, which took an **ascending** course, and in a few days reached up behind the cæcum and colon, and spread to the anterior and upper surface of the liver.

The **phrenico-pleural** phase now appeared; cough, dyspnoea, and pain were its signs. The diaphragm was not perforated, but, thanks to the lymphatic channels, was traversed by microbes of undiminished virulence. It might well be said of this process, *Vires acquirit eundo*. The pleural infection was so acute that the effusion rapidly reached 7 pints. Putrefaction was shown by the appearance of the pneumothorax. The lesions were so severe that events hurried on, and the patient died nineteen days after the onset of appendicitis.

The infection, which started from the appendix, had followed two courses—the one by the lymphatics, and the other by the blood. The lymphatic infection took place by extension; though it started so far down and reached so high up, yet in a few days it spread across the abdominal cavity and invaded the pleura. The infection of the left lung was due to microbic emboli.

But, it will be asked, is pleurisy a frequent complication of appendicitis? It is certainly far from rare, and in proof I can quote many cases.

I saw a very remarkable case. The patient was a lady, in whom I diagnosed appendicular pleurisy and subphrenic empyema. The double operation was performed by Segond: he operated first on the pleura, and a fortnight later he attacked the subphrenic empyema. The patient recovered.

In consultation with Brun and Jalaguier, I saw a youth with appendicular pleurisy. On May 20 he was attacked with appendicitis. Three days later Brun found severe peritonitis. Operation performed next morning. Appendix was gangrenous;



peritoneal fluid was sero-purulent and foetid. Free drainage established. During the next few days, in spite of improvement in the peritoneal condition, situation was grave, and temperature very high. No cause could be found for the persistence of these alarming symptoms. On June 2 symptoms of **right pleurisy** were discovered, and exploratory puncture drew off turbid fluid. On June 4 operation for empyema; a large quantity of dirty, horribly foetid fluid drawn off. Free drainage established. Next day diffuse inflammation at the edges of the thoracic wound, but no gaseous crepitation. During the following days the inflammation stopped; the antiseptic irrigations of the pleura seemed to indicate that the pleural focus had been effectively treated, but yet fever persisted and the situation became worse.

I then saw the patient, with Brun and Jalaguier. Minute examination revealed no fresh lesions in any organ. We were of opinion that the patient was suffering from a profound intoxication, and prescribed accordingly. About June 11 cerebral symptoms (intoxication, or perhaps meningeal infection) of photophobia, strabismus, irregularity of the pulse, and stiffness of the neck supervened, and the patient died.

### Résumé of two cases of appendicular pleurisy described by Terillon :

A young man, seventeen years of age, had suffered from appendicitis. Belly distended and painful; on the right side a painful swelling, reaching from the iliac crest to the false ribs. Affection was of six days' duration. Temperature, 103° F., and pulse rapid. Intervention decided on. On opening the abdomen, cæcum was adherent to the abdominal wall; sharp gush of pus occurred. The abscess occupied the iliac fossa behind the cæcum, and reached up towards the kidney. On the next day the temperature as high and the pulse as rapid as before operation; general condition worse. Two days later it was noticed that the respiration was embarrassed, and existence of a large **right effusion** was made out. Puncture yielded pus. After operation for empyema, fever fell at once, improvement followed, and the patient was completely cured.

Boy, fourteen years old, had suffered for five days from right iliac peritonitis. The temperature was 104° F., and the gravity of the situation was evident. Terrillon proposed immediate operation, which was not agreed to. Ten days later he was sent for in a hurry. The child was sitting up in bed, having shown symptoms of asphyxia since the morning. Large effusion present on right side. About a pint of foetid pus was drawn off by puncture, but syncope came on suddenly, and the patient died. It is probable that in this case, as in the preceding one, prompt operation would have brought about a cure.

The pleural complications of appendicitis have been studied in Germany since 1891. Here is a series of cases. Wolbrecht, in his thesis (Berlin, 1891) entitled "Pleural Complications in Typhlitis and Perityphlitis," quotes a few cases, two of which are personal.

CASE 1.—A joiner, twenty-four years of age, who some days before had experienced the symptoms of perityphlitis, came into his clinic. Signs of subphrenic abscess and of **right pleurisy**. Exploratory puncture drew off foetid pus. Operation for empyema performed; fifth rib resected; 5 pints of nauseous fluid withdrawn. Two months later patient was cured.

CASE 2.—A man suffering from perityphlitis, with circumscribed peritonitis. Ten days later **right pleurisy**, with œdema of the chest-wall. Exploratory puncture showed turbid blood-stained effusion. Three days later patient seized with violent cough, and brought up abundant foul-smelling pus. He finally recovered, after a convalescence of three months.

Larsen and Winge relate a case of appendicitis, with retrogression of the peritoneal symptoms at the end of the first week :

Pain in the right hypochondrium appeared, and **right pleurisy** developed rapidly. The dyspnœa became very intense, and death followed. Post-mortem : **pyopneumothorax on the right side** ; perforated appendix buried in an abscess ; narrow purulent track ran up behind the liver, and had perforated the diaphragm in two places.

Grawitz, under the title of "Perforation of a Perityphlitic Abscess into the Pleural Cavity," has published the following case :

A woman was taken ill a fortnight before, with pain in the right side of the belly, constipation, fever, and vomiting. Later the symptoms of **right pleurisy** supervened, and the patient died three weeks after admission to hospital. Post-mortem : the whole appendix was converted into a sac, and had perforated ; near its base was a calculus, the size of a haricot bean. The intestinal coils were adherent in the right iliac fossa, and the pelvis contained pus. From the gangrenous appendix a sinuous cavity extended upwards behind the right kidney and the duodenum. A perforation, as large as a sixpence, was present in the posterior part of the diaphragm, and established communication between the subdiaphragmatic region and the right pleura. Three pints of foetid pus in the pleural cavity.

Wollert has published similar cases :

A young man, sixteen years of age, was taken ill with appendicitis. Thirteen days later sharp pains at the right base, and intense dyspnœa. **Right pyopneumothorax**, with metallic tinkling, found. Post-mortem : peritonitis, consecutive to appendicitis. Sheet of pus over the ascending colon, the diaphragm, and the liver. The diaphragm was perforated, and the right pleura contained 8 pints of purulent fluid, with foetid gas.

Appendicular pleurisy is sometimes **serous** ; the infection is reduced to a minimum, and recovers with or without operation.

Case seen with Larcher and Monod :

Young girl suffering from appendicitis, diagnosed by Larcher, and operated upon by Monod on May 9. Everything went well until May 18, when pain appeared in the right hypochondrium and at the base of the chest. The temperature now rose to 103° F. Cough was frequent, though expectoration absent. This condition lasted for several days, without appreciable change. I was then called to see the patient, and found friction sounds and fluid on the right side. In this case we had to discuss the propriety of surgical intervention. As the general condition was not bad, we agreed to see patient again before we decided. We had the satisfaction of witnessing the successive disappearance of the tubular breathing and the friction sounds, while the fever yielded and recovery ensued at the end of four weeks.

A child, fourteen years of age, who had had several previous attacks, was again taken ill with appendicitis and pericæcal abscess. He was operated on by Jala-guier. The appendix, of the vertical type, had a bent extremity, which was adherent and communicated with the cæcum. Some days later dyspnœa came on ; dullness, with tubular breathing on expiration, and aphonic pectoriloquy, were discovered at the right base. Some effusion formed, but was absorbed eight days later.

Körte reports the case of a woman suffering from retrocæcal inflammation. In spite of incision, the inflammation passed through the diaphragm and reached the pleura. Resection of the eighth and ninth ribs ; patient recovered from the effusion, which, the author states, was serous. Another case of Körte's is that of a man, twenty-eight years of age, who was taken ill with sharp abdominal pains on the right side,



fever, and shivering. A purulent collection necessitated surgical intervention, and a putrid phlegmon, which reached from the cæcum to the diaphragm, was opened. An effusion, which was punctured, formed on the right side. The author describes it as serous. The patient finally recovered.

**Description.**—The first point to note is the manner in which the appendicular infection reaches the pleura. How does a remote lesion like appendicitis attack the pleural cavity? Why does appendicitis, which is a very small lesion, succeed in a few days in producing putrid pleurisy, with several pints of fluid? Let us try to answer these questions.

The infection, which becomes more virulent in the closed cavity of the appendix, causes the pathogenic microbes, *Bacillus coli*, and other aerobic or anaerobic germs to spread to the peritoneum. Sometimes perforation or gangrene of the walls of the appendix occurs, and the microbic pulp spreads widely over the peritoneum; at other times the migration of microbes takes place through the unperforated wall, as we may see from specimens.\*

The infection, after reaching the peritoneum, behaves in different ways. It may only spread a short way from its seat of origin, being limited or not by adhesions, or it may excite remote foci, which show no apparent relation to the original focus. These remote abscesses are due to microbes carried to a distance, but in what way we do not always know. Can these remote abscesses arise in the pleural, as in the peritoneal, cavity? In other words, can remote pleurisy occur in the course of appendicitis? Certainly; but most often it is by proximity, through the peritoneal track, by way of the adhesions and of the lymphatics that extension to the pleura takes place. Under such circumstances the appendicitis is nearly always of the **ascending type**. The direction of the appendix is an essential factor. In appendicitis of the descending type the purulent collections and adhesions often form in the pelvis near the bladder and rectum, but in the ascending type the pus and membranes pass up towards the liver. The infection takes place from below upwards, because the initial focus, or *primum movens*, gives it this direction. It reaches the hypochondrium, passes through the diaphragm, and invades the pleural cavity, having marked its route by purulent tracts, so that it is possible to follow the infection from its small origin in the appendix to its full maturity in the thorax. Appendicular pleurisy nearly always occurs on the **right side**; there are very few exceptions to this rule.

Let us now turn to the clinical side of the question. At what moment may pleural complications ensue in appendicitis? They appear about a week or a fortnight after the onset of appendicitis. The infection, which starts from the appendix, does not begin its migration before the third or fourth day. Surgery has, therefore, time to intervene. By suppressing

\* Dieulafoy, *Clinique Médicale de l'Hôtel-Dieu*, 1897, pp. 345, 346, 347.

the infecting focus at its commencement, the disease is cut off at the root. Let us not forget that appendicitis, whether slight or severe, may be followed by pleurisy, just as it may be followed by abscess of the liver; in the case of appendicitis **the benign nature is only apparent**. Pleural complications are hardly likely when the active process of appendicitis is extinct.

The appearance of appendicular pleurisy is usually heralded by the symptoms which I have called **abdomino-phrenic**—viz., pain in the right hypochondrium radiating to the shoulder, dyspnœa, and apparent increase in the size of the liver. These symptoms are explained by the perihepatitis and the subphrenic empyema which often precede the stage of pleurisy. Pleural symptoms, pain in the side, and cough appear in their turn, and blend with the abdomino-phrenic ones.

The abdomino-phrenic symptoms, which usually precede those of pleurisy, may themselves be preceded by abdominal symptoms of pain in the right flank, puffiness, and dullness; they indicate the passage of purulent infection from the right iliac fossa to the hypochondrium.

As appendicular pleurisy has not always the same course, we must study its various forms. As a rule, the pleurisy is putrid, with much effusion; the fluid forms rapidly. The fever is variable; dyspnœa, anxiety, loss of strength, weak pulse, sallow and earthy tint of the skin, bear witness to the gravity of the situation. It is true that the pleural infection is not the only cause: the peritoneal infection is responsible for its share. Œdema of the wall is not rare.

We frequently find amphoric breathing and hippocratic succussion, which point to pneumothorax. At first we think of pleuro-pulmonary perforation, and do not admit the possibility of pneumothorax from putrefaction.

In considering putrid right pleurisy, we must always think of appendicitis, even though it has been so slight as to attract but little attention. We must reconstruct the appendicular and abdominal stages, which have preceded the phrenic-pleural stage by six or eight days.

Whether there be pneumothorax or not, as soon as pleurisy develops suddenly, with grave symptoms, we should **without delay** investigate the nature of the fluid. Exploratory puncture is performed at once, and turbid, non-homogeneous, greyish or brownish foetid fluid is drawn off. In such a case, without waiting for the result of aerobic or of anaerobic cultures, operation for empyema must be performed **immediately**. The fluid, in a test-tube, divides into two layers; the lower one forms a dense, dark-coloured deposit; the upper has a serous or turbid appearance.

Appendicular pleurisy has little tendency to **vomica**; its course is so rapid and complications occur so quickly that the vomica has, doubtless, no time to occur. I know of only two cases:



One (Krohne) concerns a young girl with appendicitis and pericæcal peritonitis. Some weeks later subphrenic empyema occurred, and was followed by fits of coughing and fœtid vomica. Laparotomy, performed forty-eight hours later, revealed a large subdiaphragmatic abscess, opening into the thoracic cavity. The condition might, perhaps, have been due to the opening of a subphrenic abscess without concomitant pleurisy.

I would say as much concerning the following case, reported by André and Michel :

A young woman had appendicitis and peritonitis. Laparotomy evacuated a quantity of fœtid pus ; pleuro-pulmonary signs appeared later ; exploratory punctures made, in order to ascertain the nature of the fluid, gave no result, and an exploratory incision had no better success. The patient was seized with vomica and died. No autopsy was allowed.

Such is the history of putrid appendicular pleurisy in its most common form. In some cases the fluid, as I have said, is serous, the infection is reduced to a minimum, and the condition resembles serous pleurisy, which absorbs spontaneously or yields to puncture. These benign cases of appendicular pleurisy are not accompanied by the grave symptoms of the preceding variety.

**Appendicular Subphrenic Empyema.**—I have mentioned above how the infection ascends from the appendix to the pleura. In its course this infection marks its route by purulent collections, of which one is **subphrenic empyema**. The collection may be of little or of serious import ; we may find from 1 to 3 pints of fluid. If the collection is small in amount the signs are difficult to recognize, but yet the subphrenic pain is a good guide. If the collection is abundant, we find, besides the pain, which may be made worse by pressure in the subphrenic region, deformity and bulging in the epigastric and right hypochondriac regions ; the liver, which is pushed down by the effusion, appears enlarged, though it is only displaced, and, on the other hand, the dullness, which ascends towards the thoracic cavity, simulates a non-existent effusion in the pleura.

Although the liver is surrounded by pus it **remains free**, and this point distinguishes it radically from appendicular infection of the organ, which is then riddled with abscesses. The reason is that the mode of infection is quite different in the two cases. In the former case the infection spreads by the peritoneum, and has nothing to do with the liver, while in the latter case, the infection is carried to the liver tissue by the portal vein.

The infection, in its upward course, does not always reach the pleura, but is arrested in its progress and causes subphrenic empyema **without** consecutive pleurisy. Cases of this nature are not numerous ; they have been described by Leyden, by Mayal, Lang, and Greig Smith ; and in France have been treated in works which are reviewed in Lapeyre's monograph. Spillman has published the following case :

A youth with appendicitis was, on the seventh day, seized with dyspnoea and very sharp pain between the nipple and the false ribs on the right side ; absolute dullness in the axilla and at the right base ; punctures were negative ; acute dyspnoea and death some days later. Post-mortem : purulent tracts and subphrenic collection, estimated at 4 pints, in addition to appendicitis ; the lungs were œdematous, but the pleura was free.

Subphrenic empyema does, therefore, occur **without** concomitant pleurisy, but most often both lesions are present.

Cases in which the abscess contains gas have been quoted, and deserve therefore the name of **appendicular subphrenic pyopneumothorax**. Speaking generally, pyopneumothorax is consecutive to perforation of a neighbouring organ, to ulcerations of the stomach and duodenum, to perforation of the diaphragm, and communication with the thoracic cavity, etc. These cases of pyopneumothorax are very rare, and when gas is found in the subphrenic focus it nearly always comes from perforation of a neighbouring organ, such as the stomach, intestine, but especially the diaphragm and air-passages.

In a case published by Starcke, subphrenic pyopneumothorax was consecutive to appendicitis, but the presence of gas was explained by perforation of the diaphragm and communication with the bronchi. Greig Smith speaks of subphrenic pyopneumothorax following appendicitis. Here also the presence of gas was due to perforation of the diaphragm. Vanlair says a child presented an epigastric tumour (probably subphrenic empyema) after appendicitis. Three days later **pneumothorax** suddenly appeared on the right side ; the skin over the epigastric tumour became thin. An incision was made, letting out foetid fluid and gas. We may suppose that the gases in the subphrenic collection came from the concomitant pneumothorax.

CASE 1 (reported by Sallet).—A child had a swelling in the epigastric angle, following appendicitis. At the same time abolition of vocal fremitus, ægophony, and dullness were found at the base of the right side of the chest. Below the dull area amphoric resonance and bruit d'airain. Thoracentesis yielded foul pus. Next day lower ribs resected and the diaphragm incised. Pus and gas came out, and death supervened three weeks later. Post-mortem, the subphrenic pocket contained no pus, and the pleura only some serous fluid.

CASE 2.—A child had appendicitis ; eight days later painful swelling in the right hypochondrium ; diagnosis of subphrenic abscess, verified by operation ; death next day ; no autopsy.

Eisenlohr writes : A youth had appendicitis ; symptoms of peritonitis supervened a week later. Some days afterwards tubular breathing at the right base, marked tympanites and bulging of the hypochondrium. Patient died, and post-mortem, mild appendicitis, causing all these troubles, was found. Right side of the peritoneal cavity invaded by pus and adhesions. Between the liver and the diaphragm a pocket full of pus and gas. This pocket communicated through a perforation in the diaphragm with the right pleura, which also contained pus and gas.

Appendicular subphrenic pyopneumothorax, therefore, exists. It is ushered in by the symptoms of subphrenic abscess, given above, with bulging, tympanites of the epigastrium or hypochondrium and acute dyspnoea. It may, therefore, be difficult to know whether pyopneumothorax is supra- or subdiaphragmatic. Does the gas in the subphrenic pocket come from a



communication with the air-passages, or can a putrid collection, as in putrid pleurisy, produce the gas? The analysis of the cases which I have just quoted nearly always shows production of gas by effraction, perforation of the diaphragm, and communication of the subphrenic focus with the pleura or with a bronchus. In two cases only, this production by effraction has not been noted. It is possible that in subphrenic pyopneumothorax, as in putrid pleurisy, gas may result from putrefaction.

Surgical intervention is the only treatment in subphrenic empyema. It must be early, and is often double; it may be triple, and comprise the empyema, the pleurisy, and the appendicitis.

In a case reported by Margery three successive operations were performed: first, laparotomy, to open the abscess in the iliac region and resect the appendix; secondly, ten days later, opening of the subphrenic abscess; and, lastly, operation for empyema some days afterwards. Remarkable to relate, the case terminated in recovery.

## XV. FŒTID, PUTRID, AND GANGRENOUS PLEURISY.

This section is devoted to the pleurisy in which foul-smelling fluid is present. I propose to unite them under the general term of **ozænous pleurisies** (ὀζειν, to smell badly). The symptom which first attracts attention is the foul smell of the pleural fluid, whether it is evacuated by puncture, incision, or by vomica. A few drops drawn off by exploratory puncture are often sufficient to exhale a foul odour.

I, for my part, have seen several instructive cases which I have described in my lectures at the Hôtel-Dieu.\* We have, therefore, material for clearing up the question of **ozænous pleurisy**, which I shall divide into three groups—foetid, putrid, and gangrenous.

### Fœtid Pleurisy.

This name must be reserved for pleurisy in which the fluid is foul-smelling, while the stench is not due to putrefaction or gangrene. The term “foetid” implies that the pleurisy is neither putrid nor gangrenous. It is not putrid, for it has none of the characters of putrefaction and does not give off gas in the pleural cavity (the patient, therefore, has not pneumothorax); inoculation of the fluid does not cause gaseous inflammation, and the liquid, placed on a culture medium, produces neither fermentation nor bubbles of gas in the test-tube. The pleurisy is not gangrenous, for we find neither sloughs in the fluid nor any trace of gangrene in the walls.

This proves that the odour of the fluid is not an index of putrefaction

\* Dieulafoy, “Pleurésies Fétides, Putrides, Gangréneuses” (*Clinique Médicale de l’Hôtel-Dieu*, 1903, vol. iv., 3<sup>me</sup> et 4<sup>me</sup> leçons).

or of gangrene, in the same way as foetor of the breath and of the expectoration does not always mean gangrene of the lung. This important distinction had not escaped the notice of Laënnec and Trousseau.

The cases of sero-purulent or purulent pleurisy that are simply foetid, without putrefaction and gangrene, are so frequent that it would be commonplace to publish them. The fluid was foetid in several of the cases reported in my lectures on interlobar pleurisy.

It is to be noted that encysted pleurisy is more often foetid than putrid or gangrenous, while putridity and gangrene are common in pleurisy of the great pleural cavity. Pleurisy of the great pleural cavity may not be foetid, as is proved by the following case, which is, I believe, the only one of foetid pleurisy that has been closely examined by cyto-diagnosis and cultures, and confirmed by experimental research:

A man, twenty-seven years of age, admitted for pain in right side, obstinate cough, and foetid expectoration. Disease began six weeks before. He was taken ill with shivering fits, fever, and sharp pain in the right side of the chest. Fits of coughing soon supervened, and he brought up much yellow but odourless expectoration. The pain, which extended all over the right side, had its maximum behind, below the scapula, and the expectoration became foetid. The man looked ill; respiration rate increased, and his temperature was 103° F. The sputum glass was filled with diffuent foetid expectoration of a greyish-yellow colour. It had never been blood-stained. The breath was also foetid. The foetor was, however, not like that of gangrene. Examination of the sputum revealed neither blood, elastic fibres, nor Koch's bacilli, but numerous microbes. Threads, bacilli, cocci, diplococci, and streptococci; no pneumococci.

Dullness behind most marked over middle and inferior part of chest. On auscultation, subcrepitant râles above this zone, and at the level of the eighth intercostal space, 4 inches from the median line, cavernous breathing, but no gurgling—a fact which proves that the condition was due to consolidation of the lung, and not to cavity. Below this zone, on expiration, tubular breathing of a pleuritic tone.

Exploratory puncture gave exit to a serous fluid, which was for the time being odourless. Cyto-diagnosis showed polynuclear cells; no lymphocytosis, no endothelial plaques. The absence of lymphocytosis eliminated acute tubercular pleurisy. The absence of endothelial plaques put mechanical pleurisy out of the question. We had to do with an acute infectious pleurisy, consecutive to a similar lesion of the lung. However, one thing puzzled us: the pulmonary lesion was foetid and the pleurisy was not. The lesion of the lung resembled neither pneumonia nor broncho-pneumonia. We might have considered hepatization of the lung, resembling somewhat the infarcts consecutive to septic emboli, but we found no suspicion of such an origin.

The left side of the chest was healthy and the heart was normal, while the urine contained neither sugar nor albumin.

During the next few days the situation changed. The patient looked ill, was prostrated, and had an earthy tint. The cavernous breathing diminished and the foetid expectoration was less abundant, but the pleural effusion made rapid progress. The dyspnoea increased, the temperature rose to 103° F., and exploratory puncture yielded turbid fluid, like dirty water, and horribly foetid, although some days before it was yellowish and odourless. The absence of pneumothorax eliminated putrid pleurisy. The fluid was placed on culture medium and inoculated into a guinea-pig. As the case demanded immediate surgical intervention, Legueu resected the ribs.

The incision let out 4 pints of fluid, which resembled muddy water, and had a very



marked fœtor. No gas, no membranes, no shreds of slough. The finger could only just reach the retracted lung. No adhesions.

Result of operation excellent. In a few days the sputum became less abundant and lost its fœtid odour. Recovery complete in six weeks.

Examination of the fœtid fluid from the second puncture (Apert). The fluid was not purulent in the true sense of the word, but turbid and analogous to dirty water. On direct examination with the microscope, this fluid, which at the first puncture was rich in polynuclear cells, now contained only cellular elements in the form of granular masses, which stained badly and did not show nuclei. They were dead leucocytes, showing granular degeneration. Further, many different microbes were found, including long thin threads, thin and short bacilli, and small micrococci. All these microbes, except some micrococci, did not stain by Gram.

On aerobic cultures (agar and broth), colonies of *Staphylococcus albus*.

On anaerobic cultures (thick agar), colonies having the appearance of white lenticular points. No development of gas among these colonies, either upon glucose agar or on ordinary agar; the cultures remained odourless. Microscopical examination of these colonies showed that they were composed of masses of a small micrococcus, which did not stain with Gram, and resembled Veillon and Zuber's *Staphylococcus parvulus*.

A cubic centimetre of freshly-drawn fluid, inoculated under the skin of a guinea-pig, produced neither pus nor gas, and the animal remained in good health.

Such was the history of this case. Let us discuss it. The patient showed two stages of infection—the one pulmonary, the other pleural. Although the expectoration and the fluid were fœtid, there was no resemblance to gangrene.

Was this a case of putrid pleurisy? \* At first sight it might have been thought so, but proof is absent. All the signs of putridity were wanting: no formation of gas in the pleura, no pneumothorax, no gaseous inflammation along the track of the aspirating needle, no bubbles of gas in the test-tubes, and no gaseous lesions in the tissues of the inoculated animal.

Fœtid pleurisy remained. This man evidently had had fœtid pleurisy, set up by a similar lesion in the right lung. There is nothing to show that, had the pleurisy been left to itself, it would have become putrid.

The fluid in fœtid pleurisy is turbid or sero-purulent, and we find aerobic or anaerobic microbes in great variety, which, though little apt to produce putrefaction or gangrene, may give off a foul odour (that of *asafœtida*), just as other microbes (chromogenes) give off colouring substances.

This variety is the least formidable of the group of ozænous pleurisy; it does not rapidly cause grave symptoms (adynamia, tendency to collapse, and syncope), but it must be discovered as quickly as possible by exploratory puncture and operated on without delay.

The differentiation of fœtid from putrid and gangrenous pleurisy is not always easy at first, and the degree of fœtor is not sufficient to make a diagnosis; laboratory experiments and operation are sometimes necessary to confirm it.

\* "La Pleurésie Médiastine" (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 1<sup>re</sup> leçon).

### Putrid Pleurisy.

**Putrid pleurisy** is distinguished from the simply foetid variety by the fact that we find the signs of putrefaction—*i.e.*, formation of gas in the pleural cavity (pneumothorax) and in the test-tube; formation of gaseous inflammation along the track of an exploratory puncture in the walls of the chest; appearance of gaseous inflammation in the tissues of an inoculated animal; rapid prostration of the patient; extreme gravity of the illness; quick, compressible pulse, with tendency to collapse and syncope. Before describing putrid pleurisy, I will present some cases which give an exact idea of its course.

On October 24 a woman who was very ill came under my care. My first impression of her was bad: ghastly colour, marked fever, acute dyspnoea, profound weakness, imminent collapse, and considerable wasting, showed the gravity of the case. I was struck with the foetor of her breath; the cough was frequent, and the sputum-glass contained diffluent, sanious, greyish expectoration, which was very foetid. It was neither the purulent fluid from a vomica nor the expectoration of bronchiectasis. This quasi-adiynamic state, associated with foetor of the breath and sputum, led me to think of pulmonary gangrene. We shall see that the condition was not so.

We examined the patient. Nothing on the left side of the chest, but on the right an effusion of about 20 ounces, with moist râles above. Pleuritic effusion and a pulmonary lesion were therefore present. The onset had been very clear. On September 15 sudden acute pain on the right side of the chest. At this time the patient was at home, convalescent from an operation, performed on August 19, for double salpingitis and uterine fibroma. The uterus and ovaries had been removed. On the day of the operation she had such profuse hæmorrhage that vaginal plugging was at once performed. The loss of blood caused syncope, and for some days she was very weak. Her strength returned slowly, and on September 10, as the wound had healed, she returned home. On September 16, six days after leaving hospital and four weeks after the operation, rigors, fever, and sharp pain in the right side appeared. The cough became very painful, and, some days later, she brought up diffluent and foetid sputum. Apert found effusion in the right pleura, and drew off a pint of dirty, odourless fluid. No benefit followed. Fever and dyspnoea persisted, expectoration continued to be foetid, and the patient therefore came into the Hôtel-Dieu. I have described her condition on admission. Puncture with a Pravaz syringe yielded sanious, greyish fluid, which was so foetid that we could not but think of gangrene. Yet the diagnosis was not easy. We knew that she had a lesion in the lung, with foetid expectoration, and also pleural effusion that was foetid; we knew that the lung lesion was the older, because the foetor of the sputum preceded that of the effusion; but it remained to find out the nature and the origin of the lesions. We had to discover whether the lesions were gangrenous, or whether, under the aspect of gangrene, we had to do with putrid infection, without mortification of the tissues, which gives a very different prognosis. And, in any case, what was the entrance gateway of this infection? Had the germs been carried by the blood or by the air? These questions were of the utmost importance, and it was necessary to attempt to answer them.

Let us not forget that she had undergone an operation four weeks before the appearance of the thoracic complications; we might therefore think of embolic infection of abdominal origin. To this hypothesis it might truly be objected that the thoracic trouble had supervened four weeks after the operation, and, further, that the result of the operation had been so satisfactory as to dismiss all idea of infection. These objec-



tions carry weight, but they are not sufficient, for I could quote examples of remote embolic infection weeks after the extinction of the original focus—viz., appendicitis and otitis. Indeed, the history of **appendicitis** teaches us that remote infections of the liver, the lung, and the pleura may supervene when the appendicular focus is extinct, because the migration of the pathogenic agents takes place silently, during the active phase of the process in a closed cavity. I have discussed this question at length under Appendicitis.

The history of **otitis** also teaches that such remote infections as abscess of the cerebrum and cerebellum, and gangrene of the lung may supervene, although the otitis appears to be extinct. I have given full details in the sections on Abscesses of the Cerebellum and on Gangrene of the Lung.

In the same manner the course of events after a confinement gives material for careful reflection. The confinement has been quite normal, the most rigid asepsis has been observed, no apparent infection has been discovered, and yet microbial emboli may produce septic infarcts in the lung, with or without purulent pleurisy. I insist, therefore, on the fact that an ill-extinct infecting focus, whether it be abdominal, appendicular, aural, uterine, or elsewhere, may be the origin of severe infections.

It might therefore be asked if the thoracic lesions in my patient had originated from some abdominal focus. We shall see that nothing of the kind had occurred.

In the course of our interrogation she had spoken of a foetid vaginal discharge which had lasted for a month, in spite of daily injections. Digital examination was made, and a large mass, enclosed, as it were, in a funnel, was felt at the fundus vaginae. Examination with the speculum revealed a greyish mass, surrounded by oedematous mucous membrane. The parts were bathed in sanious fluid, which was horribly foetid. After repeated douches, the mass, which proved to be the forgotten plug, was seized with a pair of forceps and with-drawn.

After the extraction of this foreign body, which had remained so long *in situ*, about 3 ounces of foetid fluid came away. After copious douches the mucosa of the fundus vaginae showed shallow ulcerations, with sanious base and red edges, which were clean cut. The ulcerations bled readily, when the speculum was opened. We now held the offending body, and could understand the chain of events. The fundus vaginae had been transformed into a closed cavity by the forgotten tampon. In this culture medium ulcerations occurred. The microbes, in the form of septic emboli, entered the venous system, and set up foci in the lung and the pleura.

The removal of the tampon was followed by a fall of temperature, as was natural, since we had just removed the infective focus; but the condition of the patient remained just as bad, and the fever reappeared, because the thoracic lesions continued their course.

Were these lesions putrid but not gangrenous? This question had to be elucidated, but for the moment the urgent indication was to suppress the pleural focus, and to see what could be done for the pulmonary one.

I asked Marion to operate. A pint of extremely foetid fluid gushed out, but the pus contained no trace of sloughs.

Their absence eliminated the hypothesis of pleural gangrene, just as the absence of elastic fibres and sloughy elements in the sputum excluded gangrene of the lung.

After the operation the fever fell, and the general condition rapidly improved. The foetid expectoration ceased in ten days. Three weeks later the patient was completely cured. See temperature chart on p. 372.

Apert examined the foetid fluid from the vagina, the sputum, and the fluid from the pleura. These three foci contained **identical microbes**, which were both aerobic and anaerobic, though the latter were the more important.

Cultures gave the following results:

**Aerobic Cultures.**—Those made with the pus from the vagina and the pleura were

very small. About a dozen tiny colonies were visible, and were formed by a streptococcus, with irregular granules which stained with Gram. Those made with the sputum yielded the same streptococcus, some colonies of *Staphylococcus aureus*, and a large white yeast fungus.

**Anaerobic Cultures.**—Anaerobic cultures were made with samples from the vagina, the pleura and the sputa: (1) on broth, in Pasteur's pipettes; (2) on agar in Liborius-Veillon's tubes. The broth rapidly became cloudy, a foetid odour developed, and bubbles of gas rose to the surface, when the tube was shaken. No streptococci were found on microscopical examination, though a large number of bacilli and fine cocci were visible.

Liborius' tubes were sown with successive dilutions (a drop of fluid put in the first tube, a drop of this diluted fluid placed in the second tube, and so on). In the first dilutions the colonies were too close together to be isolated. On the third or fourth day gas was generated, which split up the agar.

In further dilutions it was easy to study the two species in tubes sown with pus from the vagina, the pleura, and the sputum. One of these species presented the form of white, opaque, lenticular colonies, due to a large slightly elongated coccus (coccus-

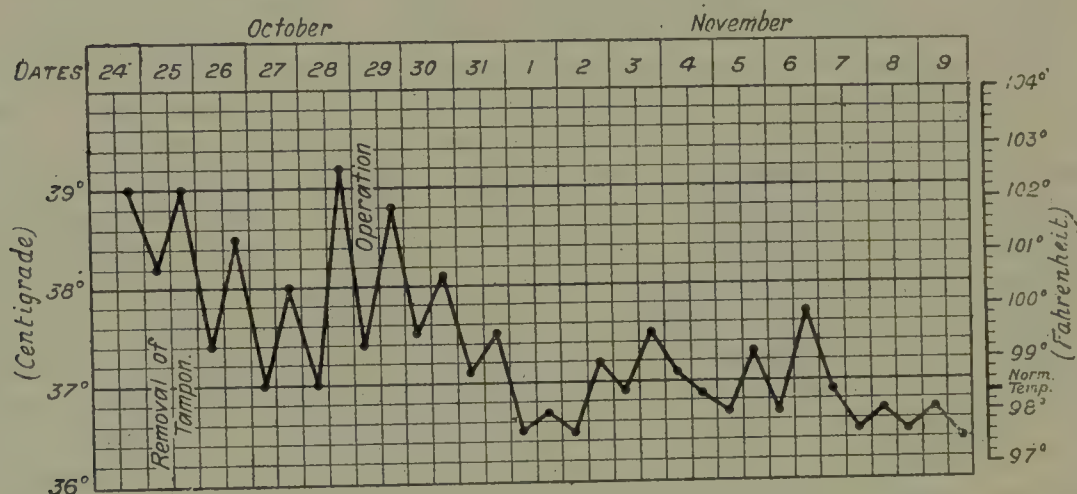


FIG. 21.—TEMPERATURE CHART.

bacillus), which was isolated or disposed in pairs and masses, and stained with Gram. The other species showed itself in the form of yellowish muriform colonies, due to fine cocci in mass. Experiments were made upon animals. One guinea-pig received under the skin  $\frac{1}{2}$  c.c. of vaginal pus, and another  $\frac{1}{2}$  c.c. of pleural pus, without result. A rabbit received  $\frac{1}{2}$  c.c. of pleural pus in the veins, without any other result than serious indisposition, which yielded after two days. These researches allowed us to reconstruct the nature and the course of the infection. The disease occurred in two stages. The toxi-infection started from the fundus vaginae. Hallé has shown that the vagina normally contains aerobic microbes in the form of a streptococcus, differing from the *Streptococcus pyogenes*, and strictly anaerobic microbes, which, after inoculation of animals in pure culture, cause abscesses and gangrene. The aero-anaerobic toxi-infection had therefore arisen in the plugged vaginal cloaca. The infectious germs had been carried by the veins to the right heart, thence to the lung, and had passed into the pleura. Infection of the lung showed itself by rigors, fever, and pain in the side. An ill-defined area of lung had been embolized and converted into a putrid, but not gangrenous, infarct. The pleural fluid, which was odourless at the first puncture, was putrid at the second one. We thus built up the vaginal, pulmonary, and pleural stages of the infectious process.

In this series of infections the infection was putrid, but not gangrenous. It was :



putrid, as proved by the production of gas in the anaerobic cultures. It was not gangrenous, for we drew off from the pleura no sloughing shreds. An analysis of the sputum revealed neither fibres nor sloughs. Here the process of putrefaction was not associated with mortification.

The history of **appendicitis** furnishes us with many cases of putrid and of gangrenous pleurisy. I have discussed **appendicular pleurisy** in a preceding section. It may, indeed, be said that appendicitis is one of the most frequent sources of purulent pleurisy. As a model let me mention the following case :

A man came to us moribund, with extensive right pleurisy, complicated by pneumothorax, and died some hours before the operation. His history and the autopsy enabled us to reconstruct the morbid picture. The pleura contained 7 pints of foul-smelling pus and gas. Pleurisy and pneumothorax were the result of appendicular infection. The lesions, which started from the appendix, began in the right part of the abdomen, and reached the right thoracic cavity. The putrid infection here was not of embolic origin, as in the preceding case, but the lesions spread by continuity.

I think it useful to quote cases of putrid pleurisy :

**Widal's Case.**—Man suddenly taken ill with acute pain on the right side of the chest and violent dyspnoea. The pain, after relief from an injection of morphia, reappeared. The dyspnoea increased, the cough became paroxysmal, the general condition grew worse, and the patient was admitted under Widal. At the left base, dullness and tubular breathing (effusion); above, tympanites and amphoric breathing (pneumothorax). The dyspnoea was so acute that a puncture was at once made, and gave exit to 2 pints of puriform fluid of extremely foetid odour.

No improvement. Next day the signs of hydropneumothorax were complete. Around the puncture a bright red swelling developed, which on pressure yielded gaseous crepitation. The gaseous inflammation commenced some hours after the puncture. Operation decided upon, but the patient died before the surgeon's arrival.

Post-mortem : On opening the thorax, greyish, sanious, and extremely foetid fluid, mixed with gas, escaped from the left pleura. The pleuræ, after removal of the false membranes, were carefully examined. These membranes were fibrinous and not adherent. No trace of gangrene found on the serosa. The left lung was also examined with the greatest care : no tubercules, no foci of broncho-pneumonia, no gangrene.

This case, then, is one of putrid pleurisy without gangrene. The pathogenic agent was the *Proteus vulgaris*. A guinea-pig was inoculated under the skin with 1 c.c. of pleural fluid. Next day a large pocket developed at the point of inoculation, and gave very evident gaseous crepitation on palpation. On the skin which covered this gaseous abscess an ulcer soon formed, and gave exit to sanious and foetid fluid containing various microbes, chiefly the *Proteus vulgaris*.

**Courtois-Suffit's Case.**—A man, twenty-three years of age, who had had chills and pain in the side at the right base, came into the Beaujeon Hospital. Right pleurisy with effusion, estimated at 2 pints. In a few days the dyspnoea was marked, the patient became cyanosed, and thoracentesis was necessary. The puncture gave exit to about 1½ pints of purulent fluid, which was extremely foetid. Next day, at the seat of puncture, an cedematous and reddish swelling appeared, which had the appearance of a phlegmon, and extended as far as the base of the thorax. On palpation, fine crepitation, due to gaseous infiltration. During the next few days the situation grew worse: the temperature was about 103° F., sweats were abundant and foetid, the face was bloated and earthy, and the dyspnoea acute. Thoracotomy gave exit to fluid pus, which was extremely foetid and of a brownish tint. The pleural cavity was then washed out with a very

weak solution of permanganate of potash. After some ups and downs the patient died.

The results of the autopsy were : In the chest-wall a gaseous inflammation around the seat of the puncture, pleural cavity contained a little fluid, pleura thickened, but **no trace of gangrene** in the lung or the pleura.

**Boinet's Case.**—Well-built man, twenty-four years of age, seized with rigors, cough, and acute pain at the lower part of the left side of the chest. Left pleural effusion, with amphoric breathing and metallic tinkling (pyopneumothorax). The situation rapidly became worse. Puncture yielded sanious pus of extremely foetid odour. Next day operation for empyema gave exit to 4 pints of stinking pus and gas.

Patient died twelve days later. Post-mortem, Boinet found three pockets in the pleura, containing putrid fluid mixed with gas. No communication with the lung, no tubercular lesions, and **no gangrene** in the lung or the pleura.

**Netter's Case.**—Child with pyopneumothorax. Puncture gave exit to foetid pus and gas. **Subcutaneous emphysema** then developed. Broca operated for empyema, and the child made excellent progress. With regard to these cases of putrid pleurisy, Netter says that he has found an anaerobic bacillus in the form of long, thin filaments, mixed with other micro-organisms.

**Description.**—These cases give a clear idea of putrid pleurisies. They chiefly affect the great pleural cavity, unlike foetid pleurisy, which is usually encysted. The fluid is sero-purulent, turbid, greyish, and not homogeneous ; when placed in a test-tube, it divides into two layers—the lower dense and opaque, the upper more transparent.

These cases frequently have an embolic origin ; the original focus may be in the vagina or the appendix, as was the case with our patients, in otitis, in osteomyelitis, in phlebitis, etc. In some cases putrid pleurisy results from neighbouring lesions (lung or mediastinum). It may be set up by abdominal mischief, renal or hepatic suppurations, subphrenic empyema, with or without perforation of the diaphragm. In other cases the cause and the origin remain unknown.

The microbes, which are chiefly anaerobic, lead to the formation of gas (putrefaction) in putrid pleurisy. The appearance of pneumothorax without perforation of the pleural cavity is thus explained. This pneumothorax, formerly called essential, is quite different to pneumothorax by perforation. The latter is due to the introduction of air into the pleura, while the former arises without the presence of air.

From the clinical point of view we find in both forms the same physical signs—viz., tympanites, amphoric breathing, metallic tinkling, and hippocratic succussion.

Gas may form in the walls of the thorax and produce gaseous phlegmon. Simple aspiratory puncture sows the germs in the chest-wall, and a gaseous phlegmon with oedema follows in a few hours.

Experimental research yields analogous results. The inoculation of a drop of pleural fluid in the cellular tissue of an animal provokes gaseous phlegmon (Widal).



Lastly, the development of gas may be abundant in anaerobic cultures of the pathogenic agents of putrid pleurisy.

The **diagnosis** of putrid pleurisy is impossible before puncture. Every case is accompanied by general symptoms, which rapidly become grave, and comprise small, quick pulse, dyspnoea, prostration, and collapse—symptoms which are rarely found in other varieties of pleurisy. In such a case the additional presence of pneumothorax is in favour of putridity, but yet the putrid nature of the effusion can only be established by exploratory puncture. Accordingly, if we are in doubt, even though the effusion is scanty, we must confirm the diagnosis by early puncture. Furthermore, puncture should immediately be followed by thoracotomy, with or without resection of ribs, for the needle track may rapidly become infected, and diffuse gaseous inflammation may develop in a few hours.

But it will be said puncture only shows us the foetor of the fluid; it does not at once reveal putridity. To this I would reply that it matters little whether the pleurisy be foetid or putrid: **every foul-smelling pleurisy should be operated on without delay.**

### Gangrenous Pleurisy.

The phenomena of putrefaction just described are common to putrid and to gangrenous pleurisy. These two varieties are distinguished, not by the foetor of the fluid, but by the shreds of slough which float in the effusion or are adherent to the walls.

The description of putrid and gangrenous pleurisy present many similar points, yet the latter may assume two forms which are of importance: sometimes the lung is not involved; at other times the gangrene is pleuropulmonary, and is then far more grave. I shall here quote cases of the former variety:

**Comby and Vogt's Case.**—Young girl, eleven years of age, taken ill with left pleurisy. Some days later she woke up with true orthopnoea. Next day Comby and Vogt found extreme dyspnoea, temperature 104° F., signs of effusion at the base of the left chest, and above those of pneumothorax. First diagnosis was pneumothorax from tubercular perforation of the lung. Four days later puncture gave exit to a pint of very foetid pus. The first diagnosis was then changed to gangrenous pyopneumothorax. Comby incised the seventh intercostal space, and came on a mass of false membranes, which he broke up with the finger. Four or five pints of horribly foetid pus came out. The child finally recovered.

**Rendu's Case.\***—A man who looked phthisical was admitted for pneumothorax. He was wasted, cyanosed, and much distressed; cough frequent, breath had no odour, and temperature was normal. Pleural effusion at the right base and above pneumothorax. Succussion splash and metallic tinkling. This condition was said to have begun with violent pain in the right side. On the day after admission his condition

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\* Meeting of February 3, 1899. Although the author has entitled his paper "Putrid Pleurisy," the putrid pleurisy was in reality gangrenous, because he found "a large shred of sloughing tissue, in which elastic fibres were discovered under the microscope."

was alarming. Dyspnœa was acute, cyanosis was marked, and the heart was displaced; puncture yielded extremely foetid pus. Rist at once operated. One and a half pints of foetid pus came out, and the cavity was washed out with a solution of permanganate of potash. Two days later painful œdema with emphysematous crackling was found at the right base. An incision was made, and very foetid serous fluid, mixed with bubbles of gas, let out. Later a phlegmonous patch of the same nature appeared on the left arm, at a spot which had been vaccinated some days before. The condition of the patient became very alarming. Restlessness and delirium were acute; the pus from the empyema again became very foetid, and washing-out of the pleura brought away a large slough of blackish gangrenous tissue, which was extremely foetid, and showed elastic fibres under the microscope. Patient finally recovered. Bacteriological examination showed the presence of anaerobes.

Let us now turn to those cases in which we find gangrene of the lung and of the pleura; it is usual for gangrene of the lung to precede that of the pleura. To avoid repetition, I would ask the reader to turn to the section on Gangrene of the Lung.

Pleuro-pulmonary gangrene, like that of the lung, may have an embolic (otitis, appendicitis, etc.) or an aerial origin. As the points have been discussed under Gangrene of the Lung, I shall not repeat them.

The descriptions of gangrenous and of putrid pleurisy in part blend. Acute pain in the side, fever, bad pulse, earthy tint of the skin, dyspnœa, prostration, and tendency to collapse, show the extreme gravity of gangrenous pleurisy; foetor of the breath, stinking and blood-stained sputum, show the participation of the lung in the gangrenous process.

As regards prognosis, the addition of gangrene marks a further step in the gravity of putrid pleurisy; but in reality these two varieties are so closely allied that they almost come under one description. The same aero-anaerobic agents which produce pleurisy—that is simply putrid—may cause gangrene either in the patient or in animals used for experimental research. In Widal's case the gangrene was absent, but yet inoculation of the pleural fluid in a guinea-pig "produced a gaseous and gangrenous abscess in the animal, which died after extensive sloughing of the skin of the abdomen and thorax." In Rendu's case the pleurisy was labelled putrid, but yet a gangrenous shred was found later in the thoracic cavity. The line of clinical demarcation between putrid and gangrenous pleurisy is, therefore, not absolute; putrefaction and mortification may appear together or in succession.

The spread of gangrene from the pleura to the lung can only be recognized by foetor of the breath and of the expectoration, and by the presence of elastic fibres and sloughs in the sputum. Such is the history of **stinking** pleurisies and their three groups—foetid, putrid, and gangrenous: the foul smell of the effusion, which is the first step in diagnosis can only be revealed by early puncture, and immediate surgical intervention is the only method of treatment.



## XVI. VOMICÆ.

**Definition.**—Considering only its etymology, the word **vomica** (from **vomere**, to vomit) is improperly applied to the symptom which we are about to describe; custom, however, has consecrated it, and the word **vomica** serves to describe the rejection of pus by the respiratory channels, just as hæmoptysis denotes the rejection of blood from the same passages. Custom has even gone farther, and, by an abuse of language, has finally included under one term the symptom and the lesion, so that we speak of pulmonary, pleural, or hepatic vomica—that is to say, a purulent collection in the lung, pleura, or in the liver, which has burst into the bronchi and has been coughed up.

**Description.**—The complete study of vomicæ comprises the diagnosis both of the symptoms and of the lesion. I shall here outline the chief varieties.

1. **Pulmonary Vomica.**—Pneumonia suppurates fairly often (grey hepatization), but the pus very rarely collects in the form of an abscess. The works of Laënnec, Graves, and Trousseau, show how rarely pulmonary abscesses occur in the course of pneumonia; they are so rare, indeed, that Grisolle has only collected twenty-three proved cases. These abscesses sometimes form very rapidly, on the fifth day of pneumonia (Woillez), or on the twelfth, and are never rejected later than the twentieth day.

The pus from the pulmonary vomica is scanty, phlegmonous, sometimes mixed with blood, and of a brownish colour. The pneumococcus is the pathogenic agent. Directly after the evacuation of the abscess the physical signs change, and where the signs of pneumonia have been present we now find cavernous breathing and splashing sounds.

2. **Pleural Vomicæ.**—These vomicæ are much more frequent and follow interlobar, mediastinal, or diaphragmatic pleurisy, or pleurisy of the great pleural cavity. The vomica of interlobar pleurisy is the most frequent of all. Speaking generally, vomicæ of the pleura appear much more slowly than those of the lung. Purulent collections in the pleura open into the bronchi after three to six weeks, and later still when the great pleural cavity is affected. As an exception to this rule, when purulent pleurisy develops in a child, or in a woman during the puerperal state, the vomica may appear from the second to the third week (Trousseau).

The symptoms are different, according as the vomica is provoked by partial pleurisy, which only contains some ounces of pus, or by pleurisy of the great pleural cavity, which may contain several pints.

In encysted diaphragmatic or interlobar pleurisy the quantity of usually foetid pus brought up at the moment of the vomica does not exceed some ounces; it then gradually diminishes, and, in fortunate cases, the pleuro-

bronchial fistula closes. This mode of cure is possible in metapneumonic interlobar pleurisy. I would refer the reader to the section on **Interlobar Pleurisy**.

In the case of the great pleural cavity, when the pleura contains 4 or 5 pints of pus, the course is less simple. The bursting of pus into the bronchi often determines dyspnoea, bordering on asphyxia. Fatal cases have been quoted, and the patient, who has extreme distress, brings up streams of purulent fluid through the mouth and the nose. The first evacuation is generally followed by improvement, and then the patient, while changing his position or coughing, continues to bring up some ounces of pus. He no longer appears to vomit, but only to cough up the pus. As soon as a certain quantity of pus has accumulated in the pleura, the patient is seized with fits of coughing, and voids the pleuritic fluid five, six, or ten times a day. Sometimes the evacuation of pus stops, but is repeated one or more days later, and in some cases the breath and the fluid evacuated become horribly foetid.

Unless the fistula is so constituted as to **form a valve** (Chomel), the inspired air enters the pleural cavity, and the signs of **pyopneumothorax** are found.

Does pleurisy become purulent when the vomica has once formed? Several modes of termination may occur; one is recovery, which is rare in general but possible in interlobar pleurisy, especially in the metapneumonic variety.

In some patients the improvement consecutive to the vomica is only transient; the cavity becomes infected, and the fever does not cease, while the patient loses appetite, becomes cachectic, and finally succumbs. In other patients the cavity shrinks, but the pleura and the lung are invaded by fibrosis, with or without dilatation of the bronchi, which, sooner or later, compromises the individual's life.

The preceding description will have made it apparent that the pleural vomica only occurs in empyema. There are, however, some extremely rare cases in which sero-fibrinous pleurisy is terminated by vomica.

**3. Vomica with Hydatid of the Lung.**—The pleural vomicae which I have just described are the most frequent; there are, however, some rarer varieties, which I shall now review. Suppurating hydatids of the lung may cause vomicae which simulate pleural vomica, with this difference, however—that we find fragments of hydatid membranes and hooklets in the rejected matter.

**4. Vomica in Congestion Abscesses.**—Chenieux's paper contains nine cases of vomicae, consecutive to congestion abscess, following Pott's disease. The pus often contains bony sequestræ.

**5. Vomica from Suppuration of the Liver.**—Abscesses and suppurating



hydatid cysts of the liver may result in vomica ; adhesions are established by the intermediary of the diaphragm, perforation follows, and the patient voids the liver abscess through the bronchi. When the vomica has its origin in an abscess of the liver the pus is reddish, thick, and sometimes foetid ; when the vomica is consecutive to a suppurating hydatid of the liver, the purulent fluid is mixed with hydatid membranes.

**6. Vomica with Suppuration of the Kidney.**—Suppurating cysts of the kidney and pyonephrosis may terminate by vomica.

It is not customary to consider as a true vomica the quantity of pus which may be brought up at one time by a patient with dilatation of the bronchi ; it is, however, a pseudo-vomica of which the distinctive characters should be well known.

The **treatment** of vomica varies with its causes, and **surgery** gives the best results.

## XVII. CHYLIFORM AND CHYLOUS EFFUSIONS OF THE PLEURA.

The pleura, like the peritoneum, may contain milky effusions which have the appearance of an emulsion. On thoracentesis we expect to find a sero-fibrinous or purulent effusion, but we draw off chyliform fluid. This fluid is odourless, and has no tendency to coagulate, because it is not fibrinous ; in a test-tube it forms no deposit in its lower part, and will keep for days or weeks without putrefaction ; under the microscope it shows some leucocytes, and in most cases contains a large number of fine fatty granules, soluble in ether, while clinical analysis reveals a large increase of fatty matter.

The onset may be ushered in by acute pain in the side, but is more often unnoticed. The collection increases insidiously, like a subacute pleurisy. This latent period may last eighteen months. The effusion at length causes distress, displaces organs, especially the heart, and flattens the lung. It may become a cause of dyspnoea, but is not accompanied by the general symptoms of fever which are so frequent in purulent effusions.

Chyliform effusion shows no tendency to absorption. After it has been evacuated, it recurs obstinately. Punctures relieve the patient, who thinks himself cured ; but the fluid slowly reforms, and fresh evacuation becomes necessary after a more or less extended period, during which every morbid symptom has apparently disappeared.

Chyliform effusion at times gives way to empyema, in which case the staphylococcus is usually in evidence. Fever appears, with pain in the side ; the chest-wall becomes oedematous ; the effusion rapidly increases ; the fluid makes its exit by vomica, and death soon follows. More rarely chyliform effusion recovers after first becoming sero-fibrinous.

The clinical picture just sketched is that of tubercular chyliform pleurisy ; indeed, tuberculosis is the cause in two-thirds of the cases. I have had a remarkable example.

Patient admitted in May, 1899, to the Hôtel-Dieu, with an effusion of 3 pints, which had been practically latent. His general condition appeared excellent : no wasting, no fever ; if he had not suffered with severe dyspnœa, he would not have given up his work. About 40 ounces of characteristic chyliform fluid were drawn off. He felt better, and asked to go out. He was, however, persuaded to remain for some weeks. In three weeks 60 ounces were drawn off by successive punctures. The effusion at length seemed to dry up, and the patient went out, persuaded that he was cured. We saw him again five years later, when he came to be treated for sciatica. He was working without feeling any malaise. He looked well, did not cough, and only found that he readily became breathless, and at times felt some pain in the chest. The physical signs of effusion, however, were present, and thoracentesis yielded similar fluid to that drawn off five years before. His condition remained good for about a month, but then, without appreciable cause, his temperature ran up, and severe pain in the side developed. Exploratory puncture showed the chyliform effusion had been replaced by purulent fluid, containing staphylococci. He died four months later.

In other more rare cases the disease runs a rapid course, but the effusion is rather lactescent than chyliform, and develops in the course of confirmed phthisis. I have recently seen such a case in which the fluid was in turn sero-fibrinous, serous, lactescent, and purulent. The patient died in ten months.

While tuberculosis is present in two-thirds of these cases of lactescent effusion, cancer is only present in one-third. At times the patient has had a growth for a long while, and the chyliform effusion is only a secondary condition. At other times the pleuro-pulmonary cancer is primary. The onset of the malady is almost silent. Both acute dyspnœa and sharp pains attract the patient's notice, and we find a very large effusion, which, on puncture, shows all the characters of chyliform fluid. In some cases a few red corpuscles may be present. The fluid forms again with extreme rapidity, and we may find, after evacuation, that the resonance does not return to normal. In a short while puncture gives no relief : the aspirating needle has to travel through a much thickened wall. At last puncture yields only a little bloody fluid, and the signs of compression, the dyspnœa, and the pain still persist. The patient wastes and grows weak, and fever may appear. Such a picture is quite opposed to that of tubercular chyli-form pleurisy.

It is always easy to recognize the nature of chyliform pleurisy by the appearance of the fluid, by its microscopical analysis, and by its chemical composition. It is, however, more difficult to ascertain its pathogenic cause. If the patient has proved phthisis or evident local tuberculosis, the diagnosis will naturally be in favour of tubercular effusion. On the other hand, the existence of visceral cancer, rapidly progressive course, signs of pressure in the mediastinum, with œdema and collateral circulation,



enlarged glands in the axilla, and the appearance of phlebitis, all point to cancerous effusion. The final appeal must always be made to laboratory methods. Cyto-diagnosis, as a rule, shows only a few cells in a case of cancerous effusion. Nattan-Larrier found neoplasm cells, while endothelial plaques were discovered in a case of lactescent effusion, which supervened in the course of leucocythæmia (Sicard and Monod). Inoculation into the peritoneum, or better, into the mamma, of the guinea-pig will often prove that the chyloform fluid is tubercular. This method was employed in two cases under my care.

The causes of chyloform effusion in the pleura have been as much discussed as the causes of chyloform effusion in the peritoneum, and are quite comparable in each case. The older writers thought that the effusion of chyle in the pleura followed rupture of the thoracic duct. This opinion no longer holds good. In one of Debove's cases the thoracic duct was healthy, and a similar finding occurred in two of my cases. Granulofatty degeneration of the chronically thickened layers of the pleura (Quincke) has also been invoked. In Debove's case the pleura was covered with tissue some millimetres in thickness, made up of several superposed layers, which showed numerous fatty granules under the microscope. In one of my cases Nattan-Larrier found very dense mature fibrous tissue, which had undergone hyaline degeneration in places. Every trace of fibrinous exudate had disappeared; the cellular infiltration was but little marked; the vessels, though scanty in the superficial layer of the neo-membrane, were numerous in the deep parts. Some giant cells were also present near the lung, which was sclerosed.

Recent researches seem to show that the chyloform aspect of the fluid results from disintegration of its cellular elements (leucocytes or cancer cells). As the result of a cause which has so far escaped us, the cellular elements in suspension in the fluid are said to degenerate, and give rise to fatty and albuminous fluid if the disintegration is incomplete, and to purely fatty fluid if the transformation is perfect. This hypothesis has been confirmed by Nattan-Larrier, who has seen *in vitro* the serous fluid from cancerous pleurisy change aseptically into lactescent fluid, while the number of cells rapidly diminished.

**Chylous Effusion.**—Apart from the undoubted cases in which the effusion is not chyle, we find others which are true cases of chylothorax, the effusion being chylous. Shaw has collected twenty-cases. Traumatism was the cause in one-third of these cases. The others were said to result from an obstacle to the lymph-flow, with or without rupture of the thoracic duct, or of the great lymphatic vessels (cancerous glands or lymphadenoma in the mediastinum, obliteration of the thoracic duct, thrombosis of the subclavian vein). The diagnosis is easy when the effusion follows trauma.

A man had his chest caught between the driving-wheel of a steam-engine and a wall. He was taken to the hospital, where the left clavicle and several ribs were found to be fractured. Two days later effusion was discovered in the left pleura. Exploratory puncture yielded milky fluid of a rosy colour. Chemical and microscopical examination showed the presence of chyle, mixed with blood. Ten days later the red corpuscles had disappeared, and the effusion had the appearance and composition of pure chyle. Absorption took place without complications, and the patient recovered fairly rapidly (Handmann).

In cases of this nature the effusion results from rupture of the thoracic duct. It is situated on the left side (Wiesinger, Kummel). It has the histological composition of chyle, but its chemical composition varies with the nature of the fluid and the time of digestion; glucose and peptones may be found in it, and the proportion of fat will increase considerably after the ingestion of butter (Strauss). In cases of this nature, when the rupture is due to an injury, the prognosis is not bad.

Let us now note a third group of cases, in which the lactescence of the effusion is not the result of a local infection, but the consequence of a general alteration in the fluids of the organism. It is in cases of this kind that we see the coexistence of identical effusions in the various serous cavities; chyloform effusions have been seen at the same time in the peritoneum, the pleura, and the pericardium (Bramwell). The blood-serum (Achard), the serous fluid from blisters, and the liquid from oedematous areas may be opalescent. This opalescence of serous effusions is comparable to the opalescence of the blood-serum described by Widal. The prognosis depends upon the general condition.

Finally, in some cases chyloform effusion into the pleura may result from the presence of the *Filaria sanguinis* (Lancereaux).

### XVIII. HYDROTHORAX.

**Hydrothorax** is hydrops of the pleura. Both pleuræ are often affected, and the fluid is analogous to blood-serum, and very poor in corpuscles. The condition is a kind of serous transudation analogous to that of œdema.

Hydrothorax is the result of mechanical causes, of which the most common are lesions of the mitral orifice, and of dyscrasiæ, of which the most usual are cachexia and Bright's disease.

Hydrothorax is therefore only a symptom which supervenes as a complication in the various morbid states I have just enumerated. It establishes itself in an insidious fashion, without fever and pain; its physical signs resemble fairly closely those of pleuritic effusion. The cyto-diagnosis has been given under Pleurisy.



## XIX. PNEUMOTHORAX—HYDROPNEUMOTHORAX.

**Pneumothorax** (Itard) implies the presence of air or of gas in the pleural cavity; if fluid is also present, the lesion takes the name of **hydropneumothorax**, and if this fluid is pus, the case is one of **pyopneumothorax**.

**Pathogenesis.**—There are two chief varieties of pneumothorax: pneumothorax by perforation, and pneumothorax by putrefaction.

The former is the more frequent, and results from the passage of air into the pleural cavity. Injury, wounds of the pleura and lung, tuberculosis, emphysema, strains, infarcts, superficial hæmorrhagic nodules, general and interlobar purulent pleurisy, foci in the peribronchial glands, cysts, or abscesses of the liver and of the kidney, are the usual causes of these pleuropulmonary perforations.\*

**1. Strain and Emphysema.**—Pneumothorax may appear suddenly, following a strain, whether the patient have emphysema or not. An emphysematous patient may also be suddenly attacked by pneumothorax, even though he has made no effort. In these various cases the rupture of the air-vesicles permits the air to reach the pleural cavity, the vacuum is destroyed, the lung shrinks and collapses, and pneumothorax is produced. Galliard has collected thirty-seven cases of pneumothorax from strain.

A bugler was suddenly taken ill with pneumothorax while vigorously blowing his instrument. A clergyman was seized with pneumothorax during a fit of hearty laughter. A man was taken ill with pneumothorax while raising a chair with his arm extended. A student was seized with pneumothorax while dancing vigorously.

In this variety of pneumothorax the onset is sudden, with tearing pain in the thorax, and very acute dyspnoea. The classical signs are at once evident—tympanitic resonance and amphoric breathing. Although terrible at its onset, this form is not grave, for there were only three deaths in thirty-seven cases (Galliard). The pneumothorax remains pure, the presence of fluid (hydrothorax) is very rare, and suppuration (pyothorax) is the exception. The perforation usually becomes obliterated, the air effused into the pleural cavity is absorbed, the lung resumes its function, and recovery follows in a few weeks.

**2. Tuberculosis.**—Tuberculosis is the most usual cause of pneumothorax. Perforation of the lung may supervene, either at an advanced stage (cavity and softening) or at the onset during the first stage. Cases have, indeed, been quoted, and I have seen them myself, in which pneumothorax

\* Saussier, who has collected 131 cases of pneumothorax, classifies them, according to their causes, as follows: Pulmonary phthisis, 81; pleurisy, 21; gangrene of the lung, 7; emphysema, 5; pulmonary apoplexy, 3; cancer, 1; abscess of the lung, 1; hydatid of the lung, 1; hepatic abscess, 1 ("Recherches sur le Pneumothorax," Paris, 1841).

occurred in an apparently healthy person, and was the precursor of tuberculosis that had so far shown no symptoms. In such a case tubercular pneumothorax resembles that due to emphysema or to strain. The pathogenic diagnosis is extremely difficult. How are we to know whether pneumothorax, supervening suddenly in an apparently healthy subject, is or is not tubercular? We may perhaps use, as a control measure, an injection of 2 milligrammes of tuberculin. This means has been employed by Chauffard, and the absence of reaction in his patient proved that the pneumothorax was not tubercular.

It was formerly held that purulent change in an effusion following tubercular pneumothorax was of necessity due to secondary invasion by other micro-organisms: Friedlander's bacillus, *Staphylococcus aureus*, *Bacillus saprogenes*, etc. (Netter).

These conclusions are no longer admitted. Observations have shown that tubercular empyema may result solely from the infection of the tubercle bacillus without the presence of other micro-organisms. I have devoted a clinical lecture to "purely tubercular pyopneumothorax without super-added infection," and to the cases already observed I have added others which leave no doubt as to the pathogenesis of the pyopneumothorax.

Speaking generally, pneumothorax is more frequent in adults than in children; it has, however, been seen in children of all ages, even in those under two years of age; "but the influence of age varies, according to the causes of the pneumothorax. Tubercles produce perforation at any age; the rupture of the vacuoles of broncho-pneumonia determines it exclusively between the ages of two and four years."

The presence of air in the pleural cavity is generally followed by hydrothorax, and in 147 cases collected by Moneret the pneumothorax remained pure in sixteen only; fluid formed in all the others. Some cases have been noted in which the fluid remained serous for a very long time. I had a patient under my care in whom the fluid of a tubercular hydropneumothorax remained free from any micro-organism for eight months. These cases are exceptional; most frequently the fluid becomes purulent, and contains the microbes of suppuration, especially when the perforation is consecutive to a tubercular cavity or to an abscess; in some cases the fluid became purulent, although there was only simple rupture of the pulmonary alveoli (strain or emphysema).

The gases in the pleura vary in quantity: nitrogen and carbonic acid predominate, oxygen being in the smallest proportion, while sulphuretted hydrogen usually accompanies pyopneumothorax.

**Post mortem** one or several perforations are found; their size, shape, and situation vary, according to the cause. When the perforation is not readily visible on the surface of the lung, it must be made so. For this purpose



the pleural cavity is filled with water, and insufflation through the trachea is performed. The bubbles which appear at the mouth of the fistula indicate its position; the pleuro-pulmonary fistula, however, may be cicatrized or obliterated, and the corpus delicti passes unnoticed.

I have elsewhere described the lesions which cause pneumothorax.

**Symptoms.**—The invasion of pneumothorax is violent or quiet, according to the cause of the perforation. When the irruption of air affects a healthy pleura free from adhesions, the lung collapses, and the symptoms are sudden; the **pain in the side** is severe, and the **dyspnœa** is excessive. Under other circumstances, when the lung is already fixed by adhesions, the onset is less violent, the pain and dyspnœa are slower to appear, and are less severe; in some cases, indeed, the disease is latent. When pneumothorax results from the opening of a purulent collection into the bronchi (vomica), entrance of air into the pleura may follow the voiding of the pus through the bronchi.

The physical signs of pneumothorax are as follows: Mensuration shows enlargement of the thorax, unless chronic pleurisy has already caused retraction. Percussion yields a clear and tympanitic sound, metallic in timbre (Trousseau's *bruit d'airain*)\*. The vocal fremitus is diminished or abolished, and the heart may be displaced by gas when the pneumothorax is situated on the left side. On auscultation the breath-sounds, the cough, and the voice-sounds become amphoric, and the râles change to a silvery sound, known as metallic tinkling. There is, furthermore, a sign which I have long pointed out: If the patient is made to drink small mouthfuls of fluid during auscultation, the swallowing of the fluid produces a gurgling sound of amphoric timbre. The above signs result from the presence of air in the pleura; the simultaneous existence of fluid and of gas in the pleura shows itself by a splashing noise, audible on auscultation even at a distance, provided the patient be gently shaken. This symptom is called *hippocratic succussion*.

The **tension** of the gaseous effusion is variable, according as the perforation of the lung is obliterated or not. If it persists, the intrapleural pressure is practically the same as that of the atmosphere; if it is obliterated, the intrapleural pressure varies from  $-7$  during inspiration, to  $+3$  during expiration. In several patients I have found that these figures are variable.

Simple pneumothorax may recover in three or four weeks, but the nature of the perforation and the formation of pus aggravates the prognosis. I have, however, often seen the cure of pneumothorax and of hydropneumothorax in tubercular patients.

\* If the posterior region of the patient's chest is auscultated while the front is percussed, the percussion yields the *bruit d'airain*. This bruit is still better heard if the percussion is made by means of two coins.

In some cases pneumothorax and hydropneumothorax are localized to some portion of the thoracic cavity by previous adhesions; this condition is partial pneumothorax.

**Diagnosis.**—Large pleuritic effusions may give rise to amphoric breathing,\* but the other signs are so different that mistakes are not possible between gaseous and fluid collections. Large cavities in **phthisis** may simulate pneumothorax, but are nearly always localized to the apex of the lung; the râles become of a splashing nature, and the signs of amphorism are less marked.

In some cases **partial** pneumothorax and hydropneumothorax are very difficult to diagnose, and it is essential to make a methodical examination, dividing the thorax into three regions—**anterior**, **axillary**, and **posterior** (Jaccoud).

I have described a partial inferior pneumothorax with pleuro-peritoneal symptoms. It must not be confounded either with subphrenic abscess, which is an abdominal affection with pleural symptoms, or with certain cases of subdiaphragmatic peritonitis. Pneumothorax, or hydropneumothorax, being recognized, it is still necessary to diagnose the **cause**, for the ætiology of the perforation is the principal basis of the prognosis.

The **treatment** varies in pneumothorax, hydropneumothorax, or pyopneumothorax. In a tubercular patient with hydropneumothorax it is, perhaps, preferable not to withdraw the fluid, for the spread of tuberculosis may be hindered in the lung thus compressed. In a case of pyopneumothorax Potain obtained striking success with injections of sterilized air.

I have treated tubercular pyopneumothorax by multiple punctures; at each puncture only 2 ounces of fluid were withdrawn, and an intrapleural injection of solution of sublimate was given.

**Pneumothorax by Putrefaction.**—I have so far dealt with pneumothorax by perforation. Essential pneumothorax, independent of perforation and consecutive to exhalation of gas by the pleura, was formerly admitted. This view is no longer held, but some authors have stated that purulent fluid in the pleura may produce gas by decomposition, and give rise to pneumothorax. This latter form no doubt does exist, and is due to **putrefaction**, caused by anaerobic microbes. I have dealt with pneumothorax by putrefaction under Putrid Pleurisy. Pneumothorax by putrefaction is the opposite of pneumothorax by perforation; while the latter is due to the passage of air into the pleural cavity, the former is due to the production of gas in a cavity into which the air has not penetrated.

\* These amphoric sounds are due to a cavity, filled with gas, which plays the part of a resonating chamber. Metallic tinkling is only a râle which, in contact with this cavity, assumes a special timbre, and it is not essential in order that the tinkling may be produced to have a communication between the cavity and the bronchus in which the râle arises.



## CHAPTER VI

### DISEASES OF THE MEDIASTINUM

#### I. TUMOURS OF THE MEDIASTINUM.

**Anatomy.**—The organs of the mediastinum are so numerous, their lesions so frequent, and their disposition so important, by reason of the troubles which result from these lesions, that I will briefly describe the anatomy of this region.

The name "**mediastinum**" is given to the irregularly shaped cavity which is filled by numerous organs, and occupies the space between the sternum, the vertebral column, and the inner surface of the lungs. To understand the construction of the mediastinal cavity, it is enough to know the relations of the pleuræ in this situation. The pleuræ, after being in contact behind the sternum, open out, and in the interval which extends from the posterior part of the sternum to the root of the lung they circumscribe a space called the "**anterior mediastinum**." They continue their course from the root of the lung to the anterior surface of the vertebral column, and circumscribe a second space, known as the "**posterior mediastinum**."

The height of the **anterior mediastinum** equals the anterior vertical diameter of the thorax. Its shape may be compared to a triangular pyramid, two sides of which are lateral and one posterior. The antero-lateral sides are formed by the layers of the pleura which are attached to the sternum; they are in relation (the right especially so) with the lung. The posterior side, which is the smallest, is in relation with the œsophagus below and with the œsophagus and the thoracic cavity above. In this triangular space the following organs are contained :

The pericardium, extending vertically from the xiphoid cartilage to the middle of the first piece of the sternum, and horizontally for about 4 inches to the left of the middle line of the sternum and 1 inch to the right.

The heart, the apex of which corresponds to the sixth rib, is found about 4 inches from the median line of the sternum. The arch of the aorta, which corresponds to the middle and upper part of the sternum, contains the cardiac plexus in its concavity.

On the same plane as these vessels we find, on the right side, the innominate artery and the superior vena cava; on the left the common carotid and subclavian arteries, and more externally the recurrent and phrenic nerves.

Behind these organs is the root of the lung, formed by (1) the bronchi situated on the same plane as the inner part of the second intercostal spaces; (2) the pulmonary arteries; (3) the pulmonary veins.

The **posterior part of the mediastinum** is very different from the anterior one. Its length is nearly equal to that of the dorsal column, and its shape that of a four-sided pyramid, with its apex below. The lateral sides are formed by the pleuræ, which open out above to receive the subclavian arteries. The posterior side corresponds to the vertebral column, while the anterior is limited by the bifurcation of the trachea in its upper fourth, and by the pericardium in its lower three-fourths. In this irregular quadrangular space the following organs are met with :

The thoracic aorta and œsophagus, which are at first situated on the same transverse plane, but approach one another during their descent, so that the œsophagus is finally placed in front of the aorta, and helps to form the apex of the pyramid.

The vena azygos major occupies the right side of the vertebral column, behind the œsophagus; the vena azygos minor is placed on the left side of the dorsal column, behind the aorta; while the thoracic duct is situated between the two azygos veins.

We also find connective tissue, lymphatic glands, and twigs from the great sympathetic plexus and the pneumogastric nerves which surround the œsophagus.

The lymphatic glands have a special importance, from the numerous diseases that occur in them. As a whole, they receive the lymphatic vessels from the pleura, lungs, trachea, bronchi, heart, pericardium, and chest-walls. The groups of glands which deserve special attention have been described by Baréty: (1) Right and left peritracheo-bronchial groups; (2) right and left subbronchial groups; and (3) interbronchial groups.

**Pathological Anatomy.**—Among the numerous tumours which develop in the mediastinum, we shall consider: (1) Simple adenopathy; (2) tubercular adenopathy; (3) cancerous adenopathy; (4) degeneration of the thymus; (5) aneurysm of the aorta; (6) abscesses of the mediastinum; (7) cancer of the œsophagus; (8) glandular hypertrophy, with or without leucocythæmia.

1. **Simple Adenopathy.**—Acute diseases of the lungs and of the bronchi produce engorgement and inflammation of the tracheo-bronchial glands, especially in children. Pneumonia, capillary bronchitis, and the catarrhs associated with measles, whooping-cough, or influenza, lead to adenopathy. The glands sometimes become much enlarged; inflammatory congestion and œdema are present, and sometimes “hyperæmia and effusion of blood cause an increase in size, with red coloration of the whole gland tissue, which then resembles liver.” Acute adenitis sometimes goes on to suppuration.

2. **Tubercular Adenopathy.**—The tubercles develop along the vessels in the cavernous or in the follicular system of the gland, and present the same characters as in other tissues. The tubercle undergoes fatty change, and when the granulations are confluent, the parts situated between them may undergo caseous degeneration.

In children tuberculization of the bronchial glands is never primary, but **always** consecutive to tuberculosis of the lung; the lesion in the lung may be insignificant, while the glandular mischief is very extensive (Parrot).

3. **Malignant Adenopathy.**—Sarcoma and carcinoma of the bronchial glands are often consecutive to malignant disease of the lung; lymphadenoma, which is much more frequent than the preceding forms, is generally primary; it develops in young healthy subjects, and at times assumes the more malignant form of lymphosarcoma.

These malignant tumours grow large, invade the organs of the mediastinum, the heart, the lungs, and envelop the vessels and nerves. In other cases the infection is carried to a distance, probably by way of the lym-



phatics, veins, or serous membranes, and secondary nodules are found in the kidney, liver, etc.

4. **Degeneration of the Thymus.**—The most contradictory opinions have been enunciated on the subject of tumours of the thymus; their frequency has in turn been exaggerated and denied. It is certain that in children, and even in adults, we find sarcomata of different forms and slow evolution, which have arisen in the vestiges of the thymus.

5. **Glandular hypertrophy**, with or without **leucocythæmia**, will receive a separate description.

6. **Syphilis** may involve the mediastinal glands (see Syphilis of the Lung).

**Symptoms.**—Besides the signs and symptoms special to the pathology of each of the organs in the mediastinum (cancer of the œsophagus, aneurysm of the aorta, lymphadenoma, glandular tuberculosis, etc.), certain signs are common to all tumours, and serve to give a complete picture of the pathology of the mediastinal region. This is what I have named the **mediastinal syndrome**.

For example, the compression of a bronchus, of a venous trunk, or of a recurrent nerve, is always followed by the same effects, whether it be due to aneurysm of the aorta, glandular hypertrophy, lymphadenoma, or cancer. These common signs and uniform symptoms are nearly all the result of compression exercised by tumours upon the organs in the mediastinal region. They are as follows :

1. **Deformity.**—This deformity affects the sternal region. The first piece of the sternum may be raised, or the bulging may predominate at the sterno-clavicular joints; sometimes the bones are worn away, as in aneurysm of the aorta, and the expansile tumour takes the place of the bone. When the swelling is due to masses of glands (lymphadenoma, cancer), the glands in the supraclavicular hollow are often enlarged.

Other signs are also present: normal resonance gives place to dullness, which varies according to the size of the tumour, and may often be found behind in the interscapular region (Guéneau de Mussy). Bronchophony and bronchial breathing are proper to changes in the glands near the trachea and the bronchi, while a double centre of pulsations marks the existence of aortic aneurysm.

When the disease is of long duration, the retraction of the chest-wall, which has sometimes been observed, is probably due to the compression of a large bronchus, with impaired activity of the lung.

2. **Compression of the Bloodvessels.**—This result is very common in tumours of the mediastinum. “The arteries and the bronchial veins may be compressed by the suprabronchial and intertracheo-bronchial glands, the brachio-cephalic venous trunks, and the branches of the arch of the aorta by the retrosterno-clavicular glands” (Baréty).

The arterial vessels, being more resistant, suffer less from compression than the veins, and it is not rare to see an aneurysm of the aorta or a large mass of glands compressing the superior vena cava, the vena azygos major, and the innominate vein. When the superior vena cava is compressed, the passage of blood through this channel into the right auricle is difficult or impossible, and the result is blood-stasis in the areas which pour their blood into the tributaries of the superior vena cava—that is to say, in the head, the upper limbs, and the upper part of the thorax.

Following the blood-stasis, we observe dilatation of the subcutaneous venules; bluish networks show themselves on the thorax, shoulders, and arms, etc., while the jugular veins are dilated. This venous stasis is followed by the formation of a collateral circulation—that is to say, the blood which, as a rule, flows into the left auricle through the superior vena cava, now takes another course, and seeks to reach the same goal by the inferior vena cava. The blood passes through its altered channel by means of deep and superficial anastomoses, which join the superior to the inferior caval system, and which from actual necessity become many times larger than the normal.

These anastomoses are the large and small azygos, intercostal, internal mammary, deep epigastric, superficial epigastric, and circumflex iliac veins. By these altered channels the blood of the superior caval system attempts to pass into the inferior caval one in order to reach the right auricle. Hence in these abnormal cases the blood flows from above downwards in the cutaneous veins of the thorax and abdomen, and it is easy to prove the direction of the current by pressing back the blood in a dilated venous segment, and alternately removing the compression in the upper or in the lower end of the bloodless segment.

If the vena azygos major participates in the compression, the re-establishment of the circulation takes place only by the inferior vena cava. In the opposite case the azygos system which empties itself into the superior vena cava takes part in the re-establishment of the circulation. In short, the blood-stasis in the veins, the situation of these networks, and the direction of the blood-current give valuable information, which may point out the obstacle to the circulation in the mediastinum.

When the collateral circulation is efficient, the symptoms are slight. In the opposite case, œdema of the hands and face is seen, the lips are violet-coloured, the eyes are injected, the patient suffers from giddiness, nose-bleeding, and headache—in short, from the signs of cephalic congestion, due to venous stasis.

When the subclavian artery or the brachio-cephalic trunk is compressed, the radial pulse diminishes in size. On the affected side compression of the pulmonary artery or of its branches has several times been noted; ulceration of these vessels causes fulminant hæmoptysis.



**3. Compression of the Trachea and Bronchi.**—The left bronchus is more often compressed than the right; the vesicular murmur diminishes or disappears in the corresponding lung, but the thoracic resonance is preserved. The union of these two symptoms—absence of breath-sounds and preservation of resonance—eliminates the idea of an effusion into the pleura, and can only be explained by narrowing or by compression of the bronchus. When the calibre is much diminished, we find inspiratory sucking-in, which is most marked in the supraclavicular and epigastric hollows (Guéneau de Mussy).

Compression of the bronchi and of the trachea often produces rough and whistling inspiration, which has received the name of **stridor** (Cayol). This sound is heard whenever stenosis affects the larynx, the trachea, or the large bronchi.

**4. Changes in the Pneumogastric, Recurrent, Phrenic, and Sympathetic Nerves.**—The symptoms, consecutive to changes in these nerves, differ according as the nerve is irritated (symptoms of excitation) or destroyed (symptoms of paralysis). This is a very important distinction. Hoarse paroxysmal cough, resembling that of whooping-cough, has been noted in a great number of cases (Guéneau de Mussy) of bronchial adenopathy. “In children especially, when spasmodic cough shows itself at once or persists long after genuine whooping-cough, there is reason to suspect compression of the pneumogastric nerve by degenerated bronchial glands” (Verliac). **Dyspnœa**, with or without paroxysms, often results from compression of the vagus and the recurrent nerves. The attack may simulate asthma (Hérard), or may resemble angina pectoris. In one case the symptoms were due to irritation of the right vagus, which was congested and adherent to the swollen and degenerated glands (Baréty). I have seen an analogous case.

**Laryngeal** troubles betray themselves by the raucous nature of the voice (dysphonia), and by spasms of the glottis. The changes in the voice are explained by the paralysis of the vocal cord supplied by the affected recurrent nerve. The diagnosis is readily verified by the laryngoscope. Spasms of the glottis are due to excitation of the recurrent nerve. Excitation of one nerve alone is sufficient to provoke spasm of the glottis (Krishaber). The change in the phrenic nerves determines diaphragmatic neuralgia and attacks of dyspnœa (Bazin).

Inequality of the pupils is often seen, and arises, doubtless, from changes in the great sympathetic nerve.

**5. Dysphagia** is due to the compression of the œsophagus by mediastinal tumours, tumours of the aorta, degeneration of glands, etc., and to adherence of the œsophagus to neighbouring organs. Its perforation has been noted five times (Baréty).

**Diagnosis—Prognosis.**—The **diagnosis** is often difficult. Sometimes the **lesion betrays itself only by an isolated phenomenon** — viz., permanent contraction of one pupil, paroxysmal dyspnoea, analogous to attacks of asthma, aphonia simulating a malady of the larynx, spasmodic and paroxysmal cough resembling whooping-cough, or agonizing retro-sternal pain, which is nothing less than angina pectoris. Even when attention is directed to the seat of the disease, the difficulty is not entirely overcome, for many mediastinal tumours have symptoms in common, and it is then necessary to diagnose the nature of the tumour.

If, however, they have characters in common, they also have some special characters which aid in the diagnosis. Thus, in aneurysm of the aorta we often find a movement of expansion, a double centre of pulsations, and a double or single blowing murmur. Glandular affections of the mediastinum (lymphadenoma, leukaemia) are generally accompanied by glandular hypertrophy in other regions, as the neck, the axilla, or the groin. In tubercular adenopathy the condition of the lungs must be carefully inquired into. Cancerous degenerations sometimes involve the supra-clavicular glands.

The **prognosis** is generally grave. Tumours, by their more or less rapid progress, compromise the respiration and the circulation, and the patient dies either slowly from asphyxia, or suddenly from syncope, or from attacks of suffocation. Rapid death generally depends upon changes in the vagus and recurrent nerves.

Certain lesions of the mediastinum may be treated **surgically** (Ziem-bicki). Dr. Potarca (of Bucharest), in a work entitled "Posterior Intra-mediastinal Surgery," has reported numerous operations performed for purulent collections in the posterior mediastinum, phlegmon, mediastinitis, suppuration in the bones or the glands, and abscesses from foreign bodies.

## II. SYPHILITIC MEDIASTINITIS—MEDIASTINAL SYPHILOMA.

**Description.**—On April 11, 1910, a man, sixty-three years of age, a slater by trade, came under my care in St. Christophe ward. Until the end of the previous year, his health had been good, but from that date a series of symptoms succeeded one another, the description of which is given herewith.

The patient had, at first, experienced a pain, or rather a heaviness in his head, which soon became very sharp. He stated that he felt as if he had received a knockdown blow. He experienced this sensation of heaviness especially during his work when his head was lowered, seeing that a slater is obliged to bend forward. The same sensation was again very intense when in bed, on account of the horizontal position, and in



order to ease his sufferings, the poor fellow was obliged to be seated, or to remain standing, conditions which were unfavourable for sleeping. At the same period, a fairly marked symptom appeared, viz. the flowing of tears on his cheeks and down his nostrils. This lachrymation occurred especially when the patient bent forward during his work, or when he was in bed, his head resting on his pillow. The tears came drop by drop, and the bed linen was quite wet. At the same time, a rapidly noticeable diminution of the sense of hearing supervened. The patient could no longer carry on a conversation. He became deaf, without being able to account for the fact. To these symptoms was added so intense a dysphagia that he was able to take only soups and milk. The dysphagia was not at all painful, nor was it accompanied by regurgitations, or by vomiting. It consisted merely in mechanical discomfort in swallowing solids. His respiration also left much to be desired. The slightest effort became impossible and he was soon out of breath, especially during work. He perceived that his voice was growing weaker and weaker and that it was becoming hoarse.

An enormous swelling of the neck next appeared, in addition to the previous symptoms. He could no longer button his shirt collar, because the size of his neck had increased by four centimetres.

Losing strength, breathing badly, and feeling seriously ill, the man ceased work in February, 1910. At the same period, in a few days, œdema invaded the lower part of the face, the neck, and the upper limbs. Such was the condition of the patient when he entered the hospital on April 11.

He was examined immediately on admission. The most noticeable feature was the look of his pear-shaped head. The cheeks and the sides of the face were greatly enlarged by the indurated œdema, which was continuous with a swelling in the parotid regions and the neck. The upper part of the thorax was, likewise, affected by this œdematous swelling, which had, also, invaded the upper limbs. The arms and the forearms were enormous. They had assumed a cylindrical form which had completely invaded the muscles. The hands were thickened. At certain places, the œdematous tissues had assumed a sclerotic consistency which resembled cardboard. It was impossible to pinch the skin, or to pucker it. In consequence of this indurated œdema, the movements of the hands and arms were very limited. The patient could hardly move his fingers, or bend his wrist.

On the other hand, the œdema had scarcely affected the upper part of the face and had completely respected the lower limbs.

A very pronounced collateral venous circulation was observed in the neck, on the chest, in the back, and on the arms. In the neck, the external

jugulars formed twisted cords which were greatly distended. On the anterior surface of the thorax, there was an area of very distended veins. On each side of the abdomen, there was a voluminous arch, which started from the outer border of the right rectus muscle and, after having described a long curve, lost itself in the neighbourhood of the sternum. On this venous arch, placed between the epigastric veins on the one hand, the intercostals and the internal mammaries on the other, it was easy, by digital pressure, to demonstrate the flow of the blood, which, instead of proceeding from below upwards, flowed from above downwards, going from the superior vena cava towards the inferior vena cava.

The patient looked somewhat flurried. The weight of his head caused it to be extremely painful. It was not a case of headache. His head might have weighed 30 lbs. His eyes were full of tears, but there was no chemosis; respiration was embarrassed; the dysphagia allowed him to swallow only liquids. He had become so deaf that, even when people shouted into his ears, he could not understand them.

On examination of the organs, nothing worthy of notice was found in the heart, or in the aorta. The beat of the radial pulse was regular and synchronous. There was no pleural effusion, no pulmonary lesion, nor any abnormal tracheo-bronchial bruit, neither wheezing nor souffle, but, on percussion, the right side of the thorax was less resonant than the left one, and, on auscultation, the vesicular murmur was sensibly enfeebled. There was no dullness either in the sternal, or in the interscapular region. No glands were found anywhere.

His voice was raucous and bitonal. An examination, made with the laryngoscope by Bonnier, showed that the right vocal cord was paralysed. This paralysis was to be foreseen, for, on slightly pressing the larynx between two fingers when the patient spoke, the vibrations of the glottis, transmitted to the fingers, were considerably less on the paralysed side. Armed with these details, we could affirm that our patient was suffering from a tumour of the mediastinum. The topographical diagnosis could go even further and localize the lesion in the vicinity of the superior vena cava. **The regional distribution of the œdema and the paralysis of the right vocal cord** were signs of the utmost importance and justified this diagnosis. As a matter of fact, it was this obstruction in the region of the superior vena cava which explained the œdema that was localized in the face, the neck, the upper part of the thorax, and the upper limbs. The obstruction explained the collateral circulation, the venous distension, the reversal of the flow of the blood current, and the encephalic venous stasis which revealed itself by the terrible weight of the head. The paralysis of the right vocal cord indicated that the right recurrent or, perhaps, the right pneumogastric nerve was affected.



The nature of the mediastinal lesion remained to be decided. Had we to deal with an aortic aneurysm or with a tubercular, cancerous or syphilitic lesion of the glands?

The hypothesis of an aneurysm of the aorta would immediately be dismissed, for there was no sign to bear out such a diagnosis. Could there be a tubercular, or a cancerous mediastinal lesion? This was not impossible, but it was highly improbable, for the most minute examination failed to point in that direction. Radioscopic examination and radiographs, taken on several occasions, had given only negative results. There was a shadow in the suspected mediastinal region, but no conclusion could be drawn from it.

It would have been highly desirable to diagnose a syphilitic lesion of the mediastinum, for the treatment would have been greatly simplified and the chances of success would, doubtlessly, not have been illusory. We did what we could to find arguments in favour of this diagnosis, but failed. The patient, though questioned in detail, denied that he had ever had syphilis. Furthermore, he bore no trace and had no stigmata of either old or of new syphilis. We wondered whether this was not, perhaps, a case of concealed syphilis.

The patient was placed under observation and a milk diet prescribed, and, for want of a better remedy, he was given injections of cacodylate of soda. The symptoms, however, grew worse. The dyspnoea increased and the veins of the neck became still further enlarged. The deafness was all but complete. The neck was enormous; the œdema of the arms increased, and the weight of the head was intolerable. The nights were most trying. He grew weaker and weaker and, to sum up, the situation became alarming.

M. Guinard, one of the surgeons of the Hôtel-Dieu, having come into the ward, M. Le Play, one of my clinical assistants, begged him to examine the patient. M. Guinard was of opinion that it was a case of a mediastinal lesion in the neighbourhood of the superior vena cava. He suggested the possibility of a thymic tumour, and expressed the opinion that one might explore the case, or that Freund's operation might be performed. This operation consists in making a section of a certain portion of the cartilages which unite the ribs to the sternum, the object of the operation being to give greater play to the thorax and more room to the organs of the mediastinum. M. Le Play, therefore, sent the patient to the surgical ward, but, next morning, having thought the matter over, he had certain scruples and asked himself whether this was really a surgical case and, without delay, he had the patient brought back to our ward.

The idea of syphilis arose again, all the more so as we had seen in the wards some really astounding cases of concealed syphilis which had been

successfully treated. The first case was that of a man who had an invading ulcer, apparently of a lupus nature, in his throat.

Though the man affirmed, in all good faith, that he had never had syphilis and though no stigmata were found, I, nevertheless, gave him daily mercurial injections in increasing doses of 1, 2, 3, and 4 centigrammes of biniodide of mercury. In spite of this intensive treatment, the ulceration in the throat assumed greater and greater dimensions. The uvula was partially destroyed; the pillars were attacked, the dysphagia became terribly painful, and the patient grew visibly thinner.

Wishing to increase the patient's strength, Marion performed gastrostomy, and, for eight months, the patient was fed by means of a stomach tube. He grew fat, but the ulceration in the throat had progressed and had invaded the pharynx. Convinced that this was a case of concealed syphilis (in those days we had no means of searching for the treponema, nor were we acquainted with Wassermann's reaction), I recommenced the injections of mercury, the daily dose of which I increased to 5 centigrammes. The result was most remarkable. The ulceration which, for more than a year, had been refractory to treatment cicatrized very rapidly when the curative dose was reached. The sound was removed, and a cure was effected. This was, then, a case of concealed syphilis.

I would mention another case of a woman who, for the past five years, had suffered from violent headaches, frequent attacks of epilepsy, facial and ocular paralysis, visual troubles accompanied by papillary oedema, consecutive symptoms of basilar meningitis. In this case also, the patient affirmed, in perfect good faith, that she had never had syphilis. She had no stigmata. Furthermore, a most important point, she had given birth, at term, at the commencement of the disease, to a very well formed female child which, at that date, was in perfect health and without any hereditary taint. Such facts were sufficient to remove all hypothesis of syphilis, and yet the study of the symptoms and their evolution led me to diagnose the case as one of concealed syphilis and of basilar meningeal syphiloma. The woman was put under increasing doses of 1, 2, 3, 4 centigrammes of biniodide of mercury. No result being obtained, the daily dose was increased to 5 centigrammes and, at a certain moment, the headaches and the attacks of epilepsy ceased suddenly and did not return. The other symptoms, also, disappeared and the patient was cured. This, also, was a case of concealed syphilis.

Why not, therefore, try the effects of mercurial treatment on our patient? Might his mediastinitis not be the result of untraceable and concealed syphilis? Might he not be suffering from a mediastinal syphiloma?

Mercurial treatment was inaugurated on May 27. Every day, up to



June 14, he had injections of increasing doses of biniodide of mercury of 1,  $1\frac{1}{2}$ , 2,  $2\frac{1}{2}$  centigrammes.

The result was not long in showing itself. After the sixth or the seventh injection, the patient exhibited a notable improvement. The weight of the head had diminished, respiration was easier, the fingers were less œdematous and more mobile. On June 14, the improvement was incredible, and progressed visibly from day to day. The patient received us every morning with a smile. The terrible weight of the head had vanished. He slept well, and heard better. Deglutition was easier, the œdema diminished, and the hands and fingers were more agile.

The mercurial treatment, which had been suspended on June 14, was resumed six days later and, then, the daily dose of biniodide of mercury was increased to 3 and 4 centigrammes. Dating from June 30, the patient's condition was completely modified. The head had lost its pear-shaped form, the eyes were no longer weeping; the heaviness had gone completely. The œdema of the neck and of the arms had diminished considerably. The tissues had become more supple. The outlines of the muscles once more assumed their normal appearance. The patient regained freedom of movement. He gesticulated, and his fingers had again acquired their accustomed agility. To show how easy deglutition had become, he ate and swallowed large pieces of bread, and shook his head in all directions. His deafness had vanished. His voice had acquired its normal timbre, and, a most essential point, laryngeal examination showed that the right vocal cord was **no longer paralysed**.

The patient underwent the mercurial treatment without the slightest inconvenience, so much so that the injections were continued up to July 11. Though the œdema of the neck and of the upper limbs had, in part, disappeared, the tissues had not yet acquired their suppleness. The tissues of the external region of the forearms were still like cardboard; the veins of the neck were still very distended tortuous cords; the venous areas of the trunk and of the arms were the more apparent, as they were no longer masked by the œdema which had hidden them.

On auscultation, it was evident that the vesicular movement was restored in its integrity on the right side of the chest. The patient slept well and his appetite was excellent. During a portion of the day, he walked on the terraces of the Hôtel-Dieu and ascended the staircases without the least oppression. He had regained his strength. He felt cured, and asked leave to be discharged from hospital, so that he might resume his calling. Naturally, he was delighted, seeing that, only a few months previously, he believed himself lost. Some weeks afterwards, he revisited the hospital. He was in perfect health, had resumed his trade, and felt no fatigue whatever. It was as though he had never been ill. Nevertheless,

he promised to return for another series of injections. It will not be the last one, for the treatment will have to be resumed every three months for at least a year.

It matters little under what form this syphilitic mediastinitis developed, whether it were a gumma, a callous mediastinitis, or a sclero-gummatous diffuse syphiloma. The essential point is that the symptoms disappeared rapidly under the influence of mercurial treatment.

Here we have, then, another case of concealed syphilis, and such cases are not rare. Professor Fournier has published an important lecture on this subject. It will readily be admitted that hereditary syphilis might be concealed, but it is less easy to believe that a case of acquired syphilis could be forgotten. Nevertheless, the gateway of the infection may remain unnoticed and the patient may become infected without knowing it. Without enumerating the various cases of this kind which may present themselves, I may quote the case of one of my patients who came into hospital with tonsillar angina, apparently so trivial that there was even no sub-maxillary adenopathy. It would have been impossible to diagnose chancre of the tonsil, if examination with the ultra-microscope had not revealed the presence of numerous characteristic treponemas. A short time afterwards, the syphilitic roseola appeared. I am convinced that, in a certain number of cases, the diagnosis is missed for want of information, because the patient is ignorant of his syphilis and the physician cannot discover it. Thus, when I am face to face with organic lesions the cause of which escapes me when I cannot account for the disease, I think of the possibility of concealed syphilis and institute mercurial treatment and, when successful, I delight in proclaiming, once more, the truth of the old adage: **Naturam morborum curationes ostendunt.**

I cannot say what Ehrlich's valuable discovery, 606, has in store for us, but we have an admirable remedy ready at hand—mercurial treatment.

### III. CANCER OF THE THORACIC DUCT.

The thoracic duct, which collects the lymph from the lower limbs and the thoracic and abdominal viscera, extends from the receptaculum chyli to the left subclavian vein. It belongs, therefore, to the posterior mediastinum, where it passes up between the two azygos veins behind the oesophagus, until it crosses the posterior surface of the arch of the aorta and enters the cervical region. The thoracic duct carries matter absorbed by the digestive tract, but in pathological conditions it may transport microbes and cellular emboli to the right heart. Secondary cancer of the duct occurs when cancer cells are grafted on to its walls.

Nattan-Larrier has described two cases of secondary cancer of the duct.

The organs which usually give rise to secondary cancer of the duct



are the stomach, the uterus, and the testicle, because their lymphatics pass more directly to the duct; then come the rectum, the pancreas, the kidney, and the ovary. The thoracic duct is invaded by vascular propagation. The degeneration affects successively the lymphatics from the cancerous organ, the corresponding glands, their efferent lymphatics, and finally the thoracic duct. Invasion by effraction is quite exceptional.

In one-half of the cases the entire duct is involved, in one-quarter the growth stops at some distance from the termination of the duct, while in more rare cases the lesion is limited to the origin of the vessel. When the entire duct is invaded, it resembles a vessel injected with paraffin. "The receptaculum is as large as the thumb, and looks like a flabby, nodular, whitish cord, while a granular mass exudes on puncture. Below the receptaculum we find a network of cancerous lymphatics over the sides of the aorta. Higher up we find the duct, properly speaking; it forms a trunk 3 to 4 millimetres in diameter, winding over the posterior surface of the aorta. At some points it divides into three or four branches, which remain separate for 3 or 4 inches, and then reunite to form a trunk as large as the primary vessel. After passing round the subclavian artery the cancerous duct reaches the vein, which it perforates at the junction of this vessel with the internal jugular vein." Sections show secondary cancer of the duct. The epitheliomatous cells are fixed in the connective tissue, and form buds, which obliterate the lumen. At the termination of the duct we often find a cancerous vegetation which floats in the subclavian vein.

The subjoined case is very typical :

Woman who had suffered from dyspepsia for many years, admitted in July, 1900. Repeated melæna and hæmatemesis, complete anorexia, and marked wasting. She complained of acute epigastric pain, and palpation revealed a large tumour at the level of the greater curvature of the stomach.

She had cancer of the stomach, confirmed by a large gland in the left supraclavicular fossa. The left subclavian vein soon became thrombosed. Fever appeared, and she died from streptococcal septicæmia. Post mortem, cancer of stomach and secondary growth involving the entire thoracic duct, which was completely obliterated, and showed all the features above described.

Cancer of the thoracic duct shows itself by symptoms and signs which are at times difficult to distinguish from those due to the primary growth. Wasting and malnutrition are rapid. Yet, strange to say, obliteration of the duct is not, as a rule, accompanied by lymphostasis. In three cases only did milky effusion in the peritoneum coincide with cancer of the duct. In two cases the chyloform ascites was due to cancer of the peritoneum, and in the third case the chylous effusion was due to the spread of the growth to the subperitoneal lacteals. We must therefore admit that the lymph-flow is usually re-established by the collateral channels. Phlebitis of the left arm is fairly common, and is due to thrombosis, which starts from the

anastomosis of the duct with the vein. The phlebitis is seen in 25 per cent. of cases. The existence of glands in the left supraclavicular fossa has been noted in half of the cases.

The discovery of a prelumbar tumour, accompanied by double varicocele, would point to invasion of the thoracic duct, since the prelumbar glands are affected in all cases of epithelioma of this tube. Nevertheless, the prelumbar glands may be cancerous, while the duct is not invaded. No one of these signs has absolute value, but their appearance in cancer of the stomach or of the uterus would lead us to think of secondary growth in the thoracic duct.



## PART II

### DISEASES OF THE CIRCULATORY SYSTEM

#### CHAPTER I

#### DISEASES OF THE PERICARDIUM

##### I. ACUTE PERICARDITIS.

PERICARDITIS is inflammation of the pericardium.

Senac (1783) was the first to separate pericarditis from other diseases of the heart. In 1806 Corvisart applied to it the signs given by percussion ; in 1824 Collin, assistant to Laënnec, discovered the pericardial rub (**bruit de cuir neuf**), and some years later Bouillaud created cardiac pathology.

**Ætiology.**—The so-called **primary** (*a frigore*) pericarditis does not exist. The **secondary** form is sometimes associated with a neighbouring lesion (pleurisy, aneurysm of the aorta). It may follow injury, but usually occurs in infectious diseases and constitutional maladies.

Pericarditis associated with **pneumonia** may be parapneumonic or metapneumonic. It may be dry, sero-fibrinous, hæmorrhagic, or purulent. The fluid does not contain much fibrin, and the pus is homogeneous. Pneumococcal pericarditis may be independent of any pulmonary lesion.

The **eruptive fevers**, and especially **scarlatina**, may be accompanied by pericarditis. The fluid may be sero-fibrinous, hæmorrhagic, or purulent, and almost always contains the streptococcus.

Pericarditis in **erysipelas** is also associated with the streptococcus (Denucé).

Pericarditis consecutive to influenzal broncho-pulmonary lesions is associated with several microbes, among which the streptococcus is the chief.

Pericarditis in **Bright's disease** is said in some cases to be of toxic origin.

**Tubercular** pericarditis is sero-fibrinous, hæmorrhagic, or purulent, and due to Koch's bacillus, with which other organisms may be associated.

Purulent pericarditis consecutive to **pyæmia** is due to staphylococci or streptococci, but it is sometimes impossible to find the point of entry.

The most frequent cause is **acute rheumatism**, which is probably a microbic disease. The pericarditis appears during the first and second weeks of articular rheumatism, but may appear at the same time as the articular lesions, or even apart from them. In some cases it is associated with endocarditis or with pleurisy. It attacks patients at any age, but by preference young children and infants.

**Pathological Anatomy.**—Acute pericarditis has been divided, like pleurisy, into two varieties, according as it is dry or accompanied by effusion. In acute pleurisy, however, effusion is the rule, while in pericarditis it is the exception. In ten patients suffering from rheumatic pericarditis, effusion is perchance found but twice. From an anatomical point of view, acute dry pericarditis does not exist, for a certain quantity of exudate is always found post mortem. Clinically, however, the effusion is not considered, unless it is sufficient to cause special signs, and from this point of view pericarditis with effusion is somewhat rare.

Pericarditis may be partial or general. It is usually situated near the aorta and at the base of the heart. The visceral layer, or epicardium, is always the more affected. At first the congested vessels form a fine network on the surface of the serosa, which loses its polish and becomes covered with a fibrinous exudate, the papillary aspect of which (*cor hirsutum*) has caused it to be compared to a cat's tongue, or to slices of bread and butter which have been pressed together and then quickly separated. The papillæ are formed of fibrin, epithelial cells, and pus corpuscles. Their special form is due to the incessant movements which are given to the fibrin by the heart. The outer layer of the serosa takes no part. The connective tissue of the serosa shows embryonic infiltration, and the lymphatic vessels are crammed with fibrin and white corpuscles. The quantity of fluid varies from a few drachms to a pint or more; it may be sero-fibrinous (rheumatism, pneumonia), hæmorrhagic (tuberculosis, Bright's disease, scurvy, cancer, and cachexia), or purulent (scarlatina, pneumonia, typhoid fever, and puerperal conditions). The heart muscle often shows superficial myocarditis. Tubercular pericarditis deserves special mention. It supervenes in the course of acute or of chronic tuberculosis, or occurs as a primary local tuberculosis. Effusion may or may not be present, and in the former case the fluid is often hæmorrhagic. In recent cases the tubercular tissue contains bacilli but they may be absent in old cases (Cornil and Babès). The mediastinal glands are indurated and enlarged, while the cellular tissue around them is adherent to the pleura and the lungs. Tubercular pericarditis sometimes ends in adherent pericardium.

**Description.**—The invasion is very variable, and although this diversity of onset may not be solely imputable to its causes, pericarditis is usually insidious and latent (Stokes), and far more rarely acute and painful.



The patient sometimes complains of more or less severe oppression, with palpitation and pain in the precordial or in the epigastric region, or between the shoulders. As a rule, especially in rheumatic pericarditis, these symptoms, and especially the pain, are absent or slight. In some exceptional cases, however, the symptoms of acute pericarditis are very marked: the face is pale; the patient is anxious; the pain may be terrible, accompanied by chill and lipothymia, and analogous to the pain of angina pectoris—a fact which would prove that the cardiac plexus and the phrenic nerve are affected by the inflammation.

Auscultation during the onset of the malady reveals a **rub**. This rub, which is at first systolic, soon becomes systolic and diastolic. It is a sound that comes and goes—*i.e.*, a “**bruit de frou-frou**.” As the friction sounds are not absolutely synchronous with the heart sounds, it is preferable to say that the one is mesosystolic and the other mesodiastolic. The want of synchronism depends on the fact that for both layers of the pericardium to produce a friction sound it is necessary that “the displacement of the surface of the heart should have reached a certain degree, that the muscular contraction should have just commenced, and that, in consequence of the form and volume of the heart, the surfaces in contact be, so to say, **relaxed**, after having been dragged apart more or less suddenly” (Potain).

The **pericardial rub** has special characters. It is more rasping than the cardiac murmur, and is not propagated in the direction of the blood-stream. It has its maximum intensity about the third intercostal space, where the anterior surface of the heart is more directly in relation with the chest, and increases in intensity when the patient bends forward or when the stethoscope is firmly applied to the chest-wall. Lastly, it is not absolutely isochronous with the normal sounds of the heart. In some cases the rub is intense and general.

It often happens that the systolic rub commences a little **before** the systole; it is presystolic, and causes a triple sound, called the “**bruit de galop**” (Bouillaud). This bruit de galop has a special rhythm, and its three periods are divisible in the following manner: The first two sounds, which are short and hurried, are formed, one by presystolic friction, and the other by the normal sound of the heart, with or without friction; while the third sound corresponds to the normal second sound of the heart, or to this sound covered by a friction sound. This bruit de galop, composed of two short sounds and one long one, must not be confounded with the sounds, called “bruit de caille” and “bruit de rappel,” the rhythm of which is a sign of mitral stenosis, and is inverse to the bruit de galop, being made up of a long and two short sounds.

The single or double rub, with or without bruit de galop, is therefore a sign of dry pericarditis; but when effusion is present, the fluid separates

the layers of the pericardium, and the rub disappears. In some cases, however, the rub may still be perceptible towards the base of the heart, in spite of a large effusion. In proportion as the effusion forms, the fluid collects in the dependent regions of the pericardium. A small effusion passes unnoticed, but large ones cause the following signs and symptoms :

On inspection and palpation, we find that the cardiac waves and the impulse of the heart against the chest-wall become gradually effaced. On auscultation, the heart sounds become faint and tend to disappear. Bulging of the precordial region, so characteristic in a child suffering from pericardial effusion, is less marked in the adult, because the ribs are more resistant, and the pericardium, distended by the effusion, finds room at the expense of the diaphragm, which it pushes down, and at the expense of the posterior mediastinum, which it pushes back.

The most valuable sign of pericardial effusion is **dullness**. This dullness varies according to the quantity of fluid effused. Its shape and limits furnish valuable information. Pericardial dullness is triangular or conical in shape, with its apex reaching as high as the third rib, where the pericardium is reflected on to the great vessels ; its base blends with the diaphragm. In large effusions, which amount to about a pint, the vertical line of dullness, which extends from the apex of the cone to its base, measures 6 or 7 inches, and the line of horizontal dullness, which is close to the base of the cone, has nearly the same length. Percussion, therefore, rather than the other signs, shows the daily progress of the effusion.

When the effusion is abundant, it gives rise to more or less marked symptoms, which include dysphagia, pallor and puffiness of the face, dyspnoea, which is sometimes acute and accompanied by fainting and angina, cyanosis and oedema of the peripheral parts, small intermittent pulse, and pulsus paradoxus. Some of these symptoms—namely, the smallness and irregularity of the pulse—may be due to compression of the auricles, which offer less resistance to the effusion than the ventricles. The pulsus paradoxus is found in cases of effusion and of pericardial adhesions, and consists of three or four pulsations, followed by suppression of the radial pulse for a like period, with no interruption of the heart-beats. The disappearance of the pulsations coincides with the end of inspiration. Dyspnoea, angina, attacks of suffocation, and threatening asphyxia, may be due to the pericardial effusion when it is associated with endocarditis, pulmonary congestion, and pleuritic effusion that often accompany pericarditis, especially in the case of acute rheumatism. Several of these symptoms may also be present in the paralytic form of pericarditis, which is due to degeneration of the heart muscle. In such cases the patient may succumb from asystole.



The **course** of pericarditis is irregular ; its **duration** is uncertain, and the fever cycle indefinite. The absorption of the fluid is followed by the gradual disappearance of symptoms, and the rub (*frottement de retour*) again appears. When the pericarditis has been slight, without effusion, as in rheumatic cases, the serous membrane returns to its normal condition, and recovery takes place in one or two weeks.

**Diagnosis—Prognosis.**—Apart from the exceptional cases in which the symptoms are acute, pericarditis is a disease which **must be carefully looked for**, because it develops without warning. Repeated auscultation is necessary in patients who have one of the diseases enumerated under the ætiology of pericarditis, especially in those who are suffering from articular rheumatism, and it will then be possible to recognize the rub at its onset.

The **diagnosis** must be made in each form. The rub can be distinguished from cardiac murmurs by the signs indicated above. The diagnosis from the pleuritic rub is very simple, because the patient can at will stop breathing, and at the same time suppress the rub of pleurisy.

I will briefly recapitulate the signs by which pericardial effusion may be diagnosed. The shape and situation of the dullness are the most valuable points ; then come the disappearance of the cardiac impulse, the faintness and disappearance of the heart sounds, and the *pulsus paradoxus*. Let us not forget that the pericardial rub may persist, in spite of a large effusion. Pericardial effusion and encysted effusion in the left pleura present some analogy, but the situation and the conical shape of the dullness in the precordial region, the weakening of the cardiac impulse, and the remoteness of the heart sounds, are in favour of pericarditis. Hypertrophy of the heart and pericardial effusion have two signs in common—*i.e.*, the extent of the dullness and the weakening of the heart sounds. In hypertrophy the dullness coincides with the apex of the heart, while it descends lower than the apex in pericardial effusion (Gubler).

In its common forms acute rheumatic pericarditis is not a serious malady. The gravity of the **prognosis** depends on the abundance of the effusion, and on certain complications, such as endocarditis, pleurisy, congestion of the lungs, inflammation of the myocardium, and ventricular thrombosis, which causes sudden death. The prognosis also depends on the causes which have given rise to pericarditis. The rheumatic form, for example, is not nearly so grave as the tubercular one.

**Treatment.**—If the inflammation is intense, and especially if the pain is severe, blood-letting should be performed. We may prescribe leeches or cupping to the precordial region, and employ blisters, or apply ice-bags, which are left *in situ*. When the heart muscle is weak, digitalis or caffeine is administered, and the pain is relieved by subcutaneous injections of

morphia. If the effusion threatens to bring on asphyxia or syncope, the fluid should be withdrawn without delay.

The history of paracentesis of the pericardium and the operative technique have been minutely described by Trousseau; but this operation has been modified, and I may say much simplified, since I have employed the aspirator. I omit, therefore, the old procedure, and mention the conclusions given in my memoir on paracentesis of the pericardium ("Traité de l'Aspiration").

Experiments performed on the cadaver give the following conclusions:

1. In an adult the pericardium may contain an amount of fluid, which in exceptional cases may exceed 2 pints.

2. Whatever be the degree of fullness, the pericardium reaches its greatest transverse diameter at the level of the fourth, or sometimes of the fifth, intercostal space.

3. At this level the pericardium is not covered by the left lung. The lung, on the contrary, forms a notch, simulating a crescent, which extends from the fourth to the sixth rib, and persists even when the lung is insufflated. This notch coincides with the maximum point of the transverse diameter of the distended pericardium, and consequently leaves a space free for the aspirating needle.

4. The pericardium, distended by fluid, extends beyond the left border of the sternum for as much as 4 or 5 inches.

5. I therefore recommend paracentesis in the fifth left intercostal space, about 2 inches from the left border of the sternum.

For this purpose a No. 2 needle is employed. The aspirator is emptied of air, and the puncture is made at the proper spot. When the needle has gone  $\frac{1}{4}$  inch into the tissue—*i.e.*, as soon as the needle is no longer in relation with the external air—the corresponding tap of the aspirator is opened, and a vacuum results in the needle, which **becomes an aspirating one**. With the vacuum, so to say, in hand, we then proceed to search for the effusion. The needle is pushed in slowly until the effusion flows through the glass index.

For the dangerous operation proposed by Aran, Jobert, and Trousseau, I have substituted a simple needle-prick, which is absolutely harmless, and demands neither special skill nor exceptional surgical knowledge.

Paracentesis does not give the good results of thoracentesis. In some cases, especially when the fluid is purulent, pericardotomy is indicated.



## II. CHRONIC PERICARDITIS—ADHERENT PERICARDIUM.

**Pathological Anatomy.**—Chronic pericarditis may be primary or consecutive to acute pericarditis. The *ætiology* is the same in both cases. The false membranes and the adhesions which are the chief lesions in chronic pericarditis present different aspects. The false membranes arise in the serosa, as vascular buds, which unite to form adhesions between the two layers of the serous membrane. The adhesions may be partial or general. When partial, they fix the apex of the heart, surround its base like a ring, or form septa and cavities, filled with fluid and degenerated cells. When general, they obliterate the cavity and lead to pericarditis obliterans (Stokes) or ankylosis of the heart (Bouillaud). If the inflammatory process also affects the external layer of the pericardium, this in turn forms adhesions with the neighbouring organs—viz., pleura, lung, diaphragm, and chest-wall. The false membranes may be an inch in thickness; they are hæmorrhagic, infiltrated with tubercles, or encrusted with calcareous salts. The ossiform patches in the pericardium are no more osseous tissue than the cartilaginiform patches are composed of cartilaginous tissue, for the latter do not possess chondroblasts, and are composed of lamellar connective tissue and elastic fibres (Cornil and Ranvier).

When chronic pericarditis is accompanied by effusion, the fluid, which is variable in quantity, may be purulent or hæmorrhagic, and contains shreds of membranes. The muscular fibre of the heart, which has become flabby and yellowish, is more or less affected by fatty degeneration, and we find, as the case may be, enlargement of the cavities, hypertrophy or atrophy of the heart, and insufficiency of the tricuspid and mitral valves.

**Description.**—When chronic pericarditis ends in adhesions, it readily passes unnoticed. It is only evident if friction sounds exist, if the effused fluid is abundant, or, lastly, if the adhesions are very extensive.

At the points where the false membranes are not adherent a rasping rub is audible. If the effusion is sufficiently abundant, it shows itself by the signs described under acute pericarditis.

Adherent pericardium, if uncomplicated, may give no symptoms; but if it is accompanied by degeneration of the myocardium and dilatation of the heart, it shows itself by the subjoined signs and symptoms.

In the first place the adhesions cause trouble in the cardiac circulation. In the normal condition the intrapericardial vacuum favours the filling of the cavities of the heart during diastole; if, however, adhesions form, the expulsion of blood is hindered during the systole, and the cavities do not fill during diastole, because the vacuum is suppressed. Adherent pericardium causes the following symptoms: The patient is a prey to more or less acute and constant dyspnoea, which increases with the least effort. He experi-

ences precordial pain, which may disappear during rest, but at times returns with the least exertion or fatigue. On pressure we find tenderness of the phrenic nerve in the neck and in the region of the diaphragm.

On examination of the thorax, bulging of the precordial region is often seen. Percussion may show considerable increase in the size of the heart. On palpation the systolic shock is much less marked than in the normal condition.

General adhesions produce signs which are most marked when the adhesions involve the chest-wall: these signs are—retraction of the intercostal spaces during systole (Williams), systolic retraction of the epigastric angle, and reduplication of the heart sounds.

The muscle often degenerates, becoming less resistant, and, with the assistance of the false membranes, dilatation of the ventricles and consecutive insufficiency of the valves result.\* Congestion and œdema, asphyxia, asystole, and sudden death may follow.

Surgery has tried to remedy these results (Brauer). The operation is to remove, in front, the resistant bony parts, so as to give more play to the cardiac movements. On each side, a resection of the ribs is made, of from 7 to 8 centimetres, the cartilage included. In a case which terminated successfully, reported by M. Delbet, he resected  $7\frac{1}{2}$  centimetres of the third rib,  $8\frac{1}{2}$  centimetres of the fourth, and 9 centimetres of the fifth rib.

### III. HYDROPERICARDIUM—HYDROPNEUMOPERICARDIUM.

**Hydropericardium** is dropsy of the pericardium. It is not the result of an inflammatory process like pericarditis, but occurs in the course of other diseases. Hydropericardium is due either to mechanical causes which embarrass the circulation in the cardiac vessels, or to dyscrasiæ and cachectic states which modify the composition of the blood. The signs of hydropericardium and most of its symptoms, except fever, are those of pericarditis with effusion. **Hydropneumopericardium** denotes the simultaneous presence of gas and fluid in the pericardium. Here, as in pleurisy, it has been asked if purulent fluid may, by decomposition of its elements, produce gas. This view is no longer admitted. The common causes of hydro-pneumopericardium are injury, communication between the pericardium and an abscess of the liver (Graves), the œsophagus (Chambers), or a cavity in the lung (Dowel). The presence of gas in the pericardium shows itself by a tympanitic sound in the precordial region. On auscultation the heart sounds assume a metallic tone, and if the fluid is in sufficiently large amount, the heart, striking the fluid and gas, produces a kind of gurgling, analogous to the noise of a windmill wheel (Laënnec).

\* These secondary lesions affect not only the right ventricle and tricuspid valve, but also the left ventricle and mitral valve (Jaccoud).



## CHAPTER II

### DISEASES OF THE ENDOCARDIUM

#### I. ACUTE ENDOCARDITIS.

**Endocarditis** is inflammation of the endocardium.

**Discussion.**—It is customary to divide acute endocarditis into two groups : on the one hand simple, more or less benign endocarditis, and on the other infective endocarditis, which corresponds to the old name malignant. This division, though convenient for a theoretical description, is one which I consider artificial, as it is known to-day that every case of acute endocarditis is more or less of an infective nature, and dependent upon the presence of micro-organisms. Further, upon what are we to base our division of acute endocarditis ? Not on the nature of the lesions, for infective endocarditis, which so often causes vegetations and ulcerations, has not alone the privilege of causing these lesions. We find cases of simple endocarditis, which are so benign that they do not enter into the category of “ infective,” but yet they may be accompanied by vegetations which lead to embolism and to ulcerations of the valves or the chordæ tendineæ.

Can the **ætiology** of endocarditis and the bacteriological knowledge acquired during the past few years serve as a basis of classification ? For example, can rheumatic endocarditis be considered as being characterized by its relatively benign course, by lesions that are limited to the heart, and by the absence of embolic complications ? And can endocarditis in the puerperal state, in certain cases of pneumonia, of scarlet fever, etc., be infective and accompanied by cardiac and extracardiac lesions, by mechanical and septic emboli, and characterised by general characters and symptoms that are too often exceedingly grave ? Not so ; ætiology, although having a relative part in the anatomical and clinical evolution of endocarditis, cannot be the starting-point of a division, for exceptions would be met with every moment. Endocarditis in the course of scarlatina, of smallpox, or of erysipelas, which are eminently infectious diseases, is often benign and almost latent, while endocarditis in the course of true rheumatism may exceptionally assume a typhoid and malignant form.

The nature of endocarditis, the variety of its lesions, the diversity of its

micro-organisms, the benign or grave nature of its course, depend, as always, on the co-operation of certain causes which mutually support or oppose one another. We must especially remember the specific origin of the disease, the quantity and quality of the pathogenic agent, the previous condition of the endocardium, and the state of receptivity and of resistance of the individual. I shall, in order to facilitate description, adopt the artificial division of endocarditis into two groups, first stating the fact that they are related clinically by numerous intermediary forms. In the first group I shall describe simple acute endocarditis, and shall choose for my type the most common of all—rheumatic endocarditis; in the second group I shall have to discuss the chief forms of infective endocarditis—viz., pneumonic, pyæmic, puerperal, etc.

In the course of this description we shall find that certain cases of endocarditis do not merit the name “infective,” for they show no symptoms of infection. On the other hand, they do not merit the name of “simple,” for, in spite of the benign nature of their symptoms, they are sometimes accompanied by the formation of large emboli, which block the arteries of the limbs or of the brain. I call these cases **emboligenous**, this term being applied to emboli of a certain size, and not including, of course, capillary emboli and the formation of hæmoptoic or of septic infarcts, lesions described under infective endocarditis. We may, therefore, see simple emboligenous, and infective or malignant, endocarditis.

### Simple Acute Endocarditis.

**Ætiology.**—Many infectious diseases—blennorrhagia, scarlatina, diphtheria, varioïa, the ætiologic importance of which has been too much exaggerated (Quinquaud), erythema nodosum (Trousseau), chorea (Sée), facial erysipelas (Jaccoud), pneumonia, and malaria—may cause endocarditis, which is often microbic, and from its characters and symptoms would in many cases deserve the name of simple endocarditis; often, indeed, so simple that it would pass unnoticed without minute examination of the heart, and, in fact, several of these cases recover without leaving any sequelæ.

Such cases will not serve as the type in the present description. I would rather choose **rheumatic endocarditis**, which is the most common of all, but conforms to the type of simple acute or subacute non-infective endocarditis.

Acute, subacute, or chronic articular rheumatism may provoke endocarditis (Bouillaud), but it is chiefly in the course of **acute articular rheumatism** that endocarditis develops. I refer the reader to the article on **rheumatism**, where this question is treated in detail. Endocarditis usually appears in the second week of the rheumatic attack, but in some exceptional



cases it precedes the articular manifestations. Rheumatic endocarditis especially affects adults and children.

**Pathological Anatomy.**—The inflammation nearly always chooses the endocardium of the left heart, affects the mitral more often than the aortic valves, the central face of these valves more often than the parietal one, and their free edge rather than other parts. This localization depends on several causes : (1) the mechanical effects of pressure and friction exercised at these points by the blood-current ; (2) the lessened resistance of the affected parts of the valves, which are, at the same time, more worn out and not so well organized (Peter).

In order to understand the changes in endocarditis (acute or chronic), we must remember the structure of the endocardium, which much resembles that of the endarterium. The endocardium is composed of an endothelium made up of a single layer of flat cells ; a second layer, formed by flattened cells that are superposed and separated by lamellar substance ; and a third layer of elastic tissue and of bundles of connective tissue (Cornil).

The first layer is not found post mortem ; the second layer is very thin at the level of the valves ; the third layer (fibro-elastic tissue), at the free edge of the auriculo-ventricular valves, gives origin to the tendons of the papillary muscles. The **auriculo-ventricular** valves may be considered as a fold of endocardium, the two lips of which are united by fibrous connective tissue, insuring resistance (Ranvier). The **arterial** valves result from the folding back of the internal membrane of the arteries on the ventricular endocardium.

When endocarditis is slight and transient, it is rather exudative than proliferative. The type of proliferative endocarditis is the **rheumatic** form, which we are now considering. The inflammation chiefly attacks the mitral valve, the edges of which are swollen, thickened, and much vascularized, while in the normal state the capillary vessels are few in number. These vessels are affected by endarteritis, and their lumen is almost obliterated.

The changes develop in the layer of flattened cells. Micro-organisms often cause the embryonic elements to proliferate and form granulations, which are so small and so numerous that they give to the endocardium a rough appearance. These granulations, which were at first taken for collections of fibrin, are really composed of embryonic tissue, covered by a thin layer of fibrin. They are friable, soft, and transparent in the acute stage, and some show at their centre vessels in process of formation. The granulations may invade fairly large surfaces of the parietal, ventricular, or auricular endocardium, but are usually found at a small distance from the free edge of the valves, where they form a wavy, mammillated line.

The granulations, which sometimes reach the size of a pin's head or of

a pea, may be villous, filamentous, nummular, warty, or raspberry-like, and surround the valves like a garland. They may give rise to **emboli**, which are carried in every direction. These complications, however, will be discussed later under Emboligenous and Ulcerative Endocarditis.

**Description.**—The phenomena of **invasion** in acute endocarditis are, so to say, non-existent. Rigors, dyspnoea, and palpitation are not seen, or at least are so slight that endocarditis passes unnoticed in a case of articular rheumatism, unless the heart is examined daily.

In valvular endocarditis we find **blowing murmurs**, which are related to the seat of the lesions and will be described under Valvular Lesions. All the orifices of the heart may be affected, but for the moment it is enough to say that the mitral orifice is most generally attacked, and the aortic orifice is next in frequency. The murmur that characterizes mitral endocarditis is heard at the apex of the heart—that is to say, below and outside the nipple; this soft murmur is nearly always systolic, mitral insufficiency being the rule and stenosis the exception, during the acute stage of endocarditis (Bouillaud). The duration is rarely more than two or three weeks. After this time the trouble resolves and the murmur disappears, but the mischief may become chronic, when the murmur also persists.

Endocarditis which is consecutive to such infectious diseases as erysipelas, mumps, erythema nodosum, etc., may recover without leaving any trace. Recovery, however, is not to be relied on. A patient who has suffered from rheumatism or scarlatina, and been considered as cured because the murmur has disappeared, may yet suffer from chronic endocarditis, which is latent in its evolution. The morbid process is not extinct, but proceeds slowly and insidiously to alter the tissues which it has attacked, and often creates irremediable lesions, that may only appear after years. Simple acute endocarditis, however, presents no immediate danger, and its gravity results from the chronic valvular lesions which so often follow.

Local blood-letting, leeches, cupping, blisters to the precordial region, and the preparations of digitalis form the basis of treatment. We must never lose sight of the frequent change from the acute to the chronic state. Blisters and applications of the cautery must therefore be continued for a lengthy period when the disease has left some traces.

### **Ulcerative, Infective, or Malignant Endocarditis—Emboligenous Endocarditis.**

**Pathogenesis.**—The acute endocarditis just described presents no immediate gravity; it is only formidable in the future, because of the chronic valvular lesions which too often follow. We find, however, another form of acute endocarditis, called typhoid, ulcerative, infective, or malignant,



which at its onset or in its course may show a typhoid or septic character, and ends fairly frequently in death.

We know to-day that there is, not one, but several varieties of infective endocarditis.

Discussion of their pathogenesis is singularly simplified since the discovery of bacteria; their ætiology is no longer given up to simple hypothesis, as was formerly the case, and we can with some method group the lesions and the symptoms that are peculiar to each variety. The nature and the degree of virulence of the micro-organisms, the previous condition of the endocardium, the conditions of resistance or receptivity of the individual, are all factors which have to be taken into account.

This question may, I think, be stated as follows: The endocardium, whether healthy or previously injured so that it is **in a state of receptivity**, offers a favourable site for the arrest and development of certain pathogenic agents.

These agents may have the most varied **origins**. Some enter the economy by the skin (abrasions, excoriated corns, boils, burns, wounds, erysipelas, injuries); others are introduced through the mucosa of the uterus (abortion and accouchement), through the genito-urinary mucosa (lesions of the urethra and bladder), and through the digestive mucous membranes (stomatitis, tonsillitis, ulcerations of the stomach, the intestines and the bile-ducts); others, again, penetrate through the serous membranes (lesions of the peritoneum, pleura, and synovial membranes); some are introduced through the respiratory channels (broncho-pneumonia, ulcerative lesions of the lung, gangrene, and bronchiectasis); others may enter through bone lesions (osteomyelitis); finally, in some cases the entrance gateway cannot be found, in which event we invoke auto-infection, calling the endocarditis **primary**.

Many microbes may cause infective endocarditis. I shall first cite the pyogenic microbes, streptococci and staphylococci; then the pneumococcus, the bacillus of typhoid fever and of tuberculosis, the gonococcus, and other microbes not classified, or not yet found in other diseases (Weichselbaum, Gilbert and Lion).

These microbes are chiefly aerobic, a fact which doubtless explains their preference for the left heart and for oxygenated blood. Endocarditis of the right heart is more rare. Cases have, however, been published. These microbes, after entering the economy by one of the gateways above enumerated, are carried by the blood to the heart, and invade the endocardium, either superficially, through the crevices in the connective tissue, or deeply, by way of the small vessels. This invasion may be helped by previous changes in the serous membrane. After implantation on their culture medium, the pathogenic agents, either alone or in combination,

crown their work of destruction, and the pathological process may assume several forms. In the **first variety** the lesion leads to the formation of vegetations which are more or less bulky and friable. These vegetations become detached from the endocardium, and are launched into the vessels in the form of emboli. If the embolus is of large size, it obliterates a fairly large artery, and causes secondary complications, which depend on the obliterated vessel (hemiplegia, aphasia, and gangrene). If the embolus is of smaller size, it causes **infarcts** in the kidney, spleen, intestine, or lung, and the symptoms depend on the organ invaded. In this first category of complications the embolus causes simple obstruction of the vessels. The embolism is called **mechanical** and may be formidable, but is not septic. In short, the cases of endocarditis which form this first variety are **emboligenous**, but do not deserve the name of infective, because they do not give rise to septic or infective emboli.

In the **second variety** the morbid process goes on to necrosis and ulceration, and in most cases vegetations and ulcerations are present together. These ulcerations may perforate the valves, tear away the pillars, and detach shreds of the chordæ, giving rise to mechanical embolism. Here also the endocarditis may not be infective, but rather ulcerative, with vegetations. It is emboligenous and most grave, but the process may not be infective.

Lastly, in the **third variety** I place the true cases of infective endocarditis. Ulcerations and vegetations are as a rule present, but the important feature is that their products are **septic** and infective: they pour septic capillary emboli into the blood-stream, and their symptoms recall the picture of typhoid or purulent infection.

From this rapid enumeration we see that we should be wrong in including all cases of severe or of fatal endocarditis under the term "infective." There is a place for intermediate cases, according as the morbid process is accompanied by infecting pathogenic agents or not.

**Pathological Anatomy.**—At the onset of **malignant** endocarditis (I intentionally preserve this epithet "malignant," as it applies to every endocarditis which is not of the simple type) under the influence of micro-organisms, we find in the valvular tissue infiltration of wandering cells, hypertrophy and multiplication of cells, and desquamation of the endothelial layer, which is replaced by fibrin. In the meshes of the fibrinous reticulum colonies of bacteria are found, which "penetrate by more or less large chinks into the interior of the valves."

In cases of malignant endocarditis, the inflammation nearly always gives rise to large vegetations and deep ulcerations. In such a case we find on the sigmoid or on the mitral valves, on the chordæ tendineæ, on the papillary muscles, and on the median septum, a vegetation of the size of a



pea or of a strawberry, or a collection of smaller vegetations, of which some are flattened, papilliform, or raspberry-like, while others are pedunculated and ready to break off.

These vegetations, which are formed largely of embryonic tissue and are covered by a mass of fibrin, are very rich in microbes, which are found either on the surface in the fibrinous layers or in the deep parts of the vegetations. It is probable, therefore, that the bacteria deposited on the valves by the blood-stream develop in the superficial fibrinous layers, and then penetrate through the crevices of the connective tissues as far as the surface and the central parts of the valves. It is also possible that bacteria are primarily introduced by the vessels in the valves.

In some cases the vegetations soften into a kind of atheromatous pulp, composed of granulations, cellular débris, fatty elements, and various septic micro-organisms. These different elements, when launched into the blood-stream, cause a source of emboli, which are sometimes mechanical, at other times septic, and affect the brain, limbs, spleen, liver, intestines, kidneys, etc. The organs affected show hæmoptoic and suppurative infarcts, with miliary abscesses, abscesses of the skin and the joints, suppuration of the cranial and spinal meninges; diarrhoea, albuminuria, and enlargement of the spleen are found, and the patient often succumbs to gangrenous, typhoid, toxic, or infectious complications, etc.

The spleen is usually bulky, as happens, moreover, in many infectious diseases.

During life the **blood** contains micro-organisms which can be cultivated.

Let us now pass on to the **ulcerations** of malignant endocarditis. They commence as small superficial yellowish patches; little by little they become deeper and more extensive, and may perforate the valves, causing valvular aneurysms (Foerster, Pelvet). The aneurysms of the sigmoid valves are formed at the expense of the superior face of the valves, those of the mitral valves at the expense of the inferior face. This localization is regulated by the direction of the blood-current, the enlargement taking place on the side of the greater blood-pressure.

These ulcerations also cause perforation of the intraventricular septum and rupture of a pillar, or of the chordæ tendineæ. This condition forms a new source of emboli. In many cases, let me repeat, malignant endocarditis is at first vegetative and ulcerative. If we analyze the cases and consult the results of autopsies, we shall find a double process of exuberant proliferation and deep ulceration. These different processes explain the method of formation of emboli and their multiple varieties.

**Experimental pathology** has succeeded in producing infective endocarditis. Ribbert, in 1885, by injecting  $\frac{1}{2}$  gramme of culture of *Staphylococcus aureus* into the ear of a rabbit caused embolic foci of carditis and of

valvular endocarditis, with consecutive lesions of acute endocarditis. Wysokowitsch varied the experiments. By means of a silver sound passed into the right carotid he first injured the cardiac valves, and then introduced micro-organisms of different kinds (*Staphylococcus pyogenes*, *Streptococcus septicus*, etc.), producing not only parasitic foci of endocarditis, but he also found in the different organs emboli with infarcts and abscesses which contained the same microbes as in the endocardium.

**Description.**—In infective endocarditis the general symptoms are, as a rule, much more important than the local signs. On auscultation, one or several murmurs may be recognized, depending on the severity of the lesion and on the orifice affected. In some cases the murmur is soft or masked by pericarditis, in other cases it acquires a musical tone called **whining** (Bouillaud). This whining murmur, which is also met with in chronic valvular lesions, has a peculiar interest in ulcerative endocarditis. It may be due to the vibration of a body (pillar, cord, or vegetation) floating in front of an orifice, and has often given warning of the onset of embolism (Gubler, Potain).

Embolism, whatever its origin, may act in a mechanical way, obliterate a cerebral artery, such as the Sylvian artery, or its branches, and give rise to apoplexy, hemiplegia, aphasia, or softening of the cerebral tissues. The embolism may obliterate the artery of a limb and produce consecutive gangrene. These complications are very serious, but, I repeat, they are not infective, and the endocarditis which has given rise to them is purely emboligenous, causing complications of a mechanical and not of an infective nature.

In other cases, on the contrary, the endocarditis is essentially infective, the general symptoms dominate the scene, and we see from the first that the patient is suffering from an infective disease of a typhoid or pyæmic type.

When ulcerative endocarditis assumes the **typhoid** form, dryness of the tongue, prostration, rapid elevation of temperature, shivering fits, which appear during the first days of the attack, are symptoms that give the patient a "typhoid look"; add to this, broncho-pulmonary congestion, stupor, ballooning of the belly, enlargement of the spleen, albuminuria, and diarrhoea, and we see the difficulty of diagnosis between this typhoid state and typhoid fever. However, the diagnosis of endocarditis is aided by the murmurs audible at the orifices of the heart, by the absence of lenticular rose spots, by the course of the symptoms, by examination and culture of microbes in the blood, and by negative sero-diagnosis. This endocarditis appears to depend on the presence of encapsuled lanceolated microbes (Jaccoud).

The **pyæmic** form of ulcerative endocarditis closely resembles purulent



infection. We find repeated chills and high temperature, and, indeed, the case is really one of infection, because the endocarditis throws septic emboli and specific bacteria into the blood-stream. These capillary emboli form ecchymotic spots on the surface of the skin and the serous membranes, suppurative infarcts, miliary abscesses, and superficial or deep foci of gangrene, and provoke suppurative subarachnoid meningitis. The patient has a subicteric tint; abscesses develop under the skin, and death follows in delirium. This pyæmic form is chiefly met with when the micrococci of suppuration are present in the blood (Jaccoud).

When the valves of the **right heart** are affected, the **lungs** present ecchymotic spots, hæmoptoic, gangrenous, or suppurating infarcts and abscesses, varying in size from a pea to a nut. The diagnosis is based on auscultation of the heart, and on the finding of foci of suppuration or of phlebitis.

For description I have chosen different types of infective endocarditis, but I would hasten to say that these types are not always so clearly marked clinically, and the different varieties just described are often combined.

The **course** and **duration** of the disease vary in each case. Marked elevation of temperature is not always seen. The disease, instead of being continuous, may show successive attacks, with complete remissions of fairly long duration. The prognosis is not absolutely bad, and recovery has occurred in a fairly large number of cases (Jaccoud). The typhoid form may last several weeks, but the pyæmic form is more serious and rapid.

**Varieties.**—I shall now enumerate the chief varieties :

1. **Rheumatic Endocarditis.**—Rheumatism causes simple, emboligenous, or infective endocarditis. The simple form is much the most common, and has served as our descriptive type of acute endocarditis. Rheumatism, however, may give rise to emboligenous endocarditis, with large emboli in the cerebral arteries and in the vessels of the limbs (Jaccoud). It may also cause infective endocarditis of the typhoid form. Infective endocarditis rarely arises during a first attack of rheumatism; it usually shows itself in the individual whose endocardium is already the seat of chronic endocarditis. This, says Jaccoud, is the most usual cause of infective rheumatic endocarditis. The pathogenic agent of rheumatism is still unknown, but in some cases the lesions in malignant rheumatic endocarditis are due to the association of microbes.

2. **Pneumonic Endocarditis.**—Pneumococcal endocarditis may be parapneumonic or metapneumonic; it may precede pneumonia (prepneumonic) or be quite independent. This variety has a tendency to form vegetations rather than ulcers, and sometimes causes small abscesses in the endocardium. It affects the aortic more often than the mitral orifice, but has also been found in the right heart, affecting the tricuspid and pulmonary valves. Pneumonic endocarditis is rarely emboligenous, because the vegetations

are implanted upon a large base. It rarely gives rise to capillary emboli, but is sometimes infective and suppurative; meningitis is fairly often associated with it.

Pneumonic endocarditis may develop in patients who have had no previous cardiac defect. Old lesions of the valves are, however, singularly favourable to its development. The pneumococcus is not always the sole cause; other microbes—streptococci and certain unclassified bacilli—are sometimes associated with it.

This endocarditis usually passes unnoticed if we do not examine the heart in patients suffering from pneumonia. It fairly often assumes the simple form, and may recover without leaving any traces. Pneumonic endocarditis has been produced experimentally in the rabbit (Netter), after the valves have previously been injured.

**3. Endocarditis in Pregnancy and the Puerperal State.**—In infective endocarditis associated with **pregnancy** it is necessary to distinguish endocarditis gravidarum, which supervenes during pregnancy, from endocarditis which follows post-puerperal trauma of the uterus. The latter is only a variety of septicæmia, and is much more serious than the former. Non-puerperal trauma of the uterus may produce the same result.

Endocarditis gravidarum is less frequent than the puerperal form. Its origin is more difficult to study, for it does not appear to depend on an infection of uterine origin. This variety cannot, however, be looked upon as a simple one supervening in the course of pregnancy. The gravid state makes the prognosis of the disease worse, and the foetus itself suffers from the maternal infection. The researches of Nattan-Larrier have shown that maternal endocarditis causes important histological changes in the organs of the foetus. The toxins in the blood of the mother pass through the placenta, and determine reaction of the blood-forming organs; while the liver and kidneys show lesions of degeneration which vary directly with the severity of the infection. The evolution of gravid endocarditis is, moreover, variable, and bacteriological examinations have shown that it may be produced by the pneumococcus, the streptococcus (Netter, Weichselbaum), or by unclassified bacilli (Girode).

Puerperal endocarditis shows vegetations and ulcers, and the resulting capillary emboli are septic in nature, and may lead to hæmoptoic or suppurating infarcts. It is generally due to a streptococcus of extreme virulence. The clinical picture is habitually that of the pyæmic form, and though the prognosis is very grave, recovery may take place. Some years ago, with Champetier de Ribes, I saw a case of post-puerperal endocarditis, with septicæmic symptoms, multiple abscesses, and mitral lesions. The patient recovered from the infection and the cardiac lesion—at least, the murmurs disappeared.



These attenuated forms depend, doubtless, on a streptococcus of slight virulence. The streptococcus is not the only pathogenic agent which may be met with in puerperal endocarditis. Other microbes may infect the uterine wound, and give rise to septicæmia with endocardial lesions. The staphylococcus (Ch. Leer), the pneumococcus (Schahl and Hergott), the coli bacillus (H. Rendu), have been found either in isolated cases or in epidemics of puerperal infection in which the streptococcus was not present.

**4. Endocarditis of Septicæmic and Pyæmic Origin.**—This form is very common, and is consecutive to a suppurating wound on the surface of the body or in the deep tissues of the organs (urethra, kidney, bladder). It is chiefly due to the entrance of the *Streptococcus pyogenes* and the *Staphylococcus pyogenes* into the blood, either alone, together, or associated with other microbes.

When the mischief is due to the streptococcus, the ulcerations may be extensive, but the vegetations are usually small, soft, and greyish. Infarcts are very frequent, and suppurate rarely, unlike those caused by the staphylococcus, which suppurate frequently.

When endocarditis is due to the *Staphylococcus aureus*, as in the case of boils, or of osteomyelitis, as well as of many central or peripheral suppurative lesions, the disease more usually affects the left than the right heart. Vegetations are more common than ulcerations; the vegetations are small and soft, while the infarcts are suppurative. **Miliary abscesses** are found in the spleen, kidneys, lungs, and brain. The clinical picture recalls the description of purulent infection, with typhoid state, eruptions, and cutaneous suppurations. In the Necker Hospital I had a patient suffering from malignant endocarditis of the typhoid form, caused by the *Staphylococcus albus*, which was found in a pure state during life, and after death in the pus from the miliary abscess in the endocardium.

Streptococcal endocarditis consecutive to **erysipelas** is extremely rare. Curable endocarditis, however, is found fairly frequently during the course of erysipelas.

**5. Typhoid Endocarditis.**—Primary typhoid endocarditis, due to Eberth's bacillus, is very rarely seen. Cases of secondary typhoid endocarditis, due to the coli bacillus and other microbes, are more frequent.

**6. Biliary lithiasis** may provoke infective endocarditis, by reason of germs derived from the bile-ducts. The infection appears to be due to micro-organisms derived from the intestine.

**7. Tubercular Endocarditis.**—This form has only been seen in acute miliary tuberculosis. It appears either in the form of isolated granulations—i.e., miliary tubercles, which exist most often on the free edge or auricular face of the mitral valve—or in the form of vegetations.

In certain cases Koch's bacilli have been found in these lesions, which

may therefore be considered specific. Further, tubercular endocarditis has been produced experimentally in the rabbit by intravenous injection with cultures of Koch's bacillus, preceded by injury of the aortic valves. The vegetations on the valves, produced at the same time as the general miliary tuberculosis, contained the tubercle bacillus. More rarely these lesions are caseous. Koch's bacilli are also present in them.

These various lesions are latent, and do not produce special symptoms.

Lesions of the endocardium in tubercular patients are not absolutely rare, but they are not always due to Koch's bacillus. The latter are cases of old endocarditis, independent of tuberculosis or of recent more or less vegetating lesions, which have nothing specific as regards histological structure, and in which bacteriological examination sometimes reveals other micro-organisms than Koch's bacillus. These recent lesions found post mortem in tubercular patients, most frequently upon the mitral valve, may therefore be considered as due to secondary infections (P. Tessier).

8. **Blennorrhagic Endocarditis.**—If the reader will turn to the chapter on Blennorrhagia, he will find cases of endocarditis due to the gonococcus.

## II. CHRONIC ENDOCARDITIS.

**Ætiology.**—**Chronic endocarditis** arises from the same causes as acute endocarditis, rheumatism claiming the largest share. In alcoholic, syphilitic, gouty, or elderly persons it is sometimes secondary to fatty, fibrous, or atheromatous changes which invade the arterial system. The coexistence of tuberculosis and mitral endocarditis has been noted by Potain and Tessier. Chronic endocarditis often follows the acute form, but at other times it is primary, and becomes established in an insidious manner. Its course is slow. The valvular lesions develop unknown to the patient, and in rheumatic cases many people, whose health is apparently excellent, only show the first symptoms of valvular mischief after a forgotten attack of acute articular rheumatism.

**Pathological Anatomy.**—The **vegetations**, which are soft and friable in acute endocarditis, are here hard and fibrous, and the vessels that are so numerous in the acute stage over the swollen mitral valve disappear in proportion as the lesion becomes chronic. The inflammatory process, which commences in the layer of flattened cells, reaches the subjacent layer, which is rich in connective tissue, and gives rise to **fibrous** tissue. In this fibrous tissue we find islets undergoing fatty change, **atheromatous** centres and **calcareous** incrustations, lesions which present great analogy with endarteritis deformans. This change in the endocardium, and the retractile property of the fibrous tissue explain the alterations in the valves and orifices of the heart.



Every part of these orifices is invaded. The **chordæ tendineæ** and the auriculo-ventricular valves are shortened and indurated; the **fibrous zone** which surrounds the orifices is hypertrophied. The **musculi papillares** are also affected. They are sometimes thickened and shortened—the valves may be three or four times larger than normal—or, on the other hand, atrophied or adherent. Their edges are fused, jagged, thickened, and covered with granulations. The vegetations are chiefly situated on the auricular surface of the auriculo-ventricular valves and on the ventricular surface of the sigmoid valves. These lesions, in which adhesions, fibrosis, atheroma, and calcification are combined, finally cause deformity of the orifices, and impede the action of the valves; insufficiency of the valves and narrowing of the orifices then follow. The muscular fibre of the ventricles and auricles hypertrophies because of the increased work, and compensation is established; but later when the muscular fibre is also invaded by the morbid process, the *columnæ carneæ* and the ventricles show fibrous changes, and their vessels are affected by endarteritis, which causes malnutrition. The degenerated cardiac muscle no longer fights with the same efficiency.

In some cases (old age, alcoholism) the lesions do not begin in an inflammatory, but in a retrogressive process, which in its turn induces inflammation. **Atheroma** plays the principal part. The atheromatous degeneration appears here, as in some cases of endarteritis, to be the primary lesion (Cornil and Ranvier).

The **symptoms** of chronic endocarditis closely resemble those of valvular lesions of the heart, which will be studied in the following sections.

### III. VALVULAR LESIONS OF THE HEART.

**General Survey.**—Whether endocarditis is of microbic origin, as in most acute and also in many chronic cases, or is consecutive to the atheromatous lesions of gout and old age, the lesion may cause transient or permanent insufficiency and stenosis. An orifice is said to be **stenosed**, when instead of allowing ten parts of blood to pass at each beat of the heart, it only allows seven, five, or even less, to do so. **Insufficiency** is present when the valves which normally prevent all regurgitation of blood no longer plug the orifice, and hence allow the blood-wave to regurgitate. The four orifices of the heart (arterial or auriculo-ventricular) may be affected, but those of the left heart are much more often involved than those of the right. Stenosis and insufficiency are often combined at the same orifice; each of them may, however, show itself alone. This dissociation is chiefly seen in insufficiency due to mechanical causes. Thus enlargement of the right ventricle brings with it dilatation of the tricuspid orifice and insufficiency

of the valves, just as enlargement of the aorta, may bring with it dilatation of the aortic orifice and insufficiency of the sigmoid valves.

Further, whatever be the mechanism of the lesion, whichever be the orifice affected, as soon as the distribution of the blood-waves is not regular, and one of the parts of the cardiac machinery is affected, the whole circulation feels the shock. It is true that the shock is felt more or less slowly according to the orifice damaged and to the efficacy of compensation, but the final result is the same, and, as Jaccoud has forcibly said, valvular lesions raise the pressure in the veins and lower it in the arteries, which is equivalent to saying that they finally cause passive congestion, œdema, dropsy, thrombosis, and hæmorrhage, with all their train of functional troubles.

Valvular lesions may, nevertheless, be neutralized, even for a long time, provided they are compensated. **Compensation** is a kind of substitution, due to dilatation of the cavities of the heart and to hypertrophy of its walls. Let us take, for example, aortic insufficiency : The left ventricle is subjected to an excess of pressure by the surplus of regurgitated blood, and its muscular fibre undergoes marked hypertrophy. This mechanism of compensation, which exists in different degrees, according to the orifices affected, renders the valvular lesions harmless for a period varying in duration with the nature and the seat of the lesion.

A time comes, however, when the compensation is no longer sufficient. The cardiac muscle, which is badly nourished and invaded by fibrous tissue, while its vessels are damaged by endarteritis, has no longer the necessary energy to face the danger. The functional troubles become more marked ; congestion, blood-stasis, and dropsy progress ; malnutrition becomes general ; irremediable lesions develop in the organs ; cyanosis, dyspnœa, and coma gradually reach their maximum ; and the condition is summed up in a single word—**asystole** (Beau).

While the heart is being worn out, the small vessels which form the peripheral and local circulation become affected and lose their resistance. Each organ becomes diseased in its turn, and the disease of the heart is changed into a disease of the whole organism.

**Asystole**, however, may, under the influence of proper treatment, or even spontaneously, improve for a time ; but after some remissions, of an uncertain duration, the patient reaches the stage of **cardiac cachexia** (Andral). The disease is no longer confined to the heart, but affects the whole body. " Thus, when he has ceased to live, the patient has in reality only ceased to die " (Peter).

In patients dying from cardiac cachexia we find general changes. The **lungs** are the seat of congestion, œdema, infarcts, and hæmorrhage. The brain is congested ; the **cerebral sinuses** are engorged with dark blood.



The **liver**, which is large and indurated, presents the so-called "nutmeg change." The kidney, which is enlarged, shows multiple arborizations on its surface. The **spleen** is engorged with blood. The **heart** is generally enlarged; its muscular fibre is pale, degenerated, and studded with fatty and fibrous islets. It contains clots—some yellowish, fibrinous, firm and adherent, and consequently of old formation; others soft, cruoric, and of recent formation.

Diseases of the heart have not always this slowly progressive course. Some patients die suddenly (aortic insufficiency); others are stricken down by complications (pulmonary hæmorrhage, cerebral embolism). In some the disease is rapid, and cachexia is established in a few months, while in others infective endocarditis is grafted upon an old valvular lesion, and is sometimes fatal.

**Signs and Symptoms.**—The clinical account of diseases of the heart cannot be dealt with in a general survey, because the symptoms of mitral disease are very different from those of aortic disease. I refer the reader, then, to the separate study of each orifice. This statement does not hold good with regard to the signs of cardiac lesions; and notably to the abnormal sounds, the mechanism of which will be better grasped in a general survey.

**Mechanism of Abnormal Sounds.**—**Locally**, the valvular lesions of the heart are shown by morbid sounds called **murmurs** and **reduplications**, the interpretation of which rests upon the previous knowledge of the physiological sounds.

In the normal state two sounds are heard at the apex: the first is long and well marked, the second is duller and shorter. At the junction of the sternum and the second right intercostal space two sounds are also heard, but the rhythm is the inverse of that at the apex. The first or systolic sound is dull; the second or diastolic sound is longer and more marked. As two sounds are produced at each orifice of the heart, and as there are four orifices, it follows that eight sounds are produced in each cardiac cycle; but only four are perceptible (two at the apex and two at the base), because in the **normal condition** they blend and give rise to two sounds at the apex and two sounds at the base. To what are these sounds due? Each of them is composed of a **chief** element, caused *in situ*, and an **accessory** or propagated element.

**First Sound.**—At the **apex** of the heart the **first** sound has for its chief element the snapping of the valves (mitral and tricuspid), and for its accessory element the re-echoing of the first sound of the base, which is due to a sudden distension of the arterial walls (aorta and pulmonary artery) by the blood-wave. At the **base** of the heart the **first** sound is formed of the same elements, with this difference—that the element which was accessory at the apex becomes here the chief one, and *vice versa*.

**Second Sound.**—At the **apex** of the heart the **second** sound has for its chief element the passage of blood into the ventricles (Skoda's interpretation), and for its accessory element the snapping of the sigmoid valves. At the **base** of the heart the **second** sound is made up of the same elements, with this difference—that the element which was accessory at the apex becomes here the chief one, and *vice versa*.

In short, whether it be a case of valvular snapping or of sudden distension of the walls, all normal sounds of the heart are **solid** sounds (Monneret); that is to say, produced by the vibration of solid parts. In pathological conditions, however, the solid sounds undergo the following modifications: First, they are replaced by **murmurs**—that is to say, by **fluid** sounds, due to vibrations of a blood-wave traversing a diseased orifice; secondly, they are **reduplicated**, the reduplication occurring in the first or in the second period; thirdly, they may disappear without other modifications, and without being replaced by any abnormal sound.

The **blowing murmurs** are variable in tone and intensity. They may be **softened** and prolonged (aortic insufficiency), **hissing** like a jet of steam (mitral insufficiency), **strident** (aortic stenosis), or so little marked that they resemble rather a dull rumbling than a blowing sound (mitral stenosis). In some cases the murmur is changed into a musical sound, called **whining** (Bouillaud). The whining is due either to the nature of the lesion of the orifice, or to some floating shred thrown into vibration in front of an orifice by the blood-wave.

I cannot, however, too strongly insist on this point that the presence of a murmur or of a reduplication **is not sufficient** to prove an organic lesion. We find reduplications and murmurs which have nothing to do with the lesion of an orifice. Such are the important **extra-cardiac** murmurs, so carefully described by Potain; such are the normal reduplications, “resulting from transitory changes which the movements of respiration cause in the pressure of the blood contained in the heart and the great vessels” (Potain).

Such, also, are the aortic and mitral murmurs due to chloro-anæmia.

Let us, however, return to the organic murmurs of the heart and apply the preceding data to each of the valvular lesions.

1. **Mitral insufficiency** is characterized by a **systolic murmur at the apex**. During the ventricular systole in the normal condition the blood-wave from the left ventricle passes completely into the aorta, because the mitral valve plugs the auriculo-valvular orifice; but in the pathological state, as the valve is not able to plug this orifice, part of the ventricular blood-wave flows back into the auricle, and a **systolic** murmur is produced. This murmur has its maximum near the apex of the heart, at the level of the mitral orifice below or outside the nipple.



2. **Mitral stenosis** is characterized by a **diastolic** murmur, by a **pre-systolic** murmur, or by **reduplication of the second sound**. In order to understand the mechanism and the value of these pathological sounds which are heard at the apex of the heart, we must briefly discuss the ventricular diastole. During diastole the ventricle is dilated, the auriculo-ventricular orifice opens widely, and the blood passes through the auricle into the ventricle. The passage of the blood is, however, somewhat slow. It begins gradually during the ventricular diastole, and terminates more suddenly during the pause, when the auricle is contracted. If, then, the murmur of stenosis is produced during the first part of the passage (which is very rare), it is diastolic (Hérard); if it is produced during the second part of the passage, it corresponds to the pause, and is therefore presystolic (Gendrin). The murmur of mitral stenosis is not loud, because the blood-wave is feebly pushed through the constricted orifice, even when the auricle is hypertrophied. Accordingly, this murmur is rather a presystolic booming or rumbling (Duroziez).

Mitral stenosis is also characterized by **reduplication of the second sound**, and as the second part of this reduplication may be blowing, we find a rhythm of three beats, composed of one long and two short periods. Several theories exist as to this reduplication of the second sound: the one supposes that there is a want of synchronism between the second sounds in the two ventricles; the other holds that the want of synchronism exists between the second sound at the ventricular orifice and the second one at the arterial orifice (Jaccoud). While in the normal condition these two elements are blended, in mitral stenosis they are dissociated, the ventricular being behind the arterial sound. According to Potain, the dissociation depends on the closure of the aortic and pulmonary valves, the closure of the aortic valves being premature.

However this may be, reduplication of the second sound, with or without a presystolic murmur, is a sign of mitral stenosis.

3. **Aortic insufficiency** is characterized by a **diastolic murmur at the base**. The reasons are: at the moment when the blood-wave has just been launched from the ventricle into the aorta, the backward movement finds, normally, an impassable barrier in the sigmoid valves. When, however, these valves are unable to plug the orifice, some of the blood flows back into the ventricle, and a diastolic murmur is produced.

4. **Aortic stenosis** is characterized by a **systolic murmur at the base**. If the blood-wave thrown by the ventricle meets with a constricted instead of an open orifice, a systolic murmur is produced.

#### IV. MITRAL ORIFICE—INSUFFICIENCY AND STENOSIS.

**Mitral insufficiency** is present when the mitral valve, which normally closes the left auriculo-ventricular orifice in order to prevent regurgitation of blood, does not plug this orifice, but allows the blood to flow back into the left auricle. **Mitral stenosis** is present when the lumen of the orifice has lost its normal dimensions.

Stenosis and insufficiency are most often combined, and we then speak of mitral disease. If one only is present, we speak of pure stenosis or of pure insufficiency. In many cases the process begins with insufficiency, and stenosis occurs later. Lastly, in some patients stenosis appears to develop from special causes, and remains indefinitely in a pure condition.

**Pathological Anatomy.**—**Mitral stenosis** may be due to concentric thickening and retraction of the fibrous ring. In most cases, however, the condition is due to lesions of the valves and their chordæ. A welding of the free edge of the valves occurs at the level of the commissures. This welding might be compared to the narrowing of the aperture between the eyelids from adhesions of their commissures (Bouillaud). The tendinous pillars inserted on the edge of the valves participate in the cicatricial process. They become rigid, thickened, and retracted, and pull down the bicuspid valve, which takes the shape of a rigid and flattened funnel.

In this manner stenosis is brought about, but as a rule neither the fibrous ring nor the vegetations of the auricular surface of the valve take any part. The mitral orifice, which normally is large enough to admit the thumb, becomes so narrow that it will scarcely admit a goose-quill.

Dilatation and hypertrophy of the left auricle, followed by that of the right auricle and ventricle, are the consequence; the left ventricle remains practically normal. The slowing of the blood-stream and the blood-stasis in the left auricle favour the formation of stratified clots, which are often adherent to its posterior wall. These clots, which pass into the left ventricle, and are launched into the blood-stream during systole, may become the source of more or less bulky emboli. On the other hand, the slowing of the blood-stream and the blood-stasis in the right auricle favour the formation of clots, which are also fibrinous and adherent. These clots, which are launched from the right ventricle into the lungs, are the most common cause of pulmonary infarcts.

**Mitral insufficiency** is the result of lesions which affect the valves or the fibrous ring. The lesions of the valves are thickening, induration and incrustations of the edges. There is no welding of the valvular commissures. Under the influence of the retractile tissue the shortening and retraction of the valves oppose the complete adjustment of their auricular surface during systole, and insufficiency results. The shortening



of the chordæ tendineæ, which are also thickened and indurated, opposes the complete straightening of the valve during systole, and this helps to cause insufficiency. The papillary muscles, by their fibrous retraction, may act in the same manner. Rupture of the chordæ tendineæ may be a cause of insufficiency, and I would also mention valvular aneurysms and vegetations. Mitral insufficiency, due not to a lesion of the valves and pillars, but to dilatation of the fibrous ring, does exist, but is relatively rare. In this case the left ventricle, during its dilatation, drags upon the papillary muscles and the tendinous pillars, and consequently produces abnormal tension of the valves and insufficiency. Dilatation of the left ventricle, which has previously lost some part of its contractility, is said, according to some authorities, to be a cause of pure mitral insufficiency, either transient or permanent.

Mitral insufficiency induces dilatation and hypertrophy of the left auricle, the right auricle, and the right ventricle. The left ventricle, however, which is almost unaffected in stenosis, shows slight hypertrophy in insufficiency.

**Description.**—We see persons among the leisured classes (rather than in hospital patients), who for many years have suffered from some mitral lesion, without having shown any symptoms, because the heart muscle is not overworked, and the lesion is well compensated. The functional hyperactivity of the muscle and the compensatory hypertrophy of the right ventricle struggle successfully against the evil distribution of the blood-waves, and **undertake the maintenance of equilibrium**. Dilatation and hypertrophy of the right ventricle compensate as far as possible for the increase in the pulmonary arterio-venous tension, so that mitral insufficiency and stenosis may be latent for a long while. When, however, the cardiac muscle becomes feeble, or is attacked by some morbid change, such as fibrosis, endarteritis of the small vessels, or granulo-fatty degeneration, which endangers its nutrition and its tone, we find visceral congestions, peripheral and splanchnic oedema, which result from the cardiac disease.

Breathlessness and dyspnœa, with or without palpitation, are generally the first functional troubles in mitral lesions, because the pulmonary circulation, which is **so close** to the mitral lesion, is naturally the first to be affected; bronchial catarrh, congestion, oedema, and hæmorrhage of the lungs are among more or less remote results. These lesions produce increasing distress, with attacks of dyspnœa and hæmoptysis.

**Breathlessness** may for a long time be the only symptom of the mitral lesion. The patient is "short of breath"; he does not notice it while at rest, but when he walks upstairs or makes an effort, distress appears. At this period the embarrassment of the pulmonary circulation does not betray itself by any physical signs on auscultation. The slowing of the blood-

stream and the marked pressure to which the flow in the pulmonary artery is subjected are two of the causes of this dyspnœa. On account of the mitral lesion the blood tarries or flows back into the left auricle, the blood-pressure increases in the auricle, the pulmonary veins and arteries, and the distension of the capillaries of the pulmonary alveoli provokes rigidity and swelling of the lung. Indeed, according to some authors, this rigidity and swelling is said to be the chief cause of cardiac dyspnœa.

Later, as the result of pulmonary œdema, the dyspnœa becomes more persistent or intense, and auscultation reveals subcrepitant râles, which are usually present at both bases of the lungs. During this progressive dyspnœa, or even apart from it, the patient sometimes experiences attacks that are more frequent by night than by day, and similar to those of asthma, so that this paroxysmal dyspnœa has been called "**cardiac asthma.**" This name is bad. Mitral disease does not produce true asthma; the attack has not the characters of the dyspnœa seen in asthma. Let us analyze such an attack in a cardiac case. The breathing is rapid and panting, inspiration and expiration are short and jerky, palpitation is frequent, the pulse is small, the face is pale, and the lips are cyanosed. In asthma the picture is quite different: the breathing is not accelerated, inspiration is slow and painful, while expiration is whistling, spasmodic, and three or four times as long as inspiration; palpitation is absent, and the pulse preserves its normal rate. In some cardiac cases the attacks of dyspnœa form the chief symptom, and the only sign of mitral disease may at first be attacks of cardiac dyspnœa, just as in certain cases of Bright's disease the renal lesion is heralded by attacks of uræmic dyspnœa.

The mitral lesion also shows itself by such symptoms as palpitations and feeling of weight or of constriction in the precordial region. These symptoms are especially increased by strain or by active movements.

**Hæmorrhage from the lung** (known under the faulty term of **pulmonary apoplexy**) and **hæmoptysis**, which accompanies it, are more common in mitral than in other lesions of the heart. These hæmorrhages may be due to the mechanical obstruction of the pulmonary circulation, but are in most cases the result of small emboli, which arise from clots in the right auricle, are arrested in the pulmonary arterioles, and, like all capillary emboli, produce a hæmorrhagic infarct. These infarcts, described under **Pulmonary Embolism**, are frequently followed by hæmoptysis. Hæmoptysis may be seen at different periods of mitral disease, more usually at a somewhat advanced one. According to the classical description, it appears in the form of blood-stained sputum of a blackish colour and alliaceous odour (Guéneau de Mussy), and lasts for days and weeks (Grisolle). Cardiac hæmoptysis may, however, appear before any other symptom, and through a determinant cause (fatigue, excess, or pregnancy).



Pulmonary infarcts, especially when they are cortical, cause sero-fibrinous or puriform pleurisy, that is usually insidious and more frequent on the right side.

**Peripheral œdema**, starting over the malleoli, is fairly often seen in mitral disease. At first the œdematous infiltration is absent in the morning after the night's rest, but later it becomes permanent, and may affect the thighs, the scrotum, and the trunk (anasarca). The skin of the legs and of the thighs is thickened, red, and prone to erythema, erysipelas, and gangrene, especially after acupuncture. At an advanced period of heart disease the pleura, peritoneum, and pericardium often contain fluid. Deep-seated œdema affecting the cellular tissue of the splanchnic cavities is of great importance, as we shall see later under Treatment.

**Visceral congestions** appear at different periods. I have already said that congestion of the lung, with or without pulmonary œdema, is generally the first to supervene. The obstacle to the pulmonary circulation has a double result: first, hæmatisation being incomplete, the general nutrition suffers; second, the embarrassment in the lesser circulation gradually reaches the greater circulation, the right heart grows weak, and general circulatory distress follows.

The **liver** often shows congestion (nutmeg or cardiac liver, with or without cirrhosis). The lesion shows itself by increase in the size of the organ, with acute pains in the hypochondrium, jaundice, and epistaxis. The cardiac liver is often accompanied by ascites. These lesions are most marked in cardiac patients who are also alcoholics.

**Gastro-intestinal** congestion provokes dyspeptic troubles, with indigestion, feeling of weight in the stomach, and somnolence.

Congestion of the **kidneys** is shown by scanty urine, with sediment and albumin, lesions which rarely end in Bright's disease.

Congestion of the **encephalon** and faulty oxygenation in the **bulb** produce insomnia (Peter), nocturnal delirium, maniacal excitement (Raynaud), and convulsions.

Such are the lesions and symptoms of mitral disease. They usually run a slow course, and may last many years before they endanger life. The predominance of pulmonary, hepatic, or gastric troubles masks for a time the true cause of the disease. At length the affected muscle can no longer struggle against the obstacles at the periphery. The disease is no longer limited to the heart; every organ is affected on its own account, and the entire organism is invaded. This progressive course is interrupted by periods of **asystole**. The patient, who very rarely dies suddenly (contrary to aortic insufficiency), at length reaches the final stage of cardiac cachexia.

**Cerebral embolism** may supervene at any period of mitral disease. It

is more common in stenosis than in insufficiency, and produces troubles, such as cerebral softening, right hemiplegia, aphasia, etc., which will be discussed under Cerebral Embolism. In some cases the embolus follows another course—obliterates the peripheral arteries of the limbs and may produce gangrene.

The preceding description refers to mitral disease as a whole—that is to say, the double lesion of insufficiency and stenosis. The functional troubles of mitral insufficiency in some respects resemble those of stenosis. Auscultation, therefore, must differentiate between these two lesions.

We find, however, a **variety of pure mitral stenosis**, which merits a separate description. It is much more frequent in women than men, and is seen in young girls and young women. It is independent of rheumatism and of the other causes which usually produce endocarditis. This stenosis is sometimes associated with chlorosis, and has also been looked upon as a lesion of evolution—*i.e.*, an aplasia—analogueous to the stenosis of the aorta and the arteries which, according to some authors, is said to be the anatomical characteristic of chlorosis. Potain, in fifty-five cases of pure mitral stenosis, with autopsies, has found pulmonary tuberculosis in nine of them.

Fibrous endocarditis, which in tubercular patients goes on to mitral stenosis, is said by Teissier to be due, not to a bacillary infection, but to a tubercular intoxication. “This fibromatous process, resulting from the phthisiogenic intoxication, may be present in hereditary cases of tuberculosis, and show itself by a like evolutionary tendency. Mild local tuberculosis (pulmonary, glandular, or osseous), as well as the attenuated hereditary forms (chlorosis, lymphatism, congenital debility), may be the actual factor or the hereditary cause of a pure mitral stenosis.”

Whatever may be its origin and its pathogenesis, this constriction causes such slight functional troubles that it may be latent for years ; but it is none the less serious, for it is frequently accompanied by cerebral embolism, with hemiplegia and aphasia.

**Examination of the Heart and Pulse.**—In mitral disease the apex is displaced, because of the cardiac hypertrophy, and the beat is in the sixth or seventh space, external to the nipple. The **precordial dullness** and **bulging** are less extensive in mitral than in aortic lesions, because the hypertrophy of the right heart is less considerable than that of the left heart. If the hand is applied over the heart, we often feel a systolic thrill. The **pulse is small**, because the mitral lesion diminishes the column of the blood-wave driven into the aorta. It is **unequal** and **irregular**, because the contractions of the left ventricle are not of equal strength and do not follow at equal intervals consecutively to the muscular change in the heart (Stokes), and perhaps also consecutively to the disturbance of the



cardiac innervation (Peter). These pathological characters of the pulse are revealed by the **sphygmograph**.

The cardiac **arrhythmia**, perceptible on palpation and auscultation (G. Sée), is therefore characterized by irregular beats. This arrhythmia only shows itself at an advanced period of the disease; it is sometimes excessive and is a kind of ataxia of the heart. **Intermittences** have been divided into true and false. True intermittence affects the pulse and the heart at the same time, the arterial pulsation being absent, because the ventricular systole is lacking. The so-called false intermittence would be better named "incomplete." The cardiac systole persists, but is too feeble to launch a sufficient blood-wave, and the arterial pulsation is wanting. Bouillaud has called this a stumble on the part of the heart. These intermittences are more marked in insufficiency than in stenosis, because part of the blood-wave passes back into the auricle instead of the whole wave entering the aorta.

Mitral **insufficiency** shows itself by a somewhat rasping systolic murmur, which may be of a musical character and has its maximum below and external to the nipple. It is called the "apical murmur," in opposition to the basal murmurs, which are situated at the aortic orifice. Further, the maximum is not exactly at the apex of the heart, but rather at the middle part of the ventricle and at the level of the valvular vein (Peter). This murmur is sometimes soft and analogous to a bellows murmur; at other times harsh and vibrating, like a jet of steam. It is propagated towards the axilla, and is very clear behind, between the scapulæ.

Mitral **stenosis** shows itself by various signs, which may be isolated or associated in the same patient. When the signs are complete, and the patient has not arrhythmia, we find a special rhythm of morbid sounds. Analysis shows that mitral stenosis may be characterized by a diastolic murmur (rare), by a rumbling or presystolic murmur, or by a reduplication of the second sound, while the second portion of this reduplication may be blowing.

Mitral lesions are often accompanied by accentuation of the second pulmonary sound, from the exaggerated distension which the pulmonary vessels undergo. The coexistence of insufficiency and stenosis is shown by the simultaneous presence of the signs indicated under each of these lesions.

Sometimes, on auscultation of a patient with symptoms of mitral disease, no morbid sound is heard, and the physical signs of the lesion only reveal themselves by a disturbance of the cardiac rhythm. It must be admitted in such a case that **myocarditis is the chief lesion**, and that the valvular mischief is of but slight moment.

**Diagnosis—Prognosis.**—I repeat here that the presence of a murmur or of a reduplication is **not enough** to prove the existence of a mitral lesion.

Some reduplications have nothing to do with the lesion of an orifice. Such are the normal reduplications "resulting from transitory changes which the movements of respiration cause in the pressure of the blood contained in the heart and great vessels" (Potain). Certain murmurs do not depend upon any valvular lesion. Such are the murmurs of chloro-anæmic origin, the febrile murmurs, and the blowing sounds of extracardiac origin, described by Potain. It has been said that the murmur of mitral insufficiency is distinguished from other non-organic murmurs in that it alone is heard behind between the inner border of the scapula and the dorsal spine. This is true with regard to the mitral murmur of a certain intensity, but this distinction loses its value when the murmur is slight.

The form of **mitral disease** indicates fairly the gravity of the **prognosis**. Pure insufficiency or constriction is less serious than if the two lesions be associated.

Intercurrent diseases, such as typhoid fever, eruptive fevers, influenza, and pneumonia, may have an evil influence on the mitral lesion, since they may sometimes **graft terrible secondary infections** upon it. I would repeat here that infective endocarditis usually supervenes in patients whose previously affected valves present a **locus minoris resistentiæ**. Excess, fatigue, worry, and pregnancy have a deplorable effect upon diseases of the heart. It is opportune to recall Peter's aphorism relative to women suffering from cardiac lesions: "Daughters, no marriage; wives, no pregnancies; mothers, no nursing."

**Treatment.**—The treatment of valvular lesions in general and of mitral lesions in particular is not confined to the lesion of the orifice or of the valves, but concerns the resulting complications and also the concomitant changes in the myocardium. In this section I am dealing only with the treatment of "diseases of the heart" properly so called; the treatment of cardio-aortic lesions will be given later.

We have just seen that the symptoms consecutive to the mitral lesion only appear when the cardiac muscle is worn out. As long as the muscle is capable of facing the danger and the compensation is sufficient, all is well, or nearly so; but when compensation becomes feeble, complications which in nearly all cases have a peripheral origin—*i.e.*, visceral congestion and oedema—then appear.

Imperfectly compensated valvular lesions lower the pressure in the cardio-arterial system, and raise it in the venous system of the lesser and greater circulations. Venous stases and oedema then appear. The stases affect the lungs, kidneys, brain, liver, etc.; the oedema affects the cellular tissue of the periphery and of the splanchnic cavities, the organs susceptible of becoming infiltrated (lung, brain), and the serous cavities (pleura, pericardium, and meninges). The tendency of diseases of the heart is therefore



to change the patient into a kind of sponge ; his organs are saturated with badly circulating blood, while his connective tissue is saturated with serous fluid. In such a medium the anatomical elements and the organs partially lose their function.

Whether the disease shows itself by slight symptoms, such as trifling dyspnœa, malleolar œdema, and palpitations ; whether it betrays itself by more serious complications, such as orthopnœa, hepatic troubles, oliguria, anasarca, and arrhythmia ; or whether it finally produces the symptom-complex of asystole, the therapeutic indications are the same. It remains to be seen what drugs are most apt to answer to these therapeutic indications.

Most authorities who have discussed this question say : Since the troubles in diseases of the heart arise from weakening of the heart muscle and from lowering of the arterial tension, restore the tone and the contractile power of the heart muscle, and at the same time you will raise the arterial pressure. For this purpose employ cardiac tonics. Further, as the lowering of the arterial pressure brings about the elevation of the venous tension, with œdema and congestion, lower this venous tension, and for this purpose employ blood-letting, purgatives, and diuretics.

A table of these tonics and diuretic medicines has been drawn up. Sée has published several excellent papers on the question of cardiac drugs and of the therapeutic physiology of the heart, and I borrow the following classification from his writings :

**Cardiac tonics :** Strophanthus, strophanthin, spartein.

**Diuretic cardiac tonics :** Digitalis, digitalin, *Convallaria majalis*, convallamarin.

**Diuretic drugs :** Caffein, theobromin, potash salts, lactose.

**Respiratory drugs :** Iodide of potash, morphia.

Our patient may be a prey to arrhythmia, dyspnœa, and œdema ; he may be in a condition bordering on asystole or be suffering from marked asystole. What medicine is to be given, and what indications are to be followed ? Is strophanthin superior to spartein ? is spartein superior to digitalis ? and is not the latter inferior to digitalin ? Is theobromin superior to caffein, and caffein superior to convallamarin ? I think that many physicians will hesitate, and I have experienced myself the doubts they will experience in making a choice. Such cardiac tonics as strophanthin and spartein had at one time some vogue, in Germany especially, so that it might truly be said that, prior to the discovery of these drugs, the therapeutics of diseases of the heart were in their infancy. Caffein and theobromin are so vaunted by some clinicians that it might be said that before the discovery of these drugs we could not cause diuresis in a cardiac patient. I have closely followed the action of these drugs, and I have succeeded in forming a clear opinion as to their worth. As far as

I am concerned, I consider that the famous cardiac tonics are often medicines which **weaken** the contractile power of the muscle under the pretext of improving its tone. I have also found that theobromin, which is a valuable diuretic when properly handled, sometimes weakens the renal function under the pretext of exciting it. I am very far from contesting the worth of these drugs, but I think that most of them are far from possessing in asystole the value of digitalis, when it is well prepared and properly administered. Further, as I have neither the intention nor the pretension of undertaking here a comparative study of all the cardiac drugs, I shall simply give the results of my experience, and point out the treatment which I have employed for several years.

Let us take first the most serious case—namely, a patient suffering from symptoms bordering on asystole or from complete asystole. The face is pale and covered with sweat; the conjunctivæ are yellowish; the lips and ears are bluish; the hands are swollen and cold, and the fingers cyanosed; the pulse is small and irregular; œdema has invaded the feet, legs, thighs, genital organs, and abdomen, and tends to spread higher; the liver is enlarged and very painful; respiration is panting, interrupted by terrible attacks of suffocation, and asphyxia appears imminent. The patient, seated in his chair or resting on his pillows, dare not move for fear of suffocation. For a month he has not gone to bed, and sleep is impossible, save for snatches. The urine is scanty and contains much sediment. Auscultation of the heart is impossible; auscultation of the lungs, though difficult, yet permits recognition of râles, due to œdema and pulmonary congestion. Such is the disease: what is the remedy?

I think we must consider both the heart and the periphery. It is universally said that the condition is due to degeneration of the muscular fibre, which must be given fresh energy by means of the cardiac tonics. Letulle has shown that the muscular degeneration is not so great. If the heart is weakened, this degeneration plays only a small part. I am convinced that the most important part comes from the peripheral obstacles, from the blood which engorges the organs, from the serous fluid which impregnates the superficial and the splanchnic cellular tissue.

In the presence of these obstacles the heart becomes weakened in pumping on the blood-wave, which advances with difficulty. The arterioles fail to help it, the local circulations are at a standstill, and all the functions of the economy are in distress. I have often made the following comparison: When a cart is too heavily loaded and can advance no farther, the team will not move by whipping the tired horses, but it will do so if we lighten the load. Similarly: in the case of the heart the desired effect will not be obtained by excessive stimulation of its already weakened muscle; we shall obtain the result by lightening its work, by diminishing the excess of venous



tension, and by removing, as far as possible, the dam produced by congestion and oedema, and, above all, by improving the tone of the cardiac muscle, **which is often rather exhausted than degenerated.**

To obtain these results I employ in a fairly systematic fashion the following treatment :

Six leeches are applied to the precordial and hepatic regions, and some 5 or 6 ounces of blood are withdrawn. I cannot too strongly recommend the use of leeches, which is far too much neglected. I sometimes prescribe two or three leeches over the heart and over the liver, and I repeat this treatment several days in succession, with great benefit to the patient.

At the same time I prescribe digitalis in the form of infusion or of Trousseau's diuretic wine. The drug has a direct cardio-vascular action and an indirect diuretic action. It stimulates the tone of the heart and the vasoconstriction of the peripheral arterioles.

This combined action restores to the normal the inverted current of the interstitial exchanges. The arterioles and the heart muscle combine their energies to bring about a squeezing action on the parenchymata and the connective spaces. Digitalis, by its action on the heart and the vessels, restores to the cardio-vascular mechanism the functional energy which was lacking, and as Potain says : "It is also an indirect diuretic, which causes the fluid of dropsy and of oedema to re-enter the circulation and be eliminated by the kidneys."

The immediate result, as Loeper has shown, is a serous plethora of the blood, characterized by diminution in the total of albumin and in the red corpuscles, which are widely scattered through an increased quantity of fluid, and by increase of the chlorides and other principles which have accumulated in the tissues. The above is the first effect of digitalis, and is called the blood stage (Loeper) ; the urinary stage at once follows, and polyuria results. The polyuria varies directly with the amount of oedema due to asystole ; it often assumes the guise of a crisis which carries off in the urine the excess of chlorides, phosphates, and sulphates in the tissues. Improvement then follows. Digitalis alone, without other drugs, may yield the good results just mentioned. I feel, however, that it is preferable to add a true diuretic which acts on the kidney, while the digitalis acts upon the heart and the vessels.

Trousseau's diuretic wine is a diuretic which does not exhaust the kidney and a cardiac tonic which does not exhaust the heart. The formula is :

|                        |    |    |    |           |
|------------------------|----|----|----|-----------|
| White wine             | .. | .. | .. | 7 pints   |
| Alcohol (90 per cent.) | .. | .. | .. | 17 ounces |
| Juniper berries        | .. | .. | .. | 12 "      |
| Acetate of potash      | .. | .. | .. | 7 "       |
| Digitalis leaves       | .. | .. | .. | 2 "       |
| Squill                 | .. | .. | .. | 1 "       |

I give 1 or 2 tablespoonfuls daily for five or six days. One tablespoonful contains 3 grains of digitalis, 10 grains of acetate of potash, 15 grains of juniper berries, and  $1\frac{1}{2}$  grains of squill.

We must note that Trousseau's formula has been altered, and that the wine in the Codex and hospital formulæ contains 10 instead of 3 grains of digitalis in a tablespoonful. A physician ignorant of this incomprehensible change would thus give his patient a far larger dose than he intended. I, therefore, ask for a return to the classical formula, which should never have been tampered with.

For the diuretic wine we may substitute infusion of digitalis and theobromin given together.

Make an infusion of digitalis leaves, 15 grains in  $3\frac{1}{2}$  ounces of boiling water, filter, and add 8 drachms of syrup of currants. Give 1 ounce daily for a week ; stop the drug, and repeat if need be. At the same time theobromin is given in daily doses of 7 grains.

It is absolutely necessary for the patient to take milk as a food and as a diuretic. He may take it to his liking : boiled or fresh, hot, cold, or iced. The milk should be given every two hours in doses of 3 or more ounces, according to the tolerance of the patient. To alter its taste we may add orange-flower water or a spoonful of tea or of coffee, and we may aerate it by aid of the sparklet. If the milk is badly digested, lime-water is added. In case of intolerance, cow's milk is stopped, and recourse made to goat's or to ass's milk, kephir, or koumiss. All other food is forbidden. Ices made with coffee, vanilla, citron, or with fruits are permitted.

To produce active diuresis, I give lactose dissolved in Évian or in Vittel water. When lactose is simply thrown into the water, the patient complains of the mixture, because the sugar does not dissolve well in cold water. The following solution must therefore be made : The lactose is first dissolved in a small quantity of hot water, and the solution poured into a bottle of Évian water, from which an equivalent quantity of fluid has previously been withdrawn. We have then a drink which, although not disagreeable to taste, may be rendered more agreeable by the addition of orange-juice, lemon-juice, or champagne. A cup of milk and a cup of lactose solution are taken alternately every one or two hours.

In short, leeches, digitalis, diuretics, and especially Trousseau's wine, are used almost **systematically** for patients who come into hospital with asystole, or in a condition bordering on it. My cases are described in the hospital records, where they may be consulted, and I may say that, under this treatment, which is simple and easy, success is the rule and failure the exception. The urine, which has been as low as 6 ounces before treatment, amounts to 2 or 3 pints after a few days.

A patient, for example, who drinks daily 4 pints of milk and 2 pints of



lactose solution—that is to say, about 6 pints of fluid—sometimes passes about 8 to 10 pints of urine. The urine exceeds, therefore, the fluid ingested by 2 to 4 pints. This excess comes from the fluid which was enmeshed in the form of œdema in the superficial and in the splanchnic cellular tissue. When care is taken to weigh the patient daily, and to measure exactly the quantity of the fluid ingested and of the urine voided, we find that the weight of the patient diminishes by about 1 pound per pint of urine excreted in excess. In a case of anasarca I have verified the fact that a patient may hold 20 to 30 pints of fluid in his cellular tissue. The attention is as a rule only evoked by superficial œdema, but there is also hidden in the splanchnic cavities œdema which escapes notice and is most serious, because of the embarrassment it causes in the different functions.

The subjoined chart shows the result in a woman who was moribund and cyanosed. The amount of urine was about 3 to 4 ounces. Under

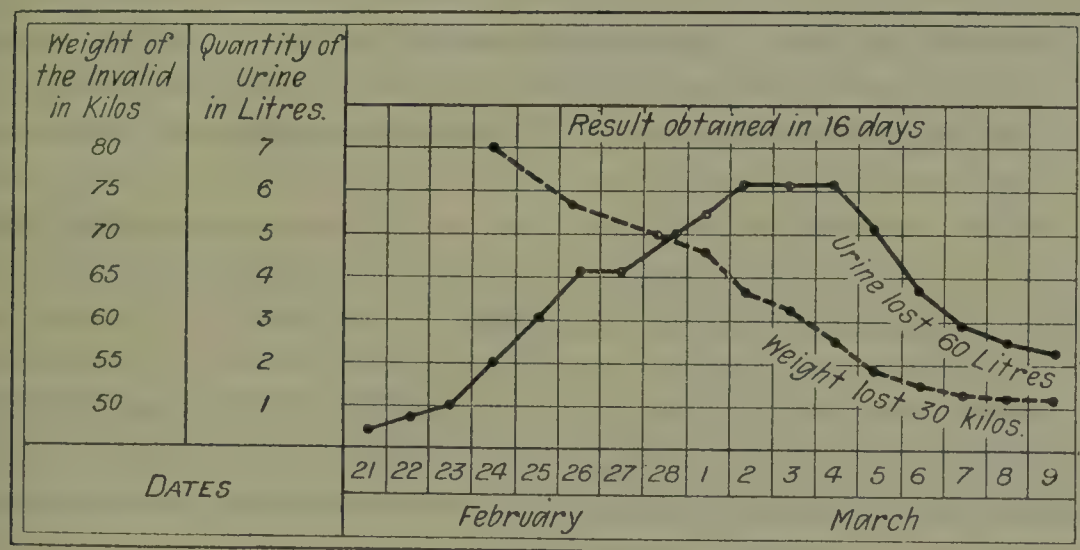


FIG. 22.—WEIGHT AND URINE CHART.

treatment the improvement was so rapid as to be a brilliant therapeutic success. In a fortnight she passed 100 pints of urine, and as the anasarca disappeared, she lost nearly 60 pounds in weight. The chlorides at the commencement of the urinary crisis amounted to 1 ounce.

By the means just quoted the œdema disappears, the visceral congestion diminishes, the dyspnœa improves, and the heart-beats become regular. In place of distress, a delightful feeling of quiet obtains, movement is possible without attacks of suffocation, the broncho-pulmonary râles tend to disappear, and the patient can lie down in bed and sleep.

In some cases, however, disappointments occur. Some patients are refractory to treatment, others vomit the milk and solution of lactose, no matter what we do. The difficulty is surmounted by giving iced water in small amounts and by enemata composed of 6 ounces of the lactose solution.

If, after some days of the above treatment, no improvement has accrued, and if the asystole becomes worse, we must lessen the work of the heart and relieve the patient's suffering by withdrawing the fluid from the pleura and the peritoneum. We must treat the œdema of the limbs and genital organs in the same way, and provide points of exit. We, therefore, make punctures, which are sometimes followed by lymphangitis, erysipelas, or patches of gangrene, in spite of antiseptic precautions, or else we apply the cautery once or twice to each limb; Vienna paste makes an issue which, once opened, acts like a fountain. In urgent cases, however, punctures are preferable.

I have not yet spoken of morphia or of heroin; not that I reject their use—on the contrary, I think that they are excellent adjuvants in severe dyspnœa, but they should be prescribed in very small doses.

I must now mention certain accidents which may occur unless the case be carefully watched. The use of digitalis or digitalin, even in small doses, must not be too long continued, because the drug may be cumulative, and lead to sudden asystole. An inexperienced or careless physician mistakes the condition for obstinate asystole, and goes on with digitalis, being lucky if he does not increase the dose. "Characteristic pallor of the face and general coldness, with cyanosis, are signs of advanced poisoning, because they indicate vasoconstriction due to the predominant action of the sympathetic nerve. Tachycardia, arrhythmia, and pulsus bigeminus are symptoms of vasoparesis; such a condition is asystole due to digitalis" (Huchard).

Another matter deserves notice. During the treatment we must carefully collect the urine passed in the twenty-four hours, and watch the parallel diminution in the weight of the patient. The reason is: Under treatment the œdema may be in part absorbed, without increase in the urine, or, in other words, the blood stage is not followed by polyuria. The serous fluid is absorbed and enters the circulation, but it is not eliminated by the kidneys in due course. Serous apoplexy may then follow, as pointed out by Andral. The symptoms are coma, convulsions, and delirium. Cheyne Stokes breathing may or may not be present. We must add that these troubles are chiefly to be feared in patients whose kidneys are already affected. In any case, we see the reason for associating with the digitalis a true diuretic in order to favour the urinary as well as the blood stage.

In addition to the troubles above noted, the patient may be seized with syncope and dyspnœa, which seem to herald early death. We may then ask whether sudden dilatation of the heart does not complicate an already dangerous situation. In this case cardiac drugs must be handled with extreme care. Perhaps it is better to omit them. It has long been my practice to place an ice-bag over the heart, and patients feel such relief that they will not dispense with it. Oxygen should be given in large doses. Elixir of maté may be given in drachm doses. Injections of oil of camphor



are useful, and strychnine may be given in the same way once or twice daily.

When the patient has got over his asystole, when the excess of chlorides has been eliminated in the urine, when the dyspnœa, anasarca, and arrhythmia are better, he must not be considered as cured, for the cardiac lesion persists, and may at a given moment reproduce the same effects. For several weeks to come he should take only milk, or milky foods, eggs, stewed fruit, and a little bread. Salt (chloride of sodium) should be proscribed, for we know that it favours the production of œdema (Widal, Merklen). Alcoholic drinks and tobacco should always be forbidden. **Local** treatment should not be neglected. It consists in an application of Vienna paste to the cardiac region, and the issue is allowed to suppurate freely.

Such is the treatment. It often dispenses with other cardiac tonics, such as strophanthin and spartein. It proves clearly that in the cardiac storm which we call asystole the true danger lies, not only in the heart, but in every organ and at the periphery, where congestion and œdema occur. The proof that the heart is worn out rather than degenerated is that, when the storm is over, it resumes its functions, and may do so sometimes for months and years before it becomes again enfeebled. We see daily such cases in hospital.

Amongst other examples I would quote the case of a moribund woman treated in this manner. In spite of her mitral lesion, she has since remained in a very satisfactory state of health. I would mention, too, the case of a blacksmith, also cured of asystole for four years. In spite of a mitral lesion, he has been able to carry on his heavy work without any fresh mishap. He reports himself at the hospital every four or five months for a fresh issue to be made, because, he says, when the counter-irritation no longer acts, he experiences slight dyspnœa and palpitation, which improve when the free issue is employed. I have thus seen a large number of asystolic patients resume their business for years.

The attentive study of all these cases has inspired me with some thoughts on the subject of prognosis in diseases of the heart. When we have seen a certain number of cardiac patients triumph once or several times over the asystolic storms, and practically resume their work, we say that the mitral disease is too often considered as a very grave malady of almost fatal termination, but it does not possess such a gloomy prognosis. The heart is a gallant organ; it is the *ultimum moriens*. We have slandered it a little, since we have been willing to assign too large a part to the lesions of its vessels and its myocardium. We must treat it carefully when it is sick, assist it when its work is too difficult, but we must avoid forcing and weakening it with cardiac tonics.

I have just discussed the treatment during asystole, but what treatment should be used when the patient only complains of dyspnœa, œdema, or of cardiac angina? In the first place, I recommend the treatment which

I have just indicated, for it succeeds the better and quicker the less pronounced the complications. If dyspnoea is the chief symptom, we may also employ iodide of potash, which is given in doses of 10 grains in the twenty-four hours.

Lastly, at the onset of the mitral disease, if there is no notable complication, and if the disease only shows itself by palpitation, subcrepitant râles from pulmonary oedema, slight dyspnoea, or some oedema over the malleoli, the physician should not remain inactive, but should foresee and prevent the more serious complications, and attempt to stop the progress of the disease. It is useful to produce in the precordial region an issue which is allowed to suppurate, and which is renewed if there is occasion.

Heavy meals, alcoholic drinks, and the use of tobacco should be forbidden. The professions which entail excitement, such as gambling and the Stock Exchange, furnish a marked proportion of cardiac cases. Peter has seen many politicians in whom the disease made rapid progress. A life free from excitement and fatigue should be advised in these cases, and conduces to longevity. I employ the above treatment in a systematic manner, but I do not mean that other treatment may not give good results. Thus, digitalis may be given as a tincture or maceration.

**Digitalin** is prescribed by Potain in an alcoholic solution (Nativelle's crystallized digitalin, or Homolle's digitalin), of which 25 drops correspond to a demimilligramme. This drug is taken at one dose in a little water, and three or four days should elapse before administering a fresh dose.

The oily solution of Nativelle's digitalin may be given by subcutaneous injection in doses of  $\frac{1}{4}$  milligramme per cubic centimetre.

**Strophanthus** is chiefly indicated as a cardiac tonic. It does not slow the heart-beats, but it raises the contractile power of the myocardium. Five to twenty drops of the tincture are given. Caffein may be given in daily doses of from 7 to 30 grains, either in solution or by hypodermic injections.

|   |                  |    |    |    |    |       |
|---|------------------|----|----|----|----|-------|
| R | Distilled water  | .. | .. | .. | .. | 3iii. |
|   | Benzoate of soda | .. | .. | .. | .. | 3i.   |
|   | Caffein          | .. | .. | .. | .. | 3i.   |

A Pravaz syringe of this solution contains 3 grains of caffein.

That excellent diuretic theobromine is given in cachets, in doses of from 7 to 15 grains. The dose is increased or repeated if necessary.



## V. AORTIC ORIFICE—INSUFFICIENCY AND STENOSIS.

**Aortic insufficiency** is present when the sigmoid valves which normally close the orifice, in order to prevent the backward flow of blood, do not sufficiently plug it to prevent the blood from flowing back into the left ventricle. **Aortic stenosis** occurs when the lumen of the orifice no longer has its normal dimensions.

The aortic orifice, from its situation, participates in lesions of the aorta more than in other lesions of the heart. Thus, inflammations of the aorta and their most usual causes, especially syphilis, are, more often than rheumatic endocarditis, the origin of lesions at the aortic orifice. According to circumstances, the lesions are sometimes those of chronic aortitis, at other times those of chronic endocarditis. Aortic insufficiency and stenosis are often combined. These lesions are due to adhesions, deformities of the sigmoid valves, vegetations, or calcareous incrustations of the valves or of the walls. **Pure** insufficiency is frequently associated with enlargement of the aorta, which, in its dilatation, pulls on the walls of the orifice.

The stenosis is not always exactly at the orifice. Subaortic narrowing (Vulpian, Peter), arising from the contraction of the portion which is just in front of the opening of the aorta, has been described.

Lesions of the aortic orifice, and especially insufficiency, cause enormous **hypertrophy** of the heart, in which the left ventricle takes so large a part (bovine heart). This hypertrophy, called providential, depends upon the hyperactivity of the heart muscle; part of the blood-wave flows back into the left ventricle, and hypertrophy takes place, because the work is exaggerated.

**Examination of the Heart and the Pulse—1. Aortic Insufficiency.**—In **aortic insufficiency** the precordial bulging and dullness are more extensive than in mitral lesions. The impulse may be diffuse and the apex-beat is in the sixth or seventh space. The **pulse** is characteristic, being regular, bounding (Corrigan) and falling (Stokes): bounding, because the blood is violently propelled by the hypertrophied ventricle; falling, because the pulse vanishes directly after the beat, the aortic blood-wave being propagated in two directions at the same time—towards the periphery and towards the ventricle. These characters are seen in the sphygmographic tracing; the line of ascent is sudden, because of the hypertrophy of the ventricle, and ends in a notch, which coincides with the return wave into the ventricle. The force of the systole communicates itself to the great arteries; their pulsations are exaggerated, and often yield a thrill. It is in aortic insufficiency that we see best the changes of colour under the nails, isochronous with the cardiac systole. This phenomenon has been called the capillary pulse.

We hear in the second right intercostal space a **diastolic murmur**, due to the propagation of a part of the blood-wave toward the left ventricle. This diastolic murmur is heard in the great vessels of the neck and limbs. Thus, when the stethoscope is applied over the femoral artery, two murmurs (double crural murmur) are heard at the point compressed—a “*souffle d’aller*,” which is not pathological, but due to the vibration of the blood-wave launched from the heart; and a **reflux murmur**, which is much softer, and due to the vibration of the blood-wave, which tends to flow backwards. In order that the reflux murmur may be produced, the artery must be firmly compressed by the stethoscope, but not too much. The second murmur is sometimes replaced by a tone which has the same value (Skoda).

The theory of flux and reflux in the arteries is hardly admitted to-day. The reflux remains absolutely true for the return of the blood-wave from the aorta into the left ventricle, but the backward return of the column of blood does not continue in the arteries. It is rather admitted that the phenomena described above result from the lowering of the arterial tension, which induces an exaggeration in the vibrations of the artery and a rapidity of the waves (Potain).

The reflux murmur and the water-hammer pulse, which are sometimes so clear in pure insufficiency, are naturally less marked if stenosis is also present, because the blood-wave no longer finds so free a passage. Auscultation at the aortic orifice also reveals the disappearance or the diminution of the second sound, which normally is due to the closure of the sigmoid valves.

**2. Aortic Stenosis.**—In **aortic stenosis** hypertrophy of the ventricle is also very pronounced, but the pulse is small, and shows in the sphygmographic tracing an inclined up-stroke, because the blood-wave passes in a threadlike stream through the constricted orifice. Auscultation in the second right space reveals a systolic murmur, which is harsh and vibrating. This murmur is propagated into the great vessels which arise from the aorta, and often extends along the descending aorta to the interscapular region.

**General Symptoms.**—These appear more slowly than those of mitral lesions, because aortic lesions are better compensated by the hypertrophy of the left ventricle, and because they have a less direct reaction upon the pulmonary and the general circulation. The patient looks anæmic, because the blood in part returns into the ventricle and does not all reach its destination. He is also affected by cerebral congestion, due to the exaggerated ventricular contractions, and shown by epistaxis, pulsations in the temporal arteries, dizziness, ringing in the ears, etc.

Some patients complain of angina pectoris, and are liable to faint. In this case the lesion of the aortic orifice is accompanied by aortitis, and



the symptoms of aortitis are associated with those due to the insufficiency and the stenosis (Peter).

The aortic lesions remain a **local malady** much longer than the mitral lesions, and it is only at a remote date (degeneration of the muscular fibre or mitral complications) that the invasion of the economy becomes general—that congestion, œdema, and the symptoms, which precede or accompany asystole, appear.

The patient may, however, be carried off suddenly before this stage. **Sudden death** is a fairly common termination in aortic lesions (Aran, Mauriac), while it is exceptional in mitral disease. Writers have attributed it to endarteritis obliterans, insufficiency of circulation in the coronary arteries (Mauriac), and angina pectoris, which is often associated with the aortic lesions (Peter).

**Treatment.**—We cannot produce much action on the lesions of the aortic orifice. The cardiac erethism should be quieted by local (leeches, cupping, ice-bags) or general treatment. The iodides are absolutely indicated. We shall see in subsequent chapters the treatment for lesions of the aortic orifice associated with aortitis or syphilis.

## VI. THE TRICUSPID ORIFICE—INSUFFICIENCY AND STENOSIS.

**Ætiology.**—Inflammatory changes in the tricuspid valve are somewhat rare, except in foetal life. They nearly always affect the valves on both sides of the heart. Duroziez, however, maintains that they are less rare than is generally believed. The fibrous and atheromatous lesions of chronic endocarditis do not often attack the right heart. On the other hand, tricuspid insufficiency is often the result of a mechanical cause—namely, dilatation of the right ventricle. By reason of the exaggerated blood-pressure which occurs in chronic diseases of the lung, or in mitral lesions, the right ventricle allows itself to be distended. It also becomes distended through dystrophy of its muscular fibres, with or without adhesions to the pericardium and the pleura, and drags with it the insertion zone of the tricuspid valve, which becomes incompetent. Some authorities, on the other hand (Potain and Rendu), refer this insufficiency to the increase in the capacity of the right ventricle. The direction of the ventricular pillars is altered; the chordæ tendineæ are not long enough to permit the closure of the valves, and functional insufficiency results. Dilatation of the fibrous ring, correctly speaking, is quite exceptional.

**Reflex spasm of the pulmonary vessels** (Potain and Franck) arising from gastro-hepatic affections leads to dilatation of the right ventricle and functional insufficiency of the tricuspid valve. Lastly, acute and chronic

myocarditis and cardiac degenerations may end in like manner, by favouring paresis of the right ventricle.

**Pathological Anatomy.**—In cases of inflammatory insufficiency the lesions are analogous to those of the mitral valves, and do not therefore require further details. In functional incompetence the valves are healthy, but the size of the right auriculo-ventricular orifice and ventricle is much increased. The ventricular walls are rather dilated than hypertrophied in insufficiency of inflammatory origin. The right auricle is dilated, and its walls are thinned. The venæ cavæ and the jugular veins are always much distended with blood. As regards the chief viscera, such as the liver and kidney, more or less marked signs of blood-stasis are present.

**Description.**—**Tricuspid insufficiency** is characterized by a systolic murmur, with its maximum at the xiphoid cartilage, while its tone is deeper and less whistling than that of mitral incompetence. As the tricuspid valve is the “regulator of the venous circulation and of the general venous tension” (Raynoud), we must look for the chief symptoms of this lesion in the venous system.

Jugular pulsation first attracts attention. On inspection and with the sphygmograph, the external jugular vein shows an expansion and retraction, which is known as the **venous pulse**, and is explained in the following manner: During the systole, part of the blood-wave flows back into the right ventricle, and thence into the venous system. This pressure causes dilatation of the veins, and the valves of the external jugular veins become incompetent. Accordingly, at each ventricular systole the blood flows back into the venous system, and the jugular veins show a **true systolic** and sometimes **dicrotic venous pulse**, synchronous with the double auricular and ventricular systole.

The reflux of blood in the inferior vena cava and the portal vein causes **hepatic pulsations**, which may be felt on palpation.

The venous pulse may be present in the veins of the periphery, especially in the saphenous veins, when they are varicose.

We know the gravity of tricuspid lesions, for “they form the bond of union between lesions of the lesser and the greater circulation” (Peter). Tricuspid insufficiency opens the door to asystole (Raynaud); so that the murmur has been rightly called the symptomatic murmur of asystole (Parrot).

When equilibrium has been restored by appropriate treatment, the asystole disappears temporarily or finally, and with it the functional insufficiency. The latter may appear and disappear several times before being finally established. The disappearance and reappearance of the characteristic signs will serve as a guide in this respect.

In some cases tricuspid insufficiency is certainly beneficial. It may



eliminate or diminish the excess of pressure in the lesser circulation, and thus prevent vascular ruptures. Indeed, the general condition sometimes improves on the appearance of insufficiency. Hence the tricuspid valve has been called the safety-valve of the pulmonary circulation.

**Stenosis.**—**Tricuspid stenosis** may be congenital or acquired.

Congenital stenosis is due to foetal endocarditis or to faults of development.

Acquired stenosis is due to the adhesion of the valves, to stenosis of the tricuspid orifice, or to obstruction of this orifice by vegetations. Adhesions between the valves is the most usual cause, both before and after birth (Leudet). Perforation of the septa and stenosis of the pulmonary artery are frequent complications.

Acquired stenosis is chiefly seen in women. It is usually due to rheumatism, and nearly always coincides with stenosis of the mitral or other orifice. The chief symptoms are cyanosis, ascites, oedema, and curving of the nails. The venous pulse in the jugular veins is often absent, and a diastolic shock, with or without thrill, is found.

The prognosis is very grave, and death usually occurs between the ages of twenty and twenty-five years. The diagnosis is possible when stenosis of the tricuspid orifice occurs without lesions of other orifices.

## VII. PULMONARY ARTERY—STENOSIS AND INSUFFICIENCY.

**Stenosis** of the pulmonary artery may be congenital or acquired.

**Congenital** stenosis usually involves the sigmoid valves, which are adherent and enclose a more or less narrow cleft. As they are in close apposition, they obstruct the reflux of blood into the right ventricle. Hence insufficiency associated with congenital stenosis is rare. In some cases the stenosis affects the infundibulum, and has been called "**prearterial**" (C. Paul). The valves are often affected in such cases. The stenosis may involve the trunk of the artery in exceptional cases. **Acquired** stenosis, which is more frequent than is supposed, nearly always affects the valves, but cases of atheroma and of gummata (Schwalbe) affecting the pulmonary artery and causing constriction have been published. Apart from such exceptional cases, acquired stenosis of the valves often accompanies dilatation of the pulmonary artery beyond the obstruction, thus forming an exact opposite to the condition in aortic stenosis. Arterial dilatation is not seen in cases of congenital stenosis.

The right ventricle is always hypertrophied and dilated. In congenital cases we often see perforation of the interventricular or interauricular septum, and sometimes persistence of the ductus arteriosus. This perforation may also be seen in cases of acquired stenosis (C. Paul) resulting from myocarditis, which often accompanies endocarditis of the right heart.

Congenital stenosis is certainly of inflammatory origin, but the cause of foetal endomyocarditis is generally obscure. In adults rheumatism, perhaps also traumatism, and especially infectious diseases, such as the eruptive fevers, puerperal conditions, broncho-pneumonia, etc., have been held responsible.

Stenosis of the pulmonary artery may long pass unnoticed. In other cases the patient soon becomes breathless, and liable to frequent fits of coughing, which may be accompanied by bloody sputum. Chilliness of the extremities and sensations of numbness and anæsthesia in the limbs are common. Cyanosis is much less constant, and its mechanism has received a different interpretation (see **Morbus Cæruleus**). It often appears at an advanced period of the disease, or as the result of some broncho-pulmonary complication.

On auscultation, stenosis of the pulmonary artery is characterized by a systolic murmur, which is loudest in the second intercostal space, and can be traced towards the clavicle. Palpation sometimes reveals the existence of a systolic thrill at the same spot, and percussion shows more or less hypertrophy of the right ventricle.

The sufferer rarely reaches adult age. Death frequently results from progressive asystole or from syncope, but the striking fact is that pulmonary stenosis appears to favour the development of **phthisis** in some cases.

**Insufficiency of the pulmonary artery** rarely exists alone, especially when it is congenital. It is nearly always associated with stenosis. In the acquired form it results from the same causes as stenosis.

It is characterized by a diastolic murmur, which is loudest in the second left space, and can be traced along the left border of the sternum. Functional troubles, chiefly in the lesser circulation, are present, and comprise dyspnoea and frequent hæmoptysis. It is often accompanied by hypertrophy and dilatation of the right ventricle, and ends in death from asystole. On the other hand, the development of tubercles in the lung is exceptional when there is no coexistent stenosis.

### VIII. MORBUS CÆRULEUS—CYANOSIS.

This disease, which is most common in early life, is characterized by a bluish colour of the skin and the mucous membranes, with dyspnoea, attacks of suffocation, torpor, inertia, and tendency to chilliness. These symptoms result from cardiac and vascular lesions, which bring about the mixture of the arterial and venous blood, and **do not allow the venous blood to be sufficiently arterialized.**

**Pathology.**—The mixture of arterial and venous blood results from abnormal communication between the arterial and venous systems, and the abnormalities which make this communication possible affect the heart and



great vessels. The **abnormalities of the heart** are represented by the persistence of Botallo's foramen (52 out of 69 cases, Gintrac), by communication between the two ventricles (33 cases, Guillon), and by fusion of the cardiac cavities, so that the heart may show only one, two, or three cavities. The **vascular abnormalities** comprise abnormal origin of the vessels and persistence of the ductus arteriosus (30 cases, Almagro). Most of these malformations are congenital, and apparently due to arrested development of the heart, to endocarditis, and especially to **stenosis of the pulmonary artery** arising during intra-uterine life.

Raynaud says of this mechanism: "When pulmonary stenosis is present, the consecutive changes in the heart are perfectly intelligible. The right ventricle hypertrophies, because the obstruction demands increased work, but the dilatation is slight or absent, because the imperfect septum allows blood to flow back into the left cavities. The formation of the septum becomes arrested as a result of the reflux in question. If the lesion has arisen before the development of the ventricular septum—that is to say, before the end of the second month—the two ventricles continue to communicate with one another, and the communication is the larger, as the pulmonary artery remains closed at a time more closely approaching that of conception. If, on the other hand, the ventricles are already shut off from one another when obliteration of the pulmonary artery takes place, the flow of blood from the right auricle into the corresponding ventricle is prevented, a powerful current is established from the right to the left side of the auricular chamber, and Botallo's foramen remains open. As the aorta, however, is from this time the only channel open to the blood-stream, the blood can only reach the lungs through the ductus arteriosus, which therefore persists after birth as a permeable tube."

These lesions, however, although they allow, more or less, the mingling of arterial and venous blood, do not always cause symptoms of morbus cæruleus. Indeed, there are many examples (Gelau, Longhurst) showing that excellent health and absence of cyanosis have for many years been compatible with the mixing of the arterial and venous blood. Another factor is therefore required to produce morbus cæruleus, and the lesions most favourable to this result are those which do not allow the mixed blood to be properly oxygenated in the lung.

Examination of the blood has shown an increase in the number of red corpuscles (**hyperglobulia**), which may reach 8,000,000 or more. This hyperglobulia is often accompanied by an increase in the diameter of the red corpuscles (Vaquez). These phenomena have been compared to the hyperglobulia of high altitudes, and have also been looked on as a means of defence on the part of the organism, which seeks to obtain compensation for the difficulty in oxygenation of the blood.

**Description.**—The **bluish coloration** is most marked in the lips, the nostrils, the lobes of the ears, the ends of the fingers and toes, and the mucous membrane of the tongue and pharynx. The cyanosis is not always of the same intensity, and diminishes during sleep or after a long rest, while slight effort causes it to return. The fingers, in addition to the violet tint, show marked deformity, the last phalanx being swollen and rounded, while the nails are thick, broad, and curved.

**Respiration** is short and painful, the voice is shrill and jerky, and the patient cannot take exercise without feeling short of breath and suffering from palpitation, angina, and syncope. He is conscious of the lowering of his temperature (95° F., Tupper), and the slowness of his movements somewhat resembles those of cold-blooded animals.

The cyanosis, dyspnoea, somnolence, apathy, and coldness are explained by the fact that the blood contains too much carbonic acid and not sufficient oxygen. The duration of life depends upon the nature and gravity of the lesion. Some patients die from asphyxia or syncope, while others become tubercular.

Examination of the heart gives uncertain information as to the precise site of the lesion. The area of dullness varies with the hypertrophy of the ventricle, and palpation sometimes reveals a continuous thrill with reinforcement.

The murmurs heard on auscultation vary. Perhaps the systolic murmur, with its maximum at the fourth dorsal vertebra, indicates **the persistence of a ductus arteriosus**.



## CHAPTER III

### DISEASES OF THE MYOCARDIUM

**Myocarditis**, or **carditis**, is inflammation of the heart. Virchow described two varieties: **parenchymatous**, or inflammation of the muscle; and **interstitial**, or inflammation of the connective tissue.

The former is usually found in **acute** cases, while the latter obtains in most of the **chronic** cases. At the present day the general term of **fibrosis of the heart** is more often used.

#### I. ACUTE MYOCARDITIS—FATTY DEGENERATION OF THE HEART.

**Ætiology.**—Acute primary myocarditis is very rare (chill, injury). The causes of the secondary form are: (1) Rheumatism. The heart alone may be affected, or may be involved with the pericardium and endocardium. (2) Infectious diseases. Amongst these we must quote, as of primary importance, typhoid fever, variola, scarlatina, diphtheria, erysipelas, puerperal fever, purulent infection, and ulcerative endocarditis. Alcoholism, syphilis, overwork, and previous cardiac lesions, also favour its development.

In certain infectious diseases, such as diphtheria, the pathogenic agent does not enter the blood, and yet acute myocarditis is fairly frequent. In these cases toxins appear to play the chief part, just as in acute myocarditis, experimentally produced by injection of a filtered culture of the *Bacillus pyocyaneus* into the veins.

In other affections, however, such as typhoid fever, the pathogenic microbe has been found between the inflamed muscular fibres, and it may then be asked whether the micro-organism acts directly on the myocardium, or secondarily by its secretory products. The latter explanation is more generally accepted. In all these cases a certain rôle must be assigned to waste products which have accumulated in the blood, and, indeed, they alone (scurvy, experimental overwork) may be capable of affecting the vitality of the heart muscle.

**Pathological Anatomy.**—The heart is generally enlarged, and its walls are flabby and pale. On section the dead-leaf tint of the muscular tissue

is very clear. The tissue tears easily, explaining the ruptures of the musculi papillares and the intracardiac hæmorrhages. Under the microscope the muscular bundles show more or less advanced changes: the striation is less clear than usual, and we find in the fibre fine fatty granules, which mask its structure. Vitreous or amyloid degeneration may be seen. The nuclei are swollen and sometimes increased in number. Metchnikoff, however, says that this nuclear multiplication is only apparent, and the increase is simply the result of the penetration of phagocytes into the inflamed fibres.

In some cases the fibres are separated, and their anastomoses broken, as though the cement had been dissolved. The arterioles present clear traces of endarteritis and periarteritis.

The question of inflammation or of degeneration has been much discussed. Virchow, Zenker, and Hayem regard the condition as a parenchymatous inflammation. Cohnheim, Cornil and Ranvier, and Rindfleisch, who base their opinion on the almost constant integrity of the interstitial tissue, hold that the lesions are degenerative. Hanot has cleared up the question by showing that the pathogenic agents may affect both the muscular and the connective tissue. Each element reacts in its own way, but in both cases the condition is due to an inflammatory process.

**Suppurative** myocarditis is rarer, and is chiefly seen in purulent infection and puerperal conditions. The pus may infiltrate the tissue, but is more often collected in the form of an abscess. The abscess may open into the pericardium or into the ventricles, and cause embolisms in the lesser or greater circulation. It may undergo caseous change and favour the formation of an aneurysm.

**Description.**—Acute myocarditis is not a self-evident affection, and its diagnosis demands careful search. Since it nearly always supervenes as a complication of some other disease, it often passes unnoticed. Weakness and irregularity of the heart-beats and of the pulse are the general signs. We also find disappearance of the first sound, while the second sound persists, but is muffled and distant. The second sound may, indeed, be replaced by a slight rustling. When the myocarditis is less severe, the first sound may be replaced by a soft murmur, which is limited to the apex, and shows a marked tendency to alter from one moment to another.

Under the same conditions we sometimes hear a “bruit de galop,” due to a diastolic shock, indicating the ventricular dilatation which accompanies the change in the myocardium. Arrhythmia, which is often present, has been divided into regular and irregular. In the former the cardiac contraction exists, but is so weak that it cannot be recognized either by auscultation or by examination of the pulse. Sphygmographic tracings alone show that the arrhythmia is only apparent, and that an abortive pulsation really corresponds to the interval of silence. Lastly, the cardiac rhythm may



resemble that of the foetal heart, the condition being called **embryocardia**. The prognosis in these cases is very bad.

Acute myocarditis is generally painless, although Peter has remarked that pain is caused by percussion of the third and fourth intercostal spaces, and radiates along the course of the phrenic nerve and the cardiac plexus. In some cases of typhoid fever painful crises resembling those of angina pectoris have also been observed.

In conjunction with the troubles affecting the power and rhythm of the heart-beats, we may note in some patients tachycardia, cyanosis, œdema, and chilliness of the extremities—in short, all the signs of **algid collapse**; in others the phenomena of acute asystole predominate (cardiac form of typhoid fever). Lastly, we may see repeated attacks of syncope, which at times prove fatal. In typhoid fever in particular, some authorities, of whom I am not one, have attributed sudden death to acute myocarditis. This question will be treated in detail under Typhoid Fever.

When recovery follows, Landouzy and Siredey think that the myocardium may preserve some relics of the profound change which it has undergone, and that this change may ultimately assist in the development of chronic mischief.

Counter-irritation to the precordial region and cardiac tonics, such as digitalis and caffein, are the measures generally employed for feeble action of the myocardium, but I would only recommend the use of these drugs with reserve. Degeneration is often irregular, especially when it is dependent upon a change in the nutrient vessels of the heart. In these cases the wall of the ventricle, especially at the apex, is often thinned.

The symptoms of acute fatty degeneration of the heart resemble those of parenchymatous myocarditis. As regards chronic fatty degeneration, the symptomatology is practically that of fibrosis of the heart, with this difference, however—that the phenomena of cardiac excitation are wanting. At first the striking fact is the weakness of the heart-beats, the increase in the size of the organ, showing its dilatation, and lastly, the phenomena of peripheral and visceral stasis. Accordingly, after some time **asystole** follows unless sudden rupture of the heart causes death.

**Fatty Degeneration.**—Fatty degeneration of the heart has more in common with acute myocarditis. Ranvier, indeed, denies the latter disease, which would therefore be characterized solely by a fatty change in the muscles of the heart.

Many conditions give rise to fatty degeneration. (1) Certain poisons (phosphorus, arsenic, etc.) cause complete degeneration in a few days (acute degeneration); (2) alcoholism, lesions of the heart and of the valves which impede the circulation in the coronary arteries, gout, and old age cause chronic degeneration.

In the second group we must also place cachectic conditions, resulting from chronic diseases, deep-seated suppuration, intense anæmia of long duration due to repeated loss of blood, chronic diarrhœa, and athrepsia.

It is, however, important to distinguish between fatty degeneration and **fatty overgrowth**. The latter chiefly occurs in obese patients. It may exist alone for a long while before the malnutrition of the heart, due to accumulation of fat on its surface and between its muscular bundles, causes changes.

Some causes (alcoholism), however, may produce simultaneous overgrowth and degeneration.

The macroscopic appearances are the same as in parenchymatous myocarditis, but the microscope shows that the nuclei of the muscular fibres are intact, while the interior of the fibres is replaced by droplets of fat, which may be large, and which have taken the place of the sarcolemma. Frequently, also, a certain degree of granulo-pigmentary degeneration is seen. The coronary vessels, especially in chronic affections of the heart, are thickened, and their lumen is constricted or obliterated, proving the permanent nature of the obstacle opposed to the nutrition of the myocardium.

## II. FIBROSIS OF THE HEART.

Fibrosis of the heart, or inflammation of the connective tissue, corresponds in part to the old **interstitial myocarditis** of Virchow.

It may be circumscribed or diffuse. When circumscribed it commences around foreign bodies in the walls of the heart—*e.g.*, hydatid vesicles or syphilitic gummata.

It is also seen in areas of pericarditis or of endocarditis. In adherent pericardium the fibrosis may extend deeply and cause **pericardogenous myocarditis** (Brouardel, Poulain). **Interstitial endocardogenous myocarditis** is much rarer. In children, however, we see constriction of the infundibulum due to this origin.

The **diffuse form** constitutes fibrosis of the heart properly speaking. It has lately been the object of important work, which has profoundly modified our conceptions of the diseases of the myocardium. It nearly always begins with arterio-sclerosis of the arteries of the heart, and occurs chiefly in persons poisoned by alcohol, lead, and tobacco; in arthritic, gouty, or syphilitic patients; or in those who suffer from Bright's disease and malarial cachexia. It is also seen in elderly or overworked persons. It is important to remember that in all these cases more or less pronounced fibrous change is also present in most of the organs. Many are dependent upon **atheroma** or on **arterio-sclerosis** (Gull and Sutton, Debove, etc.). It may also be seen, as a secondary lesion, in patients suffering from valvular



affections. According to Du Pasquier, the large fibroid heart is associated with blood-stasis and ischemia.

The fibroid heart is increased in size, of a brownish colour, and firm consistency. It cuts with difficulty, and we find at the most diseased points greyish-white patches, due to the inflammatory process. These patches are more frequent in the left than in the right heart, and more marked in the pillars of the interventricular septum, and especially near the apex. In some hearts these **islets** of fibrosis are only visible under the microscope; they then appear to be composed of fibrillary connective tissue that is more or less dense, according to the age and interweaving of the elastic fibres (Letulle). Some develop around an artery affected by endoperiarteritis, while others are situated at a distance from the arteries. In the latter case we find no trace of periarteritis. Endarteritis exists alone, and shows itself at first sight by a narrowing in the calibre of the vessel. The second variety is the more frequent. Each may exist alone, but they are sometimes found together in the same patient, the fibrosis being mixed.

The muscular bundles situated at the periphery of the fibrous islets are atrophied and broken up; at the centre of the islet they have quite disappeared. Those situated externally to the inflammatory or degenerative process are enlarged, but their contents have often undergone fatty changes or amyloid degeneration (Letulle).

These changes may lead to diminished resistance of the walls of the heart, with the formation of **aneurysms**, which generally develop at the spots where the pressure is most marked and the lesions in the muscle are most pronounced—namely, near the apex and in the left ventricle (Pelvet).

**Symptoms.**—When the fibrosis has caused an aneurysm, sudden death may occur from rupture of the heart. In some cases very sharp precordial pain, due to the tearing of some deep muscle bundles, precedes the rupture by some days (Robin). Sudden death has also been noted in many cases of diffuse or circumscribed gummatous myocarditis (Mauriac).

In general, fibrosis of the heart is of slow evolution and characterized by no pathognomonic symptoms. At the onset the patient complains of palpitation, breathlessness, dyspnoea, and sometimes of angina pectoris. The pulse is full; the impulse is forcible. The apex of the heart is lowered and carried towards the axilla; the first sound of the heart is dull; the second, on the contrary, is ringing. The first sound is often reduplicated. This gallop rhythm, however, indicates arterio-sclerosis, rather than fibrosis of the myocardium. The pulse gradually becomes weak and irregular, the respiratory troubles increase, stasis-phenomena appear, and, after repeated truces, asystole finally results, just as in the case of uncompensated valvular lesions. The symptoms, due to the concomitant fibrous lesions in the kidneys, are often present as well, and these complex cases tax the

skill of the physician in recognizing, from the symptomatology, the part belonging to each.

Huchard says that it is possible to distinguish four chief forms of cardio-fibrosis: (1) A painful form, angina pectoris; (2) an arrhythmic form, or "incurable claudication of the heart"; (3) a tachycardiac form often associated with the preceding one; (4) a myovalvular form characterized by mitral or aortic murmur, with evident fibrous lesions.

**Treatment.**—At the onset we must employ means which lower the tension—*e.g.*, milk diet, massage and passive movements, balneo-therapy at Bourbon-Lancy, Royat, or at Évian. An alcoholic solution of trinitrine, in doses of from 2 to 10 drops daily in water, may be prescribed. Tetranitrol has been advised in doses of  $\frac{1}{10}$  to  $\frac{1}{8}$  of a grain several times daily.

Iodide of sodium is especially indicated. Later we may employ, but with great care, cardiac stimulants, such as caffein, digitalis, and strophanthus, for symptoms of asystole.

We must never forget that cardio-fibrosis may be **syphilitic** in origin, in which case injections of biniodide of mercury are indicated.

### III. ANEURYSMS OF THE HEART—INFARCTS AND FIBROUS PATCHES, ETC.—RUPTURE OF THE HEART.

**Aneurysms of the heart** affect chiefly the valves, the interventricular septum, and the apex.

1. **Valvular** aneurysms result from acute and especially from infective endocarditis. "The multiplication of cells, their embryonic condition, the softening of the intercellular substance, and the disappearance of the elastic fibres—phenomena which are associated with endocarditis—deprive the valve of its power of resistance." As a result of this inflammatory process, two things may happen: the valve is **perforated**, or remains distended, and forms an **aneurysm**.

Aneurysms of the sigmoid and mitral valves always have their opening at the side upon which the blood-pressure acts. A sigmoid aneurysm opens on the upper or arterial surface of the sigmoid valves, because the blood-pressure acts on this surface, while a mitral aneurysm opens on the inferior or ventricular surface, which supports the pressure of the blood during systole, when the valve is closed. Valvular aneurysms rarely preserve their spherical shape. They are more or less torn by the blood-stream, and the valve is converted into shreds.

This rupture may show itself by a murmur which appears suddenly in the course of infective endocarditis. The murmur is due to insufficiency, and is therefore systolic if the mitral valve is affected, diastolic if the lesion affects the aortic valves. Apart from these exceptional cases, we find during life no indications of the rupture of the aneurysm.

2. Aneurysms of the **interventricular septum** usually have the same origin



as the preceding form, and are often simply the extension of a valvular aneurysm. As a rule, they are found by chance at the autopsy. When they burst by perforation, communication between the two ventricles allows arterial and venous blood to mix.

3. Aneurysms of the **apex** of the heart, also called partial, are quite different in their pathogenesis. They are due to fibrous myocarditis, or to simultaneous inflammation of the myocardium and endocardium. The fibrous spots have no reactive power, and readily yield to the blood-pressure; dilatation and, later, aneurysm result. Some authors (Kundrat, Huchard) regard obliteration of the coronary arteries as important. The area, deprived of nutrient vessels, loses its resistance, and the aneurysm forms imperceptibly. As pericardial adhesions are frequently present near these aneurysms, the question has been asked whether these adhesions may not, by repeated traction, facilitate the production of a partial aneurysm.

These aneurysms are, as a rule, situated in the left ventricle, near the apex. The tumour is so large that the size of the heart is doubled (**cœur en bissac**). The aneurysm generally opens into the ventricle, and the blood-stasis within the sac is not sufficiently complete to bring about the formation of stratified clots. Cases have, however, been noted in which the aneurysmal cavity was filled with clots of this nature. The limiting wall is sometimes infiltrated with calcareous salts.

There is, so to say, no symptomatology of these aneurysms. Sometimes one is found by chance post mortem; at other times the patient dies in asystole, or more often from rupture of the heart. The existence of a diastolic murmur at the apex, independent of aortic insufficiency, has, however, been noted, and also a diastolic sound heard over the middle portion of the heart, and differing from the gallop rhythm in Bright's disease by its clearer tone (Rendu).

### Infarcts and Fibrous Patches in the Myocardium.

Post mortem, we sometimes find on the surface of the ventricles depressions resembling scars. On section of the ventricle we see that these depressions correspond to fibrous tissue which has replaced the myocardium to a more or less large extent. Sometimes, as in one of my patients, the muscle of the wall is replaced at the sclerosed patch by fibrous tissue, which is only one-third or one-fourth of the normal thickness. We find under the microscope simply connective tissue and dilated vessels.

They are called **fibrous patches in the myocardium**. They have long been known, but were until recent years looked upon as the result of chronic inflammation. We know to-day that these lesions are distinct from localized fibrosis of the myocardium, and are the result of an infarct; they are true cicatrices.

The myocardium may indeed be the seat of an infarct, just as all organs

and tissues in which the arterial anastomoses are not sufficiently numerous. The heart answers to this disposition, for the coronary arteries, which anastomose freely at the base, become terminal at the apex. The seat of election of these infarcts, caused by obstruction of one of the coronary arteries, is thus explained. This obliteration rarely occurs from embolism. It is in the majority of cases a thrombosis, produced by the lesions of arteritis obliterans.

The infarct may show itself either as a limited yellow or red focus, the colour being due to necrosis of the damaged tissue, or as a hæmorrhagic focus. In the latter case it has been supposed that the blood in the heart cavity secondarily inundates the focus of necrosis. The formation of some aneurysms of the heart may be thus explained.

If the patient does not succumb, the infarct changes into cicatricial tissue, which may undergo calcification, and form the fibrous patch in the myocardium. When this scar is extensive, it forms a weak spot in the wall of the ventricle, and may lead to aneurysm and rupture of the heart.

Although the production of an infarct may sometimes be revealed by precordial pain and tachycardia, the lesion has, as a rule, no clinical history.

### Rupture of the Heart.

**Rupture of the heart** occurs in the course of some affections of the myocardium. Partial aneurysm of the heart, softening of the heart muscle by rapid obliteration of a coronary artery, fatty degeneration, and infective myocarditis, are the most frequent causes of these ruptures.

The rupture may take place from without inwards, but it more often occurs from within outwards, and is then due to the pressure of the blood upon the inner surface of the heart. In the former case, on the contrary, the contraction of the affected muscle produces the tear. Both mechanisms may also occur together. In this case the two lines of rupture, instead of corresponding, are separated by unbroken muscular bundles. The rupture may be double or triple, but the apex of the left ventricle is the seat of election. At this point the wall is thinned, especially in cases of partial aneurysm, or of fibrous myocarditis. The pericardium is usually full of dark coagulated blood.

The rupture is usually caused by a strain. Cases of rupture during sleep have, however, been quoted. It shows itself by inexpressible distress and acute precordial pain, which may radiate to the back and to the left shoulder. The patient utters a cry, his face becomes cyanosed, the beating of the heart is tumultuous, the pulse is small and cannot be counted, the breathing rapidly becomes embarrassed, and the patient dies within a few seconds. The rupture may be gradual, and may only become complete at the end of several days. We then see, in addition to the pain and vomiting which marked the onset of the rupture, the other symptoms just enumerated.



## IV. SYPHILIS OF THE HEART.

**Ætiology.**—Syphilitic lesions of the heart, first noted in 1859 by Virchow, are now becoming well known. They are chiefly seen during the tertiary period, about ten years after infection (Jullien), though they may appear earlier or later. Parrot has published several cases of hereditary syphilis of the heart in the new-born. Letulle and Nattan-Larrier have seen miliary gummata upon the valves in a syphilitic child who died at birth.

**Pathological Anatomy.**—Syphilis affects the valves more rarely than the myocardium, especially the left ventricle. The interventricular septum and orifices are not often involved. Gumma or fibrosis is the usual lesion. The latter is recognized by its clear localization to one part of the heart, the other zones being free, and by the severity of the arterial lesions at this point, the arteries of the other regions being healthy. These characters, however, are not specific, and at most allow a suspicion of syphilis, especially in the absence of other visceral manifestations.

Syphilitic gummata in the heart, as in other organs, constitute the only real specific lesion. Their yellowish appearance, their elastic consistency, their well-defined limits, their multiplicity, or the coexistence of miliary gummata in the neighbourhood, and the recognition of lesions in the liver, kidneys, etc., can hardly allow any mistake. In some cases they are surrounded by healthy muscular tissue, and project towards the endocardium, or towards the pericardium. The serous membranes are thickened; the layers of the pericardium may be adherent. In other cases, on the contrary, the endocardium is not thickened, but ulcerated, the gumma having emptied itself into the ventricular cavity, while the blood has entered the cavity, hollowed out at the expense of the myocardium. The possibility of partial aneurysm is readily seen.

**Description.**—The disease often remains latent, and is only discovered post mortem. In other cases we find all the signs of a chronic affection of the myocardium—namely, increased precordial dullness, indistinct and irregular heart sounds (Semmola), arrhythmic pulse, retro-sternal pain, dyspnœa on effort, œdema, and cachexia. In short, we see the evolution of cardio-fibrosis or of progressive asystole.

More striking are the cases in which a syphilitic person previously exempt from cardiac trouble is taken ill with acute asystole, that proves fatal in one or two days (Tessier). Rapid death may be due to agonizing dyspnœa, recalling some pulmonary lesion rather than a cardiac affection. Lastly, sudden death may be consecutive to rupture of the heart, to syncope, or to embolism. The pathogenic diagnosis of cardiac syphilis is a matter of supposition. However, in a patient with old syphilis, who suffers from symptoms of cardio-fibrosis, syphilis should always be thought of, and daily injections of biniodide of mercury be administered.

## V. TUMOURS OF THE HEART AND TUBERCULOSIS OF THE MYOCARDIUM.

Some rare cases of **primary spindle-celled sarcoma** are seen. **Epithelioma** naturally affects the heart only as a secondary growth. Most cases of "cancer of the heart" are really secondary nodules of epithelioma, and are quite exceptional. These nodules are secondary to cancer of the alimentary canal, the lung, etc.; in most cases they coexist with secondary nodules in the lung. They are situated in the myocardium, and chiefly in the right heart or in the interventricular septum. Cancer of the heart is not as a rule recognized during life. In some cases it may give rise to dyspnoea, tachycardia, precordial pain, etc. I have reported the case of a patient suffering from pleuro-pulmonary cancer, whose pulse-rate was extremely quickened. At the autopsy I found a cancerous nodule as large as a hemp-seed in the interventricular septum.

The other tumours are myxomata, forming pedunculated swellings on the auricular surface of the mitral valve, and hydatid cysts, which may be present in the myocardium as in other muscles. This affection is exceptional, and its sole clinical interest lies in the possibility of hydatid emboli, when the cyst opens into the cardiac cavity.

**Tuberculosis of the myocardium** is a rare affection, which has no clinical history, and is found by chance post mortem. As a rule, it is coexistent with tubercular pericarditis, and frequently with tubercular lesions of the peritracheal glands.

We may find miliary tuberculosis, but more often we see large isolated tubercles in which Koch's bacillus has been found. These large tubercles may be caseous or calcified. Koch's bacilli have been found by Péron even in the calcified lesions. Tubercular lesions of the myocardium are chiefly situated in the ventricles, and are most often seen in children.

Tuberculosis of the myocardium is frequent in domestic animals which have become tubercular (dogs, bovine animals, and pigs), while it is rare in laboratory animals inoculated with tubercular products (Péron).

## VI. HYPERTROPHY AND DILATATION OF THE HEART.

**Pathology.**—A muscle hypertrophies when it undergoes increased work. This general law includes the heart, which hypertrophies as the result of excessive or repeated contractions. The causes of this hypertrophy may be divided into two classes (Jaccoud): (1) Simple or purely functional hypertrophy; (2) hypertrophy from some mechanical obstacle.

**Simple Hypertrophy.**—This form, which is also called primary, is due to nervous palpitation, and consequently to the causes producing this palpitation (adolescence, hypertrophy of growth, excesses of every kind,



abuse of drink, tobacco, tea, or coffee, hypertrophy in exophthalmic goitre). The whole heart is affected, and the hypertrophy does not as a rule involve any special portion of the organ.

**Hypertrophy from Mechanical Obstacles.**—The causes are : (1) lesions of the heart ; (2) lesions of the vascular system ; (3) lesions or abnormal conditions of more or less distant organs.

1. The mechanism by which **valvular lesions of the heart** cause hypertrophy is readily understood. When an obstacle exists at one of the orifices, whether it is the reflux of the blood-wave, as in insufficiency, or whether this wave pass with difficulty through the orifice and the cavity, which is in front of the diseased opening, in order to overcome the excess of pressure, the walls of the cavity are subjected to exaggerated functional irritation, which finally produces hypertrophy. The hypertrophy, which is at first localized to the cavity in front of the diseased opening, finally **extends to the other cavities**. For example, lesions of the **mitral orifice** increase the blood-pressure in the left auricle, which hypertrophies ; the blood-stasis in the left auricle increases in its turn the blood-pressure in the pulmonary veins, the pulmonary artery, and the right cavities of the heart ; the right ventricle therefore is exposed to excess of work, as well as to exaggerated functional irritation, which, by its persistence, produces hypertrophy. This hypertrophy is, within limits, compensatory to the mitral lesions, but the excess of blood-pressure in the right ventricle finally reacts on the venæ cavæ and the capillaries of the greater circulation. From the capillaries the excess of tension reaches the arterial system, and finally the left ventricle shows hypertrophy.

The lesions of the **aortic orifice** produce analogous effects, but different in degree. The power of the left ventricle and the enormous hypertrophy which accompanies aortic stenosis and incompetence protect the lesser circulation for a long while, so that the excess of tension in the pulmonary vessels and the right cavities of the heart appears later, and is less complete than in mitral lesions.

2. Aneurysm of the aorta or of the great vessels, acute or chronic aortitis, endarteritis, and general atheroma, are the lesions of the arterial system which may produce hypertrophy of the left cavities of the heart. How do such lesions determine hypertrophy of the left ventricle ?

In the normal state the elasticity of the arteries diminishes the resistance experienced by the blood in passing from the heart to the vessels (Marey). Hence diseases which abolish or diminish this elasticity increase the resistance and raise the blood-pressure, the result being hypertrophy of the left ventricle.

This pathology is perfectly applicable to chronic aortitis and to more or less general atheroma, though it does not sufficiently explain other cases.

For instance, **acute aortitis** is nearly always accompanied by hypertrophy of the left ventricle, which must not be set down to a concomitant lesion of the aortic orifice, because it occurs in cases in which acute aortitis exists without complications. **Aneurysms** are often accompanied by hypertrophy of the left ventricle. In fifty-eight cases of aortic aneurysm hypertrophy of the ventricle was present in fifty-three. This hypertrophy cannot be set down to excess of pressure, for physiological experiments have proved that the presence of an elastic sac in the course of a vessel does not interfere with the circulation. Aneurysms of the arch of the aorta are by no means always accompanied by hypertrophy of the heart. In a certain number of cases (I have verified this fact three times post mortem) the heart was normal in size, although the aortic aneurysms were large.

Arterial lesions of the lesser circulation cause hypertrophy of the **right ventricle**. Congenital or acquired **stenosis** of the **pulmonary artery** leads to hypertrophy and dilatation of the cavity of the right heart.

**Arterio-sclerosis** of the heart, associated with **sclerosis of the myocardium** will be discussed under Bright's Disease.

3. The morbid condition of **certain organs** reacts upon the heart and determines hypertrophy. Chronic diseases of the **lung** react on the **right ventricle**, which dilates and hypertrophies; chronic pleurisy and costo-vertebral deformities may produce the same result; **interstitial nephritis** causes hypertrophy of the left ventricle (Traube, Potain). The mechanism in this case will be discussed under Bright's Disease. Transitory hypertrophy of the left ventricle during pregnancy is generally admitted. Potain has reported cases of cardiac hypertrophy following lesions of the brachial plexus. Chronic lesions of the **liver** may favour dilatation and slight hypertrophy of the right ventricle (Potain). Cardiac hypertrophy following growth is said to result from a want of parallel development between the heart and the other organs (Sée). Dilatation of the cavities of the heart nearly always accompanies hypertrophy (excentric hypertrophy). It may be general or local, and as it depends on the lessened resistance of the walls, it is more marked in the right cavities of the heart.

Often the **chief lesion is dilatation**, and is seen following diseases which lessen the resistance of the heart-wall. The **dystrophies** of the heart muscle, described under **Degeneration and Myocarditis**, belong to this class.

**Pathological Anatomy.**—In true hypertrophy the **size and weight** of the heart are increased. The weight may reach as much as 1,000 grammes (the normal weight is 300 grammes). The thickness of the left ventricle reaches 3 or 4 centimetres (12 millimetres being the normal); the thickness of the right ventricle amounts to from 1 to 2 centimetres (3 millimetres being the normal thickness).



The **structure** has given rise to much discussion (Letulle). Some hold that the hypertrophy is due to increase in the **number** of the primary bundles, while others admit increase in the **size** of the bundles, or both of these causes. Hypertrophy is not due solely to the change in the muscle. Lesions of the connective tissue, the endocardium, the pericardium, and the vessels are present in a hypertrophied heart. Nevertheless, the change in the muscular fibre is most important. There is no proof of the multiplication of the primary bundles, while their hypernutrition is admitted by all authorities, and each primary bundle, instead of having the normal diameter of 15 to 20  $\mu$ , reaches one of 25 to 30  $\mu$ .

The muscular hypertrophy is dispersed through the affected regions, and in proportion as hypertrophy gives place to organic decay, the heart is invaded by diffuse fibrosis, by endoperiarteritis of the small vessels, and by disseminated fatty degeneration.

In some cases more or less general **cardiac cirrhosis** is seen. The fibrous tissue appears to be localized around the small arteries in the heart muscle. The fibrosis of the myocardium is said to be consecutive to endoperiarteritis.

The **shape** of the heart shows little change when the hypertrophy is general, but is much modified in cases of partial hypertrophy. In this manner local hypertrophy of the left ventricle increases the longitudinal diameter of the heart, giving it an oval shape and an almost horizontal direction. The septum encroaches on the cavity of the right ventricle, and the papillary muscles are much enlarged. Hypertrophy of the right ventricle increases the transverse diameter of the heart, and tends to give it a spherical shape.

**Description.**—As Jaccoud has observed, care must be taken to distinguish the symptoms proper to simple hypertrophy from those of hypertrophy, accompanied by cardio-pulmonary changes. As long as the muscle has not undergone fibro-fatty degeneration, hypertrophy of the heart produces neither blood-stasis nor dropsy. On the contrary, the hypertrophied walls sometimes propel the blood with increased vigour, and cause congestion. Thus the enormous hypertrophy of the left ventricle in aortic insufficiency is often accompanied by cerebral congestion, with flushing, dimness of sight, ringing in the ears, headache, vertigo, and epistaxis. The impulse of the blood-wave is shown throughout the whole arterial system by pulsation in the carotids, bounding pulse, dilatation and elongation of the arteries, and the patient complains of palpitation, precordial distress, and dyspnoea.

When, however, the hypertrophied and dilated muscles undergo fibro-fatty degeneration, and compensation begins to fail, the train of symptoms which we have enumerated several times in the last few sections appears.

The **physical signs** of hypertrophy of the heart are as follows: The apex of the heart is displaced downwards and outwards, and the impulse has its maximum to the left of the sternum if the left ventricle is affected, and in the epigastric angle if the right ventricle is involved. The precordial bulging depends on the extent of the hypertrophy. The dullness is increased in the longitudinal direction by hypertrophy of the left ventricle, and in a transverse one by that of the right ventricle. Pure hypertrophy, apart from valvular lesions, does not show itself by any abnormal sound. The gallop rhythm present in hypertrophy of renal origin will be studied under Bright's Disease.

**Diagnosis—Prognosis.**—The diagnosis of hypertrophy of the heart is made from the symptoms above mentioned. We must be on our guard in cases where the size of the heart is masked by the exaggerated percussion resonance of emphysema. The diagnosis between hypertrophy and pericardial effusions has been discussed under **Pericarditis**. Purely functional hypertrophy is not serious, and the compensatory form is favourable, in that it diminishes for a time the extreme gravity of the valvular lesions. The phenomena of congestion due to excessive cardiac contractions may produce troublesome results. In such cases it is necessary to diminish the tension in the arterial system, and to moderate the cardiac erethism.

## VII. ASYSTOLE.

The word **asystole** must not be taken literally, for the absence of systole means death. In coining the word **asystole**, Beau supposed that, at an advanced period in valvular lesions of the heart, the contractions of the organ are so faulty that they open the road for congestion, œdema, and all the symptoms of cardiac cachexia. Beau's description remains true, but the interpretation of asystole must be modified. Asystole is produced by any cause which interferes with the contraction of the heart muscle. This class includes excessive fatigue, overwork, adherent pericardium, valvular lesions, imperfectly compensated dilatation, and especially degeneration of the muscular fibre (Stokes). It is proper to add that these causes of **cardiac origin** may be reinforced by those of **peripheral or vascular origin**, such as malnutrition and defective resistance of the small vessels.

The symptoms and the course of asystole and of cardiac cachexia have been described under Valvular Lesions of the Heart and their Treatment, so that it is unnecessary to repeat them.



## CHAPTER IV

### NEUROSES OF THE HEART

#### I. PALPITATION.

ACCORDING to many authors, palpitation is characterized by a change in the frequency, rhythm, and intensity of the heart-beats. This view of the question appears to me incomplete. The pulse-rate in fever exceeds 120, although palpitation is not present. The force of the heart-beats is much increased in some cases of hypertrophy of the left ventricle, but yet palpitation may be absent (Potain). Intermittence in mitral lesions certainly does not entail palpitation. Palpitation, then, is not absolutely associated with changes in the frequency, rhythm, and intensity: it often accompanies, but is not the consequence of, such changes.

“Palpitations,” says Peter, “are spasms of the heart,” and, I may add, distressing and painful spasms. We are not conscious of the action of the organs in life: when we are so, we recognize it by discomfort or by pain. In the normal condition the stomach and intestine execute their movements without our knowledge. When these movements change in character (spasms), they become painful.

The heart follows the same law. In palpitation the heart-beats are distressing or painful to a variable extent, from simple discomfort to pain with angina and tendency to syncope.

**Pathogenesis.**—Whatever is the initial cause of palpitation, some trouble in the nervous system of the heart must finally be acknowledged. In some cases this nervous trouble appears apart from any material change in the organ; in other cases a material change is manifest. Between these two extremes many intermediate forms are found, but their classification is impossible.

**Nervous Palpitation.**—The physiology of the innervation of the heart has been examined for an explanation of palpitation, and it is said that the pneumogastric and great sympathetic nerves are antagonists. Inhibition of the pneumogastric or excitation of the sympathetic produces practically the same effect—namely, the acceleration of the heart-beats. Hence all causes which lessen the action of the former or increase that of the latter

may give rise to palpitation. Such a statement appears inexact, because acceleration is not sufficient to produce palpitation, and consequently it is not evident how causes which diminish the action of one of the antagonistic nerves may not at the same time enfeeble the action of the other. Abnormal irritability of the nervous system, cerebro-cardiac neuropathy (Krishaber), exophthalmic goitre, excesses of every kind, abuse of drink, coffee, tea, or tobacco, are the usual causes of nervous palpitation. The so-called palpitations of growth and of **reflex** origin also enter into this class.

**Palpitation with Material Lesions.**—In this class we find palpitation, which accompanies diseases of the heart, such as pericarditis and endocarditis, myocarditis, hypertrophy and dilatation, or valvular lesions. Acute inflammation, which at first sight would appear likely to irritate the nerve endings, is rarely accompanied by palpitation, while valvular lesions frequently are, and it remains to be seen what is the mechanism in these cases. Every case of stenosis, says Peter, is accompanied by spasm. Pathologically constricted tubes, such as the urethra or the œsophagus, and those normally constricted, such as the glottis or the biliary ducts, are subject to spasmodic contraction, and stenoses of the heart also provoke spasms called palpitations. But how are we to explain palpitation in aortic insufficiency, which is the exact opposite of stenosis?

**Palpitation of Mixed Origin.**—Marey has shown that lowering of the blood-pressure increases the rate of the heart-beats. The heart beats more quickly when its load is lessened. This experimental fact has been used to explain palpitation consecutive to hæmorrhage, high temperature, or violent exercise, the blood-pressure diminishing as the result of hæmorrhage, or of dilatation in the peripheral vessels (vasomotor nerves). A fresh element—namely, the **quality** of the blood, which is poorer in red corpuscles and in oxygen—is present in the palpitation of anæmia.

**Description.**—The palpitations may be isolated or grouped in attacks. In slight attacks the heart-beats are troublesome rather than painful, and are accompanied by precordial discomfort. In violent attacks the heart “beats as if it would burst the chest,” its movements are sometimes tumultuous and disordered (arrhythmia), the pain is agonizing, the patient suffocates, his speech is interrupted, his face is pale and bathed in sweat, and his hands are cold; he is threatened with faintness and syncope.

Examination during an attack yields various results. In some cases the heart-beats are tumultuous and disordered, but in others they preserve their regularity. The pulse is not always in exact relation with the heart-beats. The pulsation in the radial artery may remain normal, in spite of the apparent intensity of the ventricular contraction.

Palpitation is often brought on by trifling causes: emotion, movement, or a heavy meal.



The **diagnosis** of the cause is the most important feature, and it is necessary to know whether the palpitations are purely nervous or are associated with some cardiac lesion. The **treatment** depends upon the recognition of the cause. The first indication is to eliminate anything which may produce palpitation (excesses of every kind—tea, coffee, tobacco, emotion, heavy meals). The preparations of digitalis are the more indicated in proportion as the arterial tension is but little raised and the pulse is weak and compressible (Jaccoud).

Bromide of potassium, valerian, the application of ice-bags to the precordial region, and hydrotherapy yield good results.

## II. PAROXYSMAL TACHYCARDIA.

Bouveret in 1889 applied the term "paroxysmal tachycardia" to a disturbance of the cardiac rhythm, characterized by crises during which the pulse beats from 180 to 220 times a minute. These crises come on suddenly without appreciable cause at various intervals, and cease just as suddenly, after a duration varying from a quarter of an hour to some days. Since Bouveret's article many cases have been published, and I have had a patient under observation for two years. His history will serve as my type, because all cases of paroxysmal tachycardia are a replica of one another.

One day, without apparent reason, he felt as though his heart were beating with extraordinary quickness and violence. The attack, which lasted an hour and a half, was painless, and passed off suddenly. Next month a second attack. The crises have since recurred once or twice a month, and lately almost daily. The attack may be thus described: Something appears to get loose in the chest, and the heart beats violently. After a period, varying from a quarter of an hour to four hours, the attack ends as suddenly as it began. On examination during a crisis nothing special is to be noted in his appearance; no distress, no angina; he talks and is often able to carry on his work. During the attack a tremor of the chest-wall is seen, and on placing the hand over the heart, as many as 200 beats a minute may be counted. This condition lasts from one to three hours. At the end of the attack the patient feels a sudden shock due to a beat of exceptional force, and the normal rhythm is then re-established.

It is curious that the patient can sometimes stop the crisis by fixing the chest in forced expiration and holding his breath; he becomes cyanosed, and the jugular veins swell. He remains for a moment in this condition, and is sometimes obliged to take another breath without having strangled the crisis, but at other times he says: "It has stopped." The tremor ceases, and the pulse suddenly falls to 70 or 80.

In all published cases the crisis has been practically identical.

Sharp emotion, blows upon the epigastrium, abuse of coffee or of tobacco, digestive, pulmonary, or uterine troubles, and physical or mental strain have been in turn invoked to explain this paroxysmal tachycardia. As a matter of fact, we do not know its ætiology. In the patient's previous history, hysteria, neurasthenia, and hereditary nervous diseases are wanting.

As a rule, no valvular lesion is found. Bouveret accordingly called the disease "**essential** paroxysmal tachycardia," in order to distinguish it from ordinary tachycardia seen in cardiac affections. There are cases, however, in which the disease, while remaining identical from the semeiological point of view, coexists with mitral or aortic endocarditis. The patient of whom I have just spoken was suffering from aortic incompetence.

The disease may last for an indefinite period. If the crisis only lasts a few hours, it is perfectly bearable. When it lasts several days, hæmoptysis, indicative of pulmonary stasis, may occur. If the crisis lasts still longer, the liver becomes large and painful, the urine is suppressed, the right heart dilates, œdema and serous effusions form, and the patient appears dying. But as soon as the crisis ceases, the circulation is re-established, the œdema is absorbed, the urine becomes abundant, and the patient, who appears moribund, recovers.

The prognosis, however, is grave, for the patient may succumb in a prolonged attack. The disease may remain stationary and be prolonged indefinitely, or it may improve and end in recovery. In my patient the attacks, which occurred daily, have become less frequent, and he has only one crisis every week or fortnight.

Post-mortem examinations have been performed in fatal cases. Dilatation of the right heart and venous stasis in every organ have been found; the lesions are evidently secondary and terminal. The primary lesions have not been found; the neuromotor system of the heart has always appeared healthy; the bulb, the spinal cord, the pneumogastric, and the sympathetic nerves are free from disease, and the heart itself only shows commonplace lesions.

The characters of the disease are so specific that it is not necessary to dwell upon the diagnosis. The crises of paroxysmal tachycardia resemble neither the ordinary palpitation in heart disease nor the reflex palpitation of dyspeptic or hepatic origin, etc. Tachycardia in Basedow's disease may supervene in crises, but does not show the sudden passage from the state of crisis to that of absolute calm and *vice versa*.

Treatment has so far been powerless to influence this disease.

### III. PERMANENT BRADYCARDIA.

The pulse-rate in health is from 70 to 72. As a result of different influences (intoxication, jaundice), the rate may fall below 50; but in this case the fall is transitory, while this section deals with **permanent** slowness of the pulse.

The slowing is so great that the pulse falls to 20 beats, or even less. In one of my patients the pulse-rate fell to 14 per minute for some days.



The pulse preserves its regularity. The sphygmographic tracing shows a short upstroke and a long-drawn-out downstroke. The arterial pressure is usually higher than normal.

The heart-beats are well marked. The duration of the first pause is not increased, and it is the increase of the second pause that causes the slowness of the cardiac rhythm. In short, each heart-beat is normal, but the interval between the beats is much increased. This interval may not be completely silent. After the diastolic sound, muffled rumbling sounds are heard. Vaquez has shown that they are due to an abortive contraction of the auricles, and radiography has confirmed his interpretation.

Bradycardia may be associated with perfect health, or may be accompanied by morbid phenomena, caused in several ways.

Any lesion that affects the cardiac innervation may result in bradycardia, either by stimulating the system that slows the rate (bulb and vagus), or by inhibiting the system that quickens it (spinal cord and great sympathetic).

Lesions may be present in the heart, and affect the intracardiac ganglia (syphilitic gumma of the interventricular septum and upper part of the left ventricle, in Rendu and Massary's case). They may affect either pneumogastric nerve, which is compressed in its intrathoracic course either by the dilated aorta (Stackler's case) or by enlarged mediastinal glands (Lannois' case). The pneumogastric may also be injured in its intracranial course (compression by a gummatous tumour of the cerebellum). Many lesions may affect the cardio-inhibitory nucleus in the bulb (injury, cervical caries, syphilitic or tubercular tumours, infectious myelitis).

Lesions of the accelerator system are less often in evidence, yet the presence of unilateral dilatation of the pupil has shown the lesion to be in the cardio-spinal centre, which is near the centre for dilatation of the pupil.

All these cases of bradycardia depend upon such gross lesions that there can be no possible doubt as to their existence; but the case is different in the syndrome which Charcot has called "Permanently slow pulse, with syncopal or epileptiform crises," and for which Huchard has proposed the name of "Stokes-Adams disease." The patients, who are of advanced years, often show signs of renal insufficiency; their arteries are hard and tortuous, and the tension is high. They are subject to attacks of syncope, which in one of my cases had the following characters:

The syncope was ushered in by a kind of aura: general malaise, feeling of weight in the chest, buzzing in the ears, cold sweat, pallor, and chilliness. The patient lost consciousness and looked almost dead, with dull eyes and cadaveric tint. After two or three minutes the colour returned, the eyes opened, and she rapidly recovered consciousness. She had no involuntary emission of urine, no epileptiform movements. After the crisis she complained of feeling bruised, as though her body had been racked; she sometimes felt sick, but did not vomit.

The syncope may be immediately followed by an epileptiform attack. Whether the attacks are syncopal or epileptiform, they are often ushered in by very marked slowing of the pulse.

The attacks succeed one another at variable intervals; the more frequent they become, the more grave the prognosis. The patient finally dies during an attack of syncope which is more prolonged than the others, and the duration of the disease does not exceed three or four years.

The pathogenesis of the Stokes-Adams syndrome has given rise to recent work of much importance. Stanley Kent, His junior, Tawara, and Keith and Flack have investigated the anatomical structure and relations of the auriculo-ventricular bundle, to which the name of **His's bundle** is now given. This bundle forms a connecting-link between the auricles and ventricles, and its function is of a conducting nature. As the bundle has a complicated structure, it is usually described in three sections: auricular, nodal, and ventricular.

In the **auricular section**, the fibres are derived from (*a*) the circular fibres of the right auricle just above the base of the septal or posterior cusp of the tricuspid valve, and (*b*) the muscular tissue of the inter-auricular septum at the front and lower part of the septum on its right side, just above the anterior end of the base of the posterior cusp. These fibres (circular and septal) enter the central fibrous body of the heart, situated between the aortic and the two auriculo-ventricular orifices where they form the **knoten**.

The **nodal section** (**knoten**) is, as already mentioned, situated in the central fibrous body of the heart; it forms an intricate network of muscular fibres, embedded in fibrous tissue.

The **ventricular section** leaves the nodal section as a single bundle, which represents the main connecting stem between the auricles and ventricles. It runs forward along the upper edge of the muscular portion of the septum between the ventricles, lying just below the *pars membranacea septi*, and beneath the mesial part of the base of the septal cusp of the tricuspid valve. In this situation, the main stem breaks up into its right and left divisions, which pass respectively into the septal wall of the corresponding ventricle. Here each division breaks up into branches, which ramify under the endocardium and form part of the system known as Purkinje's fibres.

The muscular fibres of the auriculo-ventricular bundle are much paler than the ordinary muscle-fibres of the heart. The bundle contains both nerve-fibres and ganglia; it is very distinct to the naked eye in the heart of the calf, and recognizable in the human organ by microscopic examination.

The researches of His, Humblet, Erlanger and Hering have shown that the severance of this bundle produces an absolute independence of the



auricular and the ventricular contractions. In this case, the auricular systole remains normal, whereas the ventricular beatings of the heart are retarded. Excitation of the bundle, on the contrary, induces supplementary contractions (or extra systole) of the ventricles. His's bundle is, then, a conductor of the excitations proceeding from the right auricle and going to the ventricles. When this bundle is destroyed in man, the auricles continue to contract normally, whilst the ventricles are retarded. The Stokes-Adams's syndrome is, thus, realized.

Certain post-mortem examinations have proved the well grounded theory that the disease is due to a lesion of His's bundle. The latter has been found destroyed by syphilitic gummata (Rendu, Vaquez, Esmein) (which appears to have a special predilection for this region), by congenital malformations (Morquio), by sclerotic myocarditis complicating an old valvular lesion, by cancerous nodules, by acute dégénération of the myocardium.

In these cases, nervous complications always follow the prolonged suppression of the normal ventricular contractions. They are due to ischæmia of the nervous centres which results therefrom. The duration of the circulatory trouble appears to regulate the nature of the nervous crisis. If it last five seconds, there is vertigo. If it persists from seven to eight seconds, we find syncope and ictus. Epileptiform convulsions appear on the scene beyond ten seconds. This description is somewhat schematic and, if the student look closely, he will find that things are far from occurring with such regularity.

Though certain points of this study be still obscure, bradycardia through muscular lesions is a very definite syndrome. The nervous origin of certain other cases must, however, not be rejected, such as those which I have previously mentioned. It suffices to know this pathogenesis of a nervous kind well to be convinced of its importance. There is, also, a means of distinguishing the origin of the syndrome, viz. by atropine (Dohio). To accomplish this, an injection of 1 milligramme of atropine is given subcutaneously. It is known that this alkaloid paralyzes the terminations of the pneumogastric nerve, which is a cardio-moderator. The slow pulse of nervous origin ought to disappear after this test, because the nerve which, by its excitation, produces bradycardia has its functions annihilated. But the slow pulse of muscular origin is in no way modified by the atropine. There is, however, nothing mathematical in this, and it is, perhaps, possible that this side of the question is open to revision. I have said that bradycardia may be due to syphilis; this idea yields a most important therapeutic indication and, under such circumstances, we must always think of syphilis. In such a case, Erlanger cured a patient by mercurial injections. In other cases, therapeutics help little. A milk diet gives some benefit in the case of renal insufficiency (Debove).

Rest is absolutely indicated (Hirtz). During an attack of syncope, the patient must keep his bed. Gruenbaum and Kidd state that they have obtained favourable results from thyroidine, but this is a debatable point. Application of Vienna paste to the precordial region seems, to me, to be indicated. In a general way, this revulsive treatment is too much neglected.

#### IV. EXOPHTHALMIC GOITRE—BASEDOW'S DISEASE.

##### Medical and Surgical Treatment.

The interesting disease which we are about to discuss would perhaps be more correctly included under the bulbar neuroses. I have, however, retained it under Neuroses of the Heart, because the condition of that organ figures so largely in all the descriptions of this disease. In England and America the disease is more commonly known under the name of Graves' disease. The cardinal features of this peculiar malady are very well summed up in the quotation from Trousseau's Clinical Lectures, which is given below; his description, as might be expected, is most masterly, and may be carefully studied with the greatest benefit by any one interested in the subject.

"Many patients will consult you for palpitation of the heart, but you will at once be struck by their strange look and their prominent eyes," and you will find hypertrophy of the thyroid gland.

These patients are suffering from **exophthalmic goitre**, Graves' or Basedow's disease, characterized in a typical case by the following troubles, which are mostly of nervous origin: (1) cardiac troubles; (2) ocular troubles; (3) hypertrophy of the thyroid gland; (4) motor troubles; (5) psychical troubles. According to the case, these various symptoms may appear in succession or in combination. In some cases they are predominant; in others they are indefinite.

**Description—1. Cardiac Troubles.—Acceleration of the heart-beats** is the essential symptom of Basedow's disease. This symptom is never lacking; it is found in early cases, and even after all the other symptoms have disappeared, the acceleration of the heart-beats persists for some time longer. This symptom is due to paralysis of the nucleus of the vagus nerve. In some cases the patient does not notice the tachycardia, but in others the beats are distressing and cause palpitation. This palpitation becomes gradually more severe, and supervenes in the form of attacks; 120 to 150 beats per minute may be counted during the paroxysms. The palpitation is often violent, and the cardiac impulse so strong that it raises the chest-wall, while the heart "beats as though it would burst." In spite of this disturbance, the beats may not be irregular. In another



variety the beats are irregular ; the heart seems feeble, and its pulsations are hurried, uneven, and abortive. During the paroxysms the arrhythmia and cardiac weakness may lead to acute asystole, with dyspnœa, angina, and cyanosis. In some cases the symptoms of **angina pectoris** have been recorded. The symptoms of asystole disappear between the paroxysms.

Lastly, in some patients the troubles of innervation are complicated by hypertrophy, dilatation of the cavities, or insufficiency of the tricuspid and mitral valves. The hypertrophy is attributed to the functional hyperactivity of the heart, or to an excess of tension consecutive to lesions of the valves. These valvular lesions (insufficiency of the auriculo-ventricular and aortic valves) are sometimes entirely **mechanical** in origin. They may be caused by dilatation of the ventricles ; the heart muscle, weakened by the strain, allows overdistension. These lesions are usually transient, and cease with the disease, though they may become permanent.

The **carotid** arteries are tortuous and enlarged. The pulsation raises the tissues of the neck. These beats are not the *contre-coup* of the cardiac impulse, but the vessels in the neck, both arteries and veins, beat on their own account, and appear to participate in the overgrowth and pulsation which affect the vessels of the thyroid gland. The sphere of vascular excitation appears limited to this region, for violent pulsation is not found in the abdominal aorta, the radial artery, or elsewhere. In spite of these vascular troubles, the blood-pressure remains normal. Inequality of the two radial pulses has been found.

**2. Ocular Troubles.**—Spasms of the upper eyelid may precede or accompany the exophthalmos (Wecker). When the glance is directed downwards, the upper eyelid no longer follows the ball of the eye, but remains fixed above it (de Graefe). The exophthalmos is double, and may be so extreme that the eyelids can scarcely cover the ball, giving to the physiognomy a strange expression of astonishment and terror, which Marchal de Calvi called the "tragic eye." This appearance is partly due to retraction of the levator palpebræ superioris, which causes considerable enlargement of the palpebral aperture (Stellwag).

During the paroxysms the exophthalmos increases to such a degree that luxation of the eyeball has often been seen (Pain). The conjunctiva is often injected, and the cornea, being continually exposed, may become infected and ulcerated. The sight is unaffected, and it is rare to see myopia or presbyopia. The ophthalmoscope may reveal congestion of the choroid and dilatation of the retinal vessels. Galezowski and the writer have seen retinal hæmorrhages.

In patients suffering from Basedow's disease, with or without hysteria, we sometimes see **bilateral paralysis** of the motor muscles of the eyeball, in which case the voluntary movements are chiefly affected, while the

reflex ones are in part preserved. The whole external musculature—namely, the recti and the obliqui, innervated by the third, fourth, and sixth pairs—is paralyzed. In addition, the movements of the eyeball are abolished, and only elevation of the upper eyelid is, as a rule, preserved. This inability to move the eyes gives a strange fixed look, and the patient cannot see an object to his right or his left without turning his head. The name “**external ophthalmoplegia**” has been given to this paralysis of the external musculature. The internal musculature, which comprises the ciliary muscles and the radial or circular fibres of the iris, and governs accommodation and the dilatation or contraction of the pupil, is unaffected in Basedow’s disease. External ophthalmoplegia may be seen in patients suffering from both exophthalmic goitre and hysteria, or from either malady alone.

3. **Thyroid Body.**—The enlargement of the thyroid body results from dilatation of its numerous vessels. Auscultation of the gland therefore reveals simple or double blowing murmurs, which are louder during diastole, as in a cirroid aneurysm. On palpation, we feel expansile pulsation, as in an aneurysm. The right lobe is more often invaded than the rest of the organ, and although the tumour is not as large as an ordinary goitre, it may, by compression of the trachea or by the excitation of the recurrent nerves, produce voice changes, spasm of the glottis, and paroxysmal attacks of suffocation.

4. **Motor Troubles.**—Tremors, paralysis, and choreiform movements may appear at the onset or during the course of Basedow’s disease.

**Tremor** is almost constant, but may be so slight that care is required to discover it. The tremor is most frequent in the upper limbs. The arms and hands, even during rest, show tremors, which hamper writing and all the delicate functions of the hand and fingers. In the lower limbs the tremor is also present during repose, as well as in walking. During repose the limbs show a kind of pedal movement. In exceptional cases the tremor may invade the whole body, including the face and the tongue. Fibrillary movements are usually seen in all the muscles.

In some cases tremor opens the scene, and attracts so much notice as to constitute a defaced form of Basedow’s disease. On closer inspection, however, we usually find other symptoms, such as tachycardia, palpitation, peculiar look, pulsation of the carotids, diarrhoea, rapid wasting, diminished electrical resistance, etc. (Vigouroux). Although these symptoms may only be present to a partial extent, they assist in forming a diagnosis. The syndrome may be reconstructed from the recognition of some one symptom. Hence we are not liable to confound this tremor with that due to alcoholism, mercurial poisoning, neurasthenia, or morphinomania. Lastly, we may add that the tremor in Basedow’s disease shows its special tracing (Marie), and gives about eight or nine oscillations a second.



**Paralytic troubles** are manifold (Ballet). Without mentioning external ophthalmoplegia, the paralyses of Basedow's disease may show the most varied forms—monoplegia, hemiplegia (Teissier), weakness of the upper limbs (Dreyfus-Brisac), diplegia, paralysis of the nuchal muscles (Chwostek), paresis of the lower limbs (Heyden), and complete paraplegia (Charcot).

These paralyses, whether slight and transient or severe and prolonged, have been set down to hysteria. As hysteria is sometimes associated with exophthalmic goitre, it is reasonable to admit that paralyses which are hysterical in nature may coexist with Basedow's disease. Due allowance, however, being made for the possible paralyses of hysteria, which, moreover, have their own characteristics, certain paralyses are inherent to Basedow's disease, without the need for invoking the intervention of another factor. Thus, my patient, who showed no signs of hysteria, was a perfect example of Basedow's paralysis.

As regards the upper limbs, the paralysis diminishes or abolishes the functions of the hands. My patient could neither seize an object nor hold it. She was incapable of dressing herself, or of feeding herself, so that for some time she had to be fed by others.

Paraplegia, varying in degree from paresis to total paralysis, is very common in Basedow's disease. Its prominent features have been given by Chevalier: "In the upright position, or in walking, although the patient experiences no feeling of vertigo, the limbs give way and bend suddenly. Sometimes during a walk he may fall forward upon his knees. At other times, though more rarely, the motor weakness takes months to become complete, as in a case quoted by Charcot, where the impossibility of maintaining the upright position and of walking lasted nearly a whole year. These paraplegic troubles do not remain stationary; they improve from time to time and grow worse again, and this relapse, which is followed by improvement, shows a certain analogy with the evolution of the disease. In the intervals the legs frequently collapse while walking, so that crutches may be needed."

If we do not understand these cases, the idea of paraplegia consecutive to myelitis at once enters our mind; but we should not confound the paraplegia of Basedow's disease with myelitis, for in the former case bladder troubles are absent, the sphincters are intact, and we see no trophic troubles, no bed-sores on the sacrum. Moreover, we should not confound this paraplegia with that due to hysteria, because, in addition to the numerous stigmata of hysteria, such as hemianæsthesia, narrowing of the visual field, abolition of the pharyngeal and ovarian reflexes, hysterogenous zones, etc., hysterical paraplegia is soft, and accompanied by anæsthesia of the paralyzed parts, with complete loss of the muscular sense, while the muscles do not present the diminution of electrical resistance seen in Basedow's disease.

**Choreiform movements** complete the triad of motor troubles in Basedow's disease. They were very marked in my patient at the Hôtel-Dieu, and a cursory examination revealed choreiform movements in the hands, arms, shoulders, trunk, neck, and face. It has been asked whether chorea and Basedow's disease are two associated maladies, or whether Basedow's disease may not be capable of causing choreiform movements, just as it causes tremors and paralysis. In 1864 L. Grös, comparing exophthalmic goitre with chorea, described a case of Basedow's disease, with **choreiform** movements which affected the upper and lower limbs, the neck, and the face.

In 1876, at the Congress of Clermont-Ferrand, Gagnon, while studying the relations between exophthalmic goitre and chorea, quoted the case of a neurotic young girl who never had rheumatism. For a month this child had wasted, her character had changed, and she had palpitation. This picture marked the onset of Basedow's disease. At this time the pulse-rate was 130, and hypertrophy of the thyroid body was evident. The disease followed its course, and two years later she showed **choreic** movements, which finally became general.

In 1881 Guéneau de Mussy published a case of a young girl suffering from Basedow's disease, with **choreiform** movements. For some weeks the locomotor functions had been so affected that the patient could not walk. She used to make some regular steps forwards or backwards, showing that the muscular power was much enfeebled, in addition to the incoherence of her movements. The cerebral functions were not spared. The young girl was strange, her memory was not reliable, she had troubled dreams, and even delirium. At the end of eight or ten months the **choreiform** and **paralytic** troubles disappeared, as well as the mental aberration.

Raymond and Sérieux, in a paper on Basedow's disease and mental degeneration, found in one patient permanent **choreiform** spasms of the external oblique muscle. Deléage, in 1894, communicated to the Société des Sciences Médicales of Gannat a case of exophthalmic goitre with tremors, paralytic troubles, and **choreiform** movements.

Is it a question of choreiform movements or of true chorea in these patients? Köhler is inclined to look on this symptom as a choreiform motor trouble; Dach states his opinion that it is only met with in children, which is wrong; and Möbius thinks that it is a question of chorea supervening as an accidental complication. For my part, I cannot compare this condition with Sydenham's chorea. I believe that it is only a question of choreiform movements, and my reasons for this opinion are as follows: If exophthalmic goitre and chorea are indeed two associated diseases, as some authors think, why does Basedow's disease always precede chorea? We do not see exophthalmic goitre during the course or during the decline of true chorea. Hundreds of children who are affected by chorea never develop exophthalmic goitre. I think, therefore, that choreiform movements are a part of Basedow's disease, just as are tremors and paralysis.

The choreiform movements, with the tremor and paralysis, constitute a **triad** of motor troubles. The triad may be incomplete or complete, and we can understand how great is the disturbance in a muscular system affected at the same time by paresis, tremor, and choreiform movements.



5. **Other Nervous Troubles.**—The **sensory** troubles in Basedow's disease consist in trigeminal neuralgia, intercostal neuralgia, rachialgia, and hemianæsthesia, which is usually associated with hysteria.

In most patients **vasomotor**, **trophic**, and **secretory** troubles are seen. Albuminuria, glycosuria, and polyuria, which indicate disturbance in the bulb, have been noted.

Many patients complain of an exaggerated **sensation of heat**. They open the windows, and often complain of being too heavily clad (Basedow, Teissier). This increase in temperature is appreciable with the thermometer, and sometimes reaches one degree higher than normal.

Changes in the skin, sweating, purpura, pigmented patches, discrete or confluent **vitiligo**, alopecia, and chronic **urticaria** have often been observed. I have frequently seen pigmentation of the neck, shoulders, and arms, in the form of more or less large and discrete patches. **Œdema** is frequent, and chiefly affects the lower limbs. Trousseau has seen hypertrophy of the breasts.

We may also find the following series of symptoms :

Dyspnœa, which may be associated with palpitation, is frequent. It may be continuous, intermittent, or paroxysmal, and is the chief symptom in some cases. It is probably due to changes in the nucleus of the vagus.

The digestive functions suffer in Basedow's disease. I have found hypertrophy of the liver and jaundice; boulimia succeeds anorexia, and violent pulsations are seen in the epigastric angle. We may see vomiting and diarrhœa in the form of crises lasting one or more days. In spite of the increased appetite, patients waste and pass into a condition of exophthalmic cachexia. The general wasting contrasts in a singular manner with the exaggerated development of the eyes and neck. Wasting may be the first apparent symptom. I have seen a patient who rapidly lost over a stone in weight; wasting and tachycardia were here the only appreciable symptoms for some months. At first sight such wasting leads us to think of tuberculosis or of diabetes, and, as glycosuria is often present, the diagnosis must be carefully considered in order to avoid mistakes.

**Menstruation** is nearly always affected. It may be irregular or suppressed, and the amenorrhœa is often accompanied by leucorrhœa. Re-establishment of the menstrual functions is one of the most favourable signs in prognosis. Pregnancy has sometimes a beneficial effect. In men impotence is usually associated with Basedow's disease.

Hæmorrhages are sometimes seen. I have observed purpura, hæmoptysis, and retinal hæmorrhages in the same patient. Cerebral hæmorrhage and death from apoplexy have been noted. I saw one case of this nature with Jaccoud.

Vigouroux and Köhler have noted diminished resistance to electric currents.

In some cases Basedow's disease and myxœdema have been associated; no antagonism exists between them.

**6. Psychological Troubles—Mental Condition.**—As a general rule, few patients escape psychological troubles. Trousseau was the first to describe them: "The changes of character are such that life becomes difficult for the entourage of these patients, who are irritable and exacting to such a degree that the disease forms their only excuse. In addition to these changes in character we may note insomnia—a cruel complication which reduces patients to extreme despair." Trousseau mentions psychological troubles in most of his cases, and assigns such importance to them that he gives them a prominent place in his wonderful description of exophthalmic goitre. All authors who have studied exophthalmic goitre have laid stress upon the psychological troubles. "In nearly all patients suffering from exophthalmic goitre," says Ball, "there exists a certain degree of exaltation. They nearly all have strange ideas, and these morbid manifestations may end in the most acute mania." According to Joffroy, "the patient is restless, often a prey to exaggerated activity, and yet incapable of methodical work or of prolonged mental effort. Among the first changes which exophthalmic goitre imprints upon the character of patients, we find in some cases depression; or in others excitation." Boéteau thus describes the psychological troubles: "The chief symptom is profound melancholy, which more and more enters into every one of the patient's thoughts, so that he entertains ideas of suicide. At the same time the victims become impatient, surly, and remarkably emotional. Their will-power, when not quite absent, is often very feeble; they are incapable of fixing their attention upon any subject, even for a minute; they cannot remember next day what they did the day before. They show complete indifference not only to what concerns them, but to everything which affects their family, even to those who are most dear to them; and this indifference may sometimes amount to aversion."

Psychical troubles may exist from the first—sometimes, indeed, as the initial symptom—so that the diagnosis remains in doubt until forcible beating of the heart, exophthalmos, goitre, tremors, etc., appear. The interpretation of the psychological troubles is not decided. Some authors consider them to be a direct part of the disease; others, on the contrary, look upon them as due to hysteria and neurasthenia. To those who make these psychological troubles subordinate to hysteria, we may answer that there are cases of exophthalmic goitre in which hysteria is absent, and cannot, therefore, be the cause. Neurasthenia appears to be open to the same objections. It is undeniable that individuals may suffer at once from exophthalmic goitre and from neurasthenia or hysteria, but this is no reason to bring in neurasthenia in every case.



It is perhaps more logical to admit that Basedow's disease, like chorea, awakens psychical troubles in predisposed persons. The psychical troubles of chorea show very great analogy with those of Basedow's disease. We find the same intellectual depression, the same inaptitude for work, lapses of memory, changes in character, melancholy, indifference, and irascibility. Must we say that neurasthenia or hysteria has for some weeks been associated with chorea? I think not. We must say (omitting cases in which the association does exist) that chorea can extend to the whole cerebro-spinal system, and excite slight or severe psychical troubles. The same reasoning applies to Basedow's disease. Are we to say that, at the onset, neurasthenia comes to our rescue by bringing in its contingent of psychical troubles? I think not. We must say that Basedow's disease can extend to the whole nervous system, and excite psychical troubles just as it excites nervous troubles of every kind. Further, as Toulouse has remarked, "neurasthenia has succeeded in donning the mental troubles which formerly belonged to exophthalmic goitre alone."

These psychical troubles are usually transitory, and tend to recover. I shall now deal with certain serious mental troubles or true psychoses "running crescendo through the whole gamut from transitory or fugitive delirium to inveterate mania" (Boéteau). These psychoses include acute and chronic mania; delirium with ideas of persecution, melancholia, irresistible impulses, and apprehensive madness, and have been repeatedly discussed (Raymond and Sérieux, Joffroy).

#### Résumé of cases :

Young woman, without nervous antecedents, taken ill with acute mania during Basedow's disease. She became violent and refused all food; excitation gave way to depression; her mental condition improved, but she died from cachexia.—A young woman, without personal or hereditary antecedents, had Basedow's disease complicated by melancholia. She refused food, tried to throw herself out of the window, and suffered from delusions. She recovered some months later.—A woman, suffering from typical exophthalmic goitre, was seized with irresistible impulses and delirium. She felt an impulse to kill her children, but she was able to resist the impulse, though she feared the greatest misfortunes. Her insomnia was persistent, and her distress was intolerable. A year later, however, recovery was complete.—My patient, with exophthalmic goitre, had hallucinations of sight and hearing, was haunted with ideas of persecution, and imagined that people hid in her room in order to kill her.

The psychosis in Basedow's disease, therefore, may assume very different forms. Difficulties begin when we have to interpret its intimate nature. Some authors say that these mental troubles form an integral part of the disease. Whether the cerebral symptoms are slight, as is usually the case, or whether they are severe, with hallucinations, impulses, maniacal excitement, or delirium of every kind, it is none the less true that they are symptoms of a cerebral nature, and form part of Basedow's disease, in the same way as the other nervous troubles. This conception of Basedow's

disease makes it a morbid entity, a neurosis, or a neuropsychosis, in which every part of the nervous system is more or less affected.

Other authors enunciate a different opinion and divide up the disease. They say that the mental troubles, melancholia, delirium, or delusions—in short, every form of mental alienation—must be set down as psychoses which do not spring directly from the disease, but are associated with it, just as the same authors would associate hysteria and neurasthenia with it. These morbid conditions which evolve in the same patient are not, therefore, hybrids in the true sense of the word (“in neuropathology,” says Charcot, “hybrids do not exist”), but are associated, each association preserving its autonomy, characters, degree of gravity, and therapeutic indications. This grouping of morbid conditions, neuroses, and psychoses, is said to have heredity as its cause.

This opinion is seductive, although it is far from agreeing with the facts of heredity, and though it forms an incomplete explanation of the fact that heredity is sometimes direct, sometimes intermittent, and that there may be heredity of transformation, and also homologous heredity. According to some neuropathologists, persons affected with psychoses in Basedow's disease are really degenerates. “In many cases,” say Raymond and Sérieux, “Basedow's disease may be only a special localization of functional troubles which supervene in degenerates, or in predisposed persons, in some part of the cerebro-spinal axis. Magnan's theory of this failure of equilibrium in the centres of the cortex or of the spinal cord (paralysis, or erethism of these centres) may be applied to these different manifestations.

Toulouse, in a critical review of the relations between exophthalmic goitre and mental alienation, discusses this question, and shows us the defects of too hasty conclusions. “At this time,” says he, “the tendency evident in psychiatry, thanks to the works of Morel and Magnan, is to refer mental diseases to some distant hereditary cause, and to overlook the closer causes. This psychopathic predisposition cannot be denied, but it is too general an idea, although it forms the basis of all mental alienation. The hypothesis of predisposition does not suffice to explain the appearance of mental disorders in exophthalmic goitre, as in other maladies in which psychoses appear.”

The discussion, therefore, takes this form. There is a question of fact and of theory. Clinical facts show that many persons suffering from Basedow's disease, especially women, may have psychical troubles and mental disorders as well as motor troubles (tremor, paralysis, choreiform movements) and nervous troubles of every kind (crises of diarrhoea, sweating, albuminuria, glycosuria, and visceral congestions), all of which depend upon the nervous system. Is it necessary to isolate these groups and claim for them an autonomous or hereditary origin in every case, independent of Basedow's



disease? I do not think that we need go so far. The question of soil and of acquired or hereditary disposition play, not only in neuropathology, but in the whole domain of pathology, too large a part for me to attempt to belittle their importance. I think, on the contrary, that heredity in all its forms is the conducting wire which helps us to assign things to their proper place; but it does not furnish sufficient reason for cutting up a morbid entity and disjoining the pieces. Here, as elsewhere, I repeat, acquired or hereditary predisposition plays a leading part in the production of psychical symptoms and mental troubles. These cerebral troubles, however, have undoubtedly arisen in persons who, neither in personal nor hereditary antecedents, have had anything which could explain the appearance of a psychosis apart from their Basedow's disease. Sometimes psychical troubles and mental disorders begin with this disease, increase during its paroxysms, and then disappear with the other symptoms, or even before them. There is, indeed, under these conditions, such affinity or agreement in the evolution of the different symptoms which comprise Basedow's disease, that it is very difficult in this case to see only the coupling of morbid conditions, and it appears to me more reasonable to admit the development of a series of nervous troubles of various kinds, assuming the form of neuroses or of psychoses, but having indeed a common origin. However this may be, the appearance of mental troubles is always of ill omen.

**Course—Termination.**—Basedow's disease is rarely quite typical. One of the cardinal symptoms may be absent or slight, while other nervous troubles or symptoms are prominent. Sometimes the disease is more or less abnormal in appearance.

The **course** is usually slow and progressive, and the disease may last for ten or twelve years, or even more. In some cases it runs an acute course. The symptoms follow one another rapidly, and the clinical picture is complete in a few weeks—indeed, it may be so in the space of twenty-four hours. My patient at the Hôtel-Dieu, after terrible emotion, developed in a single night, tremor, paralysis, exophthalmos, hypertrophy of the thyroid gland and cardiac palpitation, and the mental condition appeared later. Trousseau quotes the following typical example of this **sudden** onset:

A woman lost her father. She was much upset, and cried during the night. Next day she suddenly felt that her eyes swelled and the eyelids became raised, while the thyroid gland enlarged and showed unusual pulsation. She also felt violent palpitation of the heart. Four days later she came and consulted Desmares, who found exophthalmic cachexia.

Terrible **paroxysms**, described by Trousseau, may supervene in the course of the disease. After some prodromata, or, indeed, suddenly, the patient has an acute attack of dyspnoea; the thyroid gland becomes enlarged; palpitation is severe; the eyes start from their sockets; the face is bathed in

sweat; the vessels in the neck show rapid pulsation; the dyspnœa is accompanied by stridor and sucking-in, and death sometimes appears imminent. Quiet is, however, re-established, and the paroxysm ends. Acute paroxysms are much more serious than chronic ones. In a case quoted by Trousseau the patient was seized with such terrible attacks of suffocation that preparations were made for tracheotomy. The paroxysms may only recur at intervals of months or years. They show infinite variation in their duration and gravity, and may even reappear every month or every few days.

Exophthalmic goitre is a grave malady, for the mortality amounts to 20 per cent. Death results from cachexia or from intercurrent trouble, such as paroxysms, pulmonary hæmorrhage, intestinal and cerebral hæmorrhage (Hirsch), multiple gangrene, mental alienation, angina pectoris, or pulmonary tuberculosis.

**Ætiology.**—Exophthalmic goitre is a disease of the middle period of life. It is much more frequent in women than in men, and is often associated with a nervous temperament, hysteria, epilepsy, chorea, mental alienation, diabetes, chlorosis, or pathological conditions of the genital system. It may appear after moral or physical shock, accident, emotion, fright, violent anger, or injury (De Graefe). Pregnancy has sometimes a beneficial influence on the progress of the disease, but, on the other hand, the goitre may appear during pregnancy.

**Heredity** plays the chief part in its ætiology, and the parents may themselves be sufferers from goitre, or from one of the nervous diseases above quoted.

As regards direct heredity, I do not know a more interesting or conclusive case than that of the family of Les —, natives of a place near Soissons, where goitre is endemic. I have seen several members of this family, which in three generations has furnished six cases of Basedow's disease.

I could quote cases in which the children of parents suffering from Basedow's disease were born with a goitre. For many years the goitre remained the only sign of the disease, and ten or fifteen years later other symptoms, such as exophthalmos and palpitations, appeared as the result of acute emotion, menstruation, or pregnancy.

A more singular fact is that, in countries where goitre is endemic, we see goitre in persons who are not descended from parents affected with Basedow's disease, and yet at some period of their lives exophthalmos and tachycardia appear in addition to the goitre.

**Diagnosis.**—At first the diagnosis is very difficult, especially when the disease is abnormal, because we may only have a single symptom. Mistakes are rare when the chief symptom is goitre or exophthalmos; but when the patient only complains of pulsation, tremor, rapid wasting, or attacks of dyspnœa, we must be able to reconstruct the disease by grouping certain



symptoms, or traces of the more or less important symptoms above enumerated.

In women the general symptoms may simulate chlorosis, but the acceleration of the pulse, the peculiar look, the prominent eyes, the tremor of the hands and feet, the pigmented spots, the pulsation of the cervical vessels, and the slight swelling of the thyroid body should indicate the diagnosis.

As the exophthalmos in Basedow's disease affects both eyes, and is not accompanied by squint, it shows no resemblance to unilateral exophthalmos of orbital or of cranial origin. The prominence of the eyeball in myopic patients will be readily distinguished.

The origin, appearance, and progress of exophthalmic goitre do not allow confusion with goitre, properly so called. Many cases will call for a minute inquiry into the symptoms present.

**Nature of the Disease.**—Exophthalmic goitre is a cardio-vascular neurosis, resulting doubtless "from profound disturbance in the vasomotor nerves" (Trousseau). It is a neurosis of bulbar origin (Sée).

We may, I think, be satisfied with Ballet's conclusions: The possible association of external ophthalmoplegia, of paralysis of the facial, the hypoglossal or the motor branch of the trigeminal nerves, with exophthalmic goitre forms an argument in favour of the theory which refers Basedow's disease to some trouble in the central nervous system, and particularly in the bulb.

The common troubles of the disease depend on nuclear paralysis of the vagus, which causes tachycardia, dyspnœa, and gastric troubles; and on paralysis of the vasomotor centres, which gives rise to flushing of the face and neck. The goitre and the exophthalmos result from the coexistence of vasomotor paralysis and tachycardia. These paralyses do not depend upon a material lesion, but are simply functional troubles, capable of improvement, aggravation, recovery, or relapse.

It follows from the preceding statements that Basedow's disease is especially a bulbar neurosis. Troubles of medullary or of cortical origin may also appear, and therefore Basedow's disease is often associated with other neuroses, such as unsoundness of mind, epilepsy, and especially hysteria.

**Treatment.**—I will give here the treatment of Basedow's disease described in my lectures at the Faculté. Let us consider this treatment from two different points of view—(1) during the paroxysms, and (2) during the course of the disease.

In dealing with an acute paroxysm, in which the thyroid and cardiac trouble are most severe, we must at once act because the paroxysm will last for half an hour or more, and may cause death, if we do not intervene immediately. Distress and dyspnœa are extreme. The excessive swelling

of the thyroid body compresses the trachea, causing stridor and preventing the free passage of air. In such conditions tracheotomy appears imperative, and, indeed, it would be indicated but for the danger arising from the dilatation of the vessels of the neck, which are gorged with blood, and the fatal hæmorrhage which might result, as in a case quoted by Trousseau.

How, then, are we to relieve the thyroid and cardiac troubles? The first indication is fulfilled by the application of ice-bags to the thyroid until the crisis disappears. We should treat the cardiac excitation by digitalis, in large doses, as advised by Trousseau—*e.g.*, 2 grains of the dried leaves in powder every half-hour for two or three hours, according to the severity and duration of the attack, while an ice-bag is applied to the precordial region.

If this gives no relief, we must resort to bleeding, to the application of leeches to the neck, and to careful inhalations of ether or of chloroform. The paroxysm ends more or less rapidly, and the patient is for the time being saved.

We must, however, not forget that the disease remains, that it is dangerous in these cases, that other crises will appear, and that one or other of them will be fatal. Therefore we must be on our guard, and ready to act in a similar manner. It is, above all, necessary to prevent them—that is, to direct our attention to the disease itself. The physician must adopt the same course, although the patient has shown no paroxysms, because great improvement in the symptoms can be effected by rational and well-directed treatment.

Many drugs have been extolled in exophthalmic goitre, and the chief among these are iodine and the iodides. Cheadle prescribes tincture of iodine, taken internally. This drug is certainly efficacious in simple goitre, but renders no service in exophthalmic goitre. Trousseau has even given his opinion against this method of treatment, and most writers agree with his advice.

Bromides are indicated for the cardio-vascular excitation and erethism. Although this drug does not act directly on the disease, it at least has a favourable effect upon the symptoms, when given in large doses.

Extract of valerian, or valerianate of ammonia, produces manifest improvement in the palpitation and the dyspnœa.

All authors are unanimous in advising digitalis in the treatment of exophthalmic goitre. While it has the disadvantage of raising the blood-pressure, it has the advantage of slowing the heart-beat in severe tachycardia; therefore its disadvantages must be passed over, and we must only think of relieving the patient. The dose varies according to the susceptibility of the patient. In some patients 1 or 2 grains of the dried leaves will be sufficient, while in others it will be necessary to increase the dose, the



whole point being to estimate the susceptibility of the patient. Except in rare cases, digitalis is an excellent drug.

The use of belladonna has in some cases caused improvement of all the symptoms, except the hypertrophy of the thyroid gland.

Hydrotherapy, in spite of unfavourable criticism, is none the less an excellent method. The cold douche should not be given at first, but shower-baths at a temperature of 75° F. should be first given, and the temperature of the water should be gradually reduced.

Electricity is certainly very efficacious. The continuous current appears to have given the best results. The following procedure is usually adopted : Two rheophores are applied on either side of the neck over the superior cervical ganglion, then over the vagi, and a current of from 3 to 8 milli-ampères is allowed to flow for eight or ten minutes. A daily sitting is given for three to four weeks ; the treatment is then stopped and resumed at the end of a week. For some time past the faradic current has met with some credit (Vigouroux). This rapid survey of the different methods of treatment shows that digitalis, valerian, bromides, the continuous current, and hydrotherapy give the best results.

As regards treatment with thyroid extracts, opinions are much divided. Voisin has quoted a case in which sheep's thyroid gland gave excellent results. In answer to Voisin's communication, Dreyfus-Brisac and Bécclère declare that thyroid treatment for exophthalmic goitre has always, in their experience, increased the symptoms, and they are not alone in this opinion.

For my part, I have for a long time used *ipécacuanha*. The idea of giving *ipécacuanha* in Basedow's disease occurred to me on seeing the success obtained in hæmoptysis.

These patients have erethism of the cardio-vascular and vasculo-pulmonary systems. The pulse is hard and vibrating during the hæmoptysis. In such cases we give *ipécacuanha* in emetic doses if we desire to arrest abundant hæmoptysis, or in fractional doses so as to cause nausea if we are treating more chronic bleeding. The pulse diminishes in frequency and force, and the hæmoptysis improves or ceases. In Basedow's disease the treatment of cardio-vascular erethism is also one of the indications to be fulfilled.

I have therefore given *ipécacuanha* with digitalis and opium in pills :

|                               |    |    |    |                    |
|-------------------------------|----|----|----|--------------------|
| R Powdered <i>ipécacuanha</i> | .. | .. | .. | gr. $\frac{1}{12}$ |
| Powdered digitalis leaves     | .. | .. | .. | gr. $\frac{1}{30}$ |
| Extract of opium              | .. | .. | .. | gr. $\frac{1}{20}$ |

To make one pill. Take four pills at equal intervals in the twenty-four hours.

In giving *ipécacuanha* we should not cause vomiting, but only very slight nausea. We must then diminish the number of pills to three, or even

two, in the twenty-four hours, and gradually increase the dose up to the limit of tolerance. With ipecacuanha I am fond of giving valerianate of ammonia in a daily dose of 2 or 3 teaspoonfuls, and I also employ hydrotherapy.

I have treated many cases of exophthalmic goitre in this manner, and marked improvement, especially in the dyspnœa, has been the rule. It was particularly striking in two cases, and I do not think that any other method of treatment would have given a better result. The effect of this treatment is shown by the appreciable improvement after a few days, and very marked benefit after some months. The only inconvenience of this treatment is diarrhœa, which disappears as soon as tolerance commences.

**Surgical Treatment.**—Surgical treatment consists in operations upon the goitre or upon the cervical sympathetic nerves. Let us first consider operations upon the goitre. The idea of partial or total removal of the gland or of enucleation by drawing it out of the wound and allowing it to waste (exothyropexia) arises from the somewhat erroneous conception that the hyperthyroidization is the chief cause of the symptoms and complications of Basedow's disease.

According to this theory, the goitre produces too much secretion, poisons the nerve centres, and produces auto-intoxication from functional hyperactivity. This theory, to be accepted, should be applicable to every case. We find, however, nothing of the kind. The exophthalmos, tachycardia, etc., may be already marked, while the goitre is absent or slight. These defaced forms are far from rare, and we cannot, under such conditions, invoke primary hyperactivity of the thyroid gland. Abadie has quoted cases in which the exophthalmos was so marked that complete loss of both eyes had resulted, while the enlargement of the thyroid body was scarcely appreciable.

In order to appreciate the question better, let us consider the operations upon the thyroid body and their results. Allen Starr gives statistics of 190 cases of thyroidectomy in Basedow's disease, with the following results: Recovery in 74, improvement in 45, failure in 3, and death in 33 cases. As a rule death was not expected, and occurred soon after the operation, or two or three days later. Whatever theory be used to explain the deaths, the patients succumbed with nervous symptoms—viz., excessive tachycardia (pulse-rate 180 to 200), sudden rise of temperature, angina, restlessness, profuse sweating, and collapse.

Brissaud published a case in which Poncet performed exothyropexia, which is a benign operation, and consists in exposing the thyroid gland. The patient died without any warning.

Lejars witnessed the following accident:



A girl eighteen years of age had tachycardia, slight exophthalmos, tremor, etc. The goitre was very moderate; the right lobe of the thyroid body appeared to be the larger. It was firm, could be depressed, and showed neither nodules nor induration. The right lobe was removed *en masse*, after freeing the upper and lower cornua and ligaturing the vessels. The operation was satisfactory, and the patient rested well during the day. About eleven o'clock at night she was suddenly seized with intense dyspnœa and considerable acceleration of the respiratory movements. No flushing of the face, no asphyxia. She died in three-quarters of an hour. The autopsy revealed no operative lesion; the recurrent, vagus, and sympathetic nerves, with their cervical branches, were absolutely intact. The fact, says Lejars, remains that a simple operation, performed without mishap, was followed by sudden death, which could not be explained at the autopsy, but appeared to be related to acute bulbar trouble.

Jaboulay, in one of his cases, adds :

Diminution of the mass remaining after partial extirpation of the goitre usually follows when the case is one of ordinary goitre. Section of the isthmus, for example, may cause atrophy of both the hypertrophied lobes, and unilateral thyroidectomy produces diminution in the size of the remaining lobe. I have seen the opposite result in a case of Basedow's disease. Acting on the theory, which subordinates the other symptoms to perversion of the secretion, I have several times operated upon her thyroid gland, and have left it exposed on two consecutive occasions. As each intervention was only followed by temporary improvement, I decided last year to remove the right lobe. Three months later the left lobe was enlarged, and quite recently the middle lobe formed a goitre as large as a small orange. After each operation upon the thyroid itself the symptoms showed genuine improvement, and the tremor in particular speedily disappeared; but relapses occurred, with palpitation, tremor, and fresh goitre.

Poncet sums up the matter thus :

After various operations which I have performed in exophthalmic goitre, including simple exothyropexia, I have seen a fatal result. Such results, and also the frequent return of symptoms which had yielded for a very short while before the operation, have made me very circumspect. For my part, I shall not again meddle with the thyroid body in a case of Basedow's disease.

It must be admitted that the results just quoted are hardly encouraging, but for a correct decision let us also take into account the successes obtained. Tillaux has reported a case of cure :

At the Société de Chirurgie, Tuffier presented two young women suffering from exophthalmic goitre, both treated by partial thyroidectomy, involving the right lobe and the isthmus. The first patient had been under medical treatment for two years at the Salpêtrière, with no result. After thyroidectomy the exophthalmos and the nervous troubles were cured. Later the tachycardia and the tremor ceased, so that two and a half years after the operation the patient was in good health. The left lobe of the thyroid gland remained larger than normal, but had not increased in size since the operation.

The second patient was a young woman whose condition was very grave when Tuffier operated. She had taken tabloids of thyroid gland for six months, instead of tabloids of thymus glands. Symptoms of acute thyroidism, which demanded removal of the gland, appeared. Before the operation her condition was very grave: Enormous exophthalmos, violent dyspnœa, incessant palpitation, pulse 144, thyroid gland double its normal size and showing marked pulsation, insomnia, tremor, œdema of the lower limbs, and profuse diarrhœa. The results of the operation were remarkable. Next day the erethism of the vessels disappeared, the diarrhœa ceased, and the

pulse fell from 125 to 72. Condition quite satisfactory a month after operation. Exophthalmos still present, although less, tremor cured, no trace of dyspnoea, sleep good, no nervous symptoms. The future will show the ultimate worth of the operation.

In spite of the successes above set down, as well as two other cases reported by Doyen, it is none the less true that operations in exophthalmic goitre expose the patient to very grave danger.

We may therefore form a judgment upon the advantages or disadvantages of such operations in Basedow's disease. It seems, however, well established that the disadvantages outweigh the advantages. Operation, even when performed under the most favourable conditions, does not protect the patient from very grave risks, nor the surgeon from terrible surprises. The results obtained are temporary or incomplete. For these reasons I should be very loth to advise this operation.

Another operation—viz., double section of the great cervical sympathetic—was first performed by Jaboulay. Trousseau had seen that the chief symptoms and vascular troubles in Basedow's disease do not extend beyond the area of the cervical sympathetic. The carotid arteries, with the thyroid vessels and their branches, are alone affected by the expansive pulsation, while the other vessels, such as the radial, the femoral, or the abdominal aorta, beat normally. Accordingly, the great sympathetic nerve has been considered as the cause, and theories, some based upon excitation, others upon paralysis of its branches, have been brought forward. None of these theories, however, were applicable to all the symptoms. The brilliant discovery of Dastre and Morat has thrown much light upon this question. These physiologists showed that the vasodilator fibres of the cervical sympathetic have a distinct origin, and upon this fact Abadie has built up an ingenious theory.

Be this as it may, it is none the less true that double section of the great sympathetic nerve has given remarkable results. Out of six cases published by Jaboulay, one is absolutely characteristic :

A woman fifty-five years of age had for three months suffered from exophthalmos, tachycardia, and tremor. Hypertrophy of the thyroid gland was absent. She had gained no benefit from thyroid extract. When she came under Jaboulay's care, "her look was indeed alarming from the exophthalmos, which had almost dislocated the left eye from the orbit, and the tremor was excessive. She suffered from tachycardia and from such intense dyspnoea that the possibility of surgical intervention was discussed." Double ablation of the superior cervical ganglion was performed. The result was immediate. Three days later her face had lost its terrible expression, while the eyes, and the left eye in particular, had almost regained their normal position. Tremor, dyspnoea, and precordial pain completely disappeared, but the tachycardia persisted, and the pulse varied between 100 and 120.

In his communication of October 21, 1896, at the Congrès de Chirurgie, Jonnesco (of Bucharest) brought forward two cases of exophthalmic goitre, treated by double resection of the cervical sympathetic.



Reclus and Faure's case is comparable to that of Jaboulay. The result of the operation was remarkable, but it is necessary to see, as Reclus says, what will become of the patient, for it may be only a case of transient improvement.

The following case was published by Gérard-Marchant and Abadie :

A young woman suffered from a defaced form of Basedow's disease, characterized chiefly by exophthalmos. Tachycardia absent ; thyroid body showed very slight hypertrophy of the right lobe ; exorbitism was so marked that the lids did not cover the eye. Gérard-Marchant performed resection of both cervical sympathetic nerves. The exophthalmos gradually diminished, but did not quite disappear. "This result," say the authors, "was not maintained. Under the influence of emotion and fatigue, exophthalmos reappeared, although in a less degree than before the operation. It is right to add that there is no trace of goitre, and that her physical and moral condition has remained excellent."

Chauffard and Quénu have published the following case :

A man, aged twenty-five, admitted with the classical symptoms of Basedow's disease. Eyes very prominent, complete closure of the eyelids impossible. Goitre bilateral and pulsatile. Heart-beats very forcible, and rate 110 a minute ; dyspnoea and palpitation on the least effort. Carotids showed marked pulsation. Marked tremor of the hands and limbs. Patient irritable, unstable, and showed stigmata of hysteria. Quénu performed bilateral resection of the cervical sympathetic. After the operation, aggravation of the tachycardia and appearance of arrhythmia, which did not exist before, became manifest. Some days later heart showed a rate of 110. As regards the heart the benefit has therefore been nil, the goitre has been slightly affected, and the circumference of the neck, which before the operation was 38 centimetres, varies from 36½ to 37 centimetres, since the operation. The eyes may be less prominent, but this is very doubtful. In short, the benefit obtained has been nil. We may, indeed, ask whether the patient has not rather suffered than profited by operation, since in two months he has lost 6 pounds in weight.

In short, the results obtained by bilateral resection of the cervical sympathetic are so far too much at variance to yield definite information as to the value of this intervention. Besides the truly remarkable results (Jaboulay and Reclus' cases) there are others in which the good results are doubtful (Gérard-Marchant and Abadie's cases) or absent (Chauffard and Quénu's case).

It is therefore impossible at present to give surgical treatment the preference over medical means. Both of them show defects and are often ineffectual, but medical treatment at least does not endanger the patient's life (I allude to operations on the goitre), and may result in recovery.

## CHAPTER V

### DISEASES OF THE VESSELS

#### I. PHLEBITIS—PHLEGMASIA ALBA DOLENS.

**Phlebitis** is inflammation of a vein. This question is in part surgical; thus phlebitis consecutive to injury of veins, operations, inflammation, etc., is of traumatic or external origin. This variety was thoroughly described by Hunter (1795), Ribes (1816), and Dance (1828), who divided the condition into suppurative and adhesive phlebitis. This side of the question does not concern us, and, further, it is no longer of interest, since **aseptic procedures** are employed in surgery. The phlebitis which now occupies our attention arises from internal causes, and infectious phlebitis figures in the first rank. Sometimes the deep tissue of organs is the seat of phlebitis, at other times the mischief develops in the veins of the trunk and the limbs. Phlebitis may pass unnoticed when deeply hidden in the splanchnic cavities, or show itself by symptoms depending upon its localization (portal vein, sinuses of the meninges); at other times it produces embolisms described under pulmonary embolisms.

When phlebitis affects the veins of the limbs, it shows itself as **phlegmasia alba dolens**. The word phlegmasia is derived from *φλέγμα*, *φλέγματος*, which in old-time medicine signifies phlegm (œdema). The literal translation of phlegmasia alba dolens is painful white œdema. I shall here describe the different kinds of phlebitis and phlegmasia alba dolens.

**Pathological Anatomy.**—We have to study a double lesion: the one affecting the walls of the vein, or **phlebitis**; the other affecting the blood, which forms a clot inside the vein—*i.e.*, the **obturator clot** or **thrombus**. The name **thrombosis** is given to the process which ends in obliteration of the vein by a thrombus.

**1. Phlebitis—Mechanism of Thrombosis.**—A vein, when affected by phlebitis, is often obliterated by a blood-clot. Ancient theories considered phlebitis the initial phenomenon, and thrombosis the consecutive one. Later, at the instigation of Virchow, an entirely opposite theory has been admitted: thrombosis is the initial phenomenon, and phlebitis the secondary



one. In spite of the infatuation for German researches, Vulpian fought against the too exclusive doctrine of Virchow, and taught that the coagulation of blood in the veins is preceded by a change in the vascular epithelium.

Modern researches prove that Vulpian was right. In some cases the primary lesion of the vein is self-evident (varices, injury); in other cases it is at first sight less apparent. What do histology and bacteriology teach us? In the great majority of cases phlebitis, like endocarditis, is due to microbic agents. Widal has found the streptococcus of puerperal infection in the clots of phlegmasia. Bacteriological examination must be made with material from the uterine veins, because coagulation starts in them, and gradually reaches the femoral veins, although we may not be able to find any trace of characteristic streptococci in the clot which obliterates them. Chantemesse and Vaquez have found Koch's bacillus in phlebitis among tubercular patients. More often in the lesions of phlebitis we find not one specific bacillus, but the ordinary micro-organisms of supuration—viz., staphylococci or streptococci. In the latter case phlebitis supervenes as a secondary infection. In this manner the cases of phlebitis in influenza, typhoid, cancer, tuberculosis, cachexia, and possibly also chlorosis, are explained. The microbic agents are often present in the vessel wall and in the clot. Whether these agents penetrate the walls of the vein by the *vaso vasorum*, or whether the microbes circulating in the blood directly cause irritation of the vascular endothelium, matters little. The essential fact is that **phlebitis precedes thrombosis**.

How does thrombosis arise? Different interpretations have been given as to the formation of the thrombus: (1) An increase of the plasmin (spontaneously coagulable fibrin); (2) slowing of the blood-stream. Increase of plasmin is associated with cachectic conditions (cancer and phthisis), the puerperal state, and chlorosis. Slowing of the blood-stream is due to the pressure of aponeurotic laminae, situated in the course of certain veins (crural), to compression caused by the foetus, to degeneration of the heart muscle, and to cardiac paresis (Jaccoud) in severe fevers and cases of pyrexia. According to Hayem and Bizzorero, the hæmatoblasts, or blood-platelets, play a considerable part in the coagulation of the blood.

However this may be, the lesion commences with granulo-fatty degeneration of the endothelial cells; the tunica intima becomes thickened, and granular and stratified layers of fibrin are deposited upon this granular tissue. This is the origin of the thrombus. The fibrinous clot is adherent. "All attempts to break down this adhesion are useless, and it is difficult to delimit exactly the wall of the vein which undergoes progressive thickening. At the central point of the clot the wall presents a reddish bud, which arises from the deep tissue of the vein, and becomes lost in the

coagulum" (Vaquez). After these preparatory changes the partial or total obliteration of the vein by coagulation of the blood is readily understood.

**2. Thrombus.**—We may now consider the clot itself. The clot fills the cavity of the vein more or less, and may be parietal or obliterating. The portion in contact with the vein is whitish and very adherent—"the clot of pulsation" (Hayem)—while the portion which obliterates the vessel is reddish, "the clot of stasis." The central end of the clot sometimes ends in a tapering manner. This end, struck by the current of blood, or detached by the stream from a collateral vein, may become an **embolus**.

The thrombus may undergo one of the following changes: (1) Its elements may become disaggregated and disappear by absorption; (2) it may be invaded and broken up by vasculo-connective vegetations of the tunica intima, and the vein is sometimes converted into a cord of **cavernous** tissue; (3) the peripheral portions of the clot may alone undergo fibrous change and retract, so that the circulation is re-established through the centre; and (4) the thrombus may be broken up, and the detached fragment may cause **embolism** in the right heart or in the lung. The clot sometimes softens at its centre, and presents a puriform appearance which has been wrongly taken for suppuration, but is really a disintegration of the clot with granulo-fatty change. If the particles enter the blood-stream, they may cause **infarcts** in the lung; and if bacteria are present in the thrombus, the capillary embolisms are also septic, and lead to septic infarcts and miliary abscesses in the lungs or in the heart.

**Description.**—I shall give a general description of phlegmasia alba dolens, and return later to the peculiarities which each phlebitis may present, according to the cause which has given rise to it. Phlegmasia alba dolens may affect the veins of the upper and lower limbs, the neck and the face, but shows a remarkable predilection for the veins of the leg, and usually begins in the veins of the calf. It has an insidious onset, which is rarely febrile, and shows itself by pain, which is at first diffused through the whole limb, and later is localized in certain points. In the leg, which is the ordinary seat of the lesion, the pain is more marked in the calf and the groin; the limb is heavy and swollen, and the hyperæsthesia is at times excessive. In some cases the patient complains of joint pains, simulating rheumatism; in other cases pain may be completely absent. The bluish network under the skin shows that the circulation is impeded in the deep veins, and tends to re-establish itself by the superficial ones. The deep veins are sometimes transformed into a hard, tortuous cord, which may be felt in the calf and followed upwards, as far as the ring of the adductor magnus. The knee-joint is sometimes filled with fluid; the movements of the leg are difficult, as though the muscles were affected with paresis. Transient loss of power in the limb, with or without muscular atrophy, has also been noted.



The obliteration of the veins causes **œdema** which has special characters: white, because the skin is bloodless; smooth and hard, because the areolæ of the skin are distended by serous fluid; painful, from the compression of the nerve endings. It does not, therefore, resemble the œdema of cachexia, or of diseases of the heart and kidney. In some cases, however, it may be slight, or its absence may make an error in diagnosis easy. It may only appear days, or even weeks, after the pain. In some patients it is almost the only symptom of the lesion. The œdema often begins in the foot and leg, and later reaches up to the thigh. In puerperal phlegmasia, however, the œdema often begins at the root of the limb, and spreads from above downwards.

Phlegmasia has a variable duration. We see slight forms, as in a tubercular patient in whom the trouble only lasted about twelve days; as a rule, it lasts four or five weeks. Patients feel the effects of the disease for a long time. For months and years walking tires them, and severe exercise causes the œdema to reappear. The cellular tissue and the skin are often the seat of ill-defined induration.

Venous thrombosis *per se* very rarely causes gangrene, which more usually supervenes in the course of phlegmasia, because the inflammation extends from the vein to an artery.

In rare cases the clot breaks up, and is carried away by the blood-current; the thrombus becomes an embolus, which gets stranded in the right heart, or passes through into the lung. Disastrous complications are the result: cardiac embolism may produce fatal syncope, while pulmonary embolism, according to its extent, produces rapidly fatal asphyxia, attacks of dyspnoea, pulmonary gangrene, and mechanical or septic infarcts in the lung. These complications, described under Pulmonary Embolism, show how great may be the gravity of the prognosis.

It is of interest to know at what period embolism is most to be feared. It is rightly admitted that the accident in question is no longer to be feared six weeks after the appearance of phlebitis. The exceptions, however, must be reckoned with, as in the case quoted by Trousseau, where fatal embolism supervened three months after the onset of phlebitis.

The **diagnosis** is generally easy. It must not be forgotten, however, that pain and œdema, which are the chief symptoms, may be absent. Cases have been quoted in which the disease was almost latent, and yet the patients succumbed rapidly from pulmonary embolism. On the other hand, phlegmasia may be of value in diagnosis.

When we hesitate, for example, between ulcer and cancer of the stomach, the appearance of phlegmasia confirms the existence of cancer. Trousseau, who brought this fact so clearly to light, was able to apply the semeiological value of phlegmasia to his own case. The appearance of painful œdema

in the leg led him to diagnose the gastric cancer from which he died six months later.

**Varieties.**—After this general description, let us consider special cases :

1. In **puerperal women** phlebitis appears some days after accouchement, rarely later than two weeks. It is often ushered in by fever. Puerperal phlebitis is usually absent **when the accouchement has been normal**, and is generally seen only in cases in which the patient has shown some infectious phenomenon, foetid lochia, difficult or artificial delivery, slight fever, etc. In other cases, after accouchement or miscarriage, no sign of infection may occur, and yet phlegmasia develops three or four weeks later. Phlegmasia of puerperal origin improves after three or four weeks, but may last much longer. I cannot direct too much attention to a variety of phlebitis which also has a uterine origin, although it may not be puerperal. I allude to the cases of deep phlebitis or of phlegmasia alba dolens which supervene after operations for diseases of the ovaries and uterus, and especially fibroids.

2. Phlegmasia in **typhoid fever** usually supervenes during the decline of the disease or during convalescence. Its onset is often febrile. It may affect the veins of the upper limbs and the neck, and may be associated with arteritis.

3. Painful phlebitis occurs in *influenza*, and recent epidemics have provided material for its careful study. In the published cases the phlebitis affected the veins of the lower limbs or of the arm.

4. Phlegmasia is frequently associated with **tuberculosis**. It usually supervenes in the cachectic stage. In some cases, however, it may appear early.

5. I would make the same remark concerning cancer. In most cases phlegmasia supervenes in the stage of cachexia, but in other cases (Trousseau was himself a memorable example) it appears during the first stage of cancer, before other positive symptoms. It is therefore a typographical error which states that Trousseau diagnosed cancer of the stomach a month before his death, on the appearance of phlegmasia. It was **several months** before his death that the appearance of phlebitis in the calf led Trousseau to diagnose cancer, although no other positive signs of organic disease were present. I am able to state this fact because I saw the case.

6. Phlegmasia associated with **chlorosis** presents peculiar interest. Possibly this phlebitis, like so many other cases, is due to a superadded infection. Certainly phlegmasia of chlorotic origin is far from being rare, and in some cases has been followed by pulmonary embolism and death. In several cases death has supervened from phlebitis of the pulmonary veins.

7. Phlebitis has been noted in acute articular rheumatism and in pneu-



monia (Obrecht). The importance of gouty phlebitis will be considered under Gout.

8. Phlebitis of the limbs may occur in the course of blennorrhagia.

9. **Appendicular phlebitis** is not uncommon. I have seen it in a woman suffering from appendicitis, which was operated on later by Segond. The phlebitis usually affects the left leg. It may appear as an infectious complication, even though the appendicitis be slight.

**Treatment.**—In a patient suffering from phlegmasia we must avoid massage, and movement of every kind which may favour the displacement of a clot and the formation of an embolus.

The affected limb should be placed in a trough-splint in order to obtain complete immobility. The pains may be relieved by the following ointment:

Vaseline, 10 parts; methyl salicylate, 3 parts. Salt must be excluded from the diet. Since Vidal showed that the œdema in Bright's disease disappears by excluding salt, Chantemesse has applied this treatment successfully to phlegmasia in enteric fever. I have seen the good effects of milk diet in women suffering from post-operative phlebitis. The cure at Bagnols (Department of Orne) is of much service.

## II. SYPHILITIC PHLEBITIS.

**Description.**—Although syphilitic phlebitis may not appear till some one or two years after infection, it is usually a much earlier manifestation of the disease. In one of Roussy's cases it appeared five months after the chancre. In one of my cases it supervened two and a half months after the chancre. Thibierge has recorded phlebitis of the internal saphenous and cephalic veins two months after the chancre. Fournier noted its appearance in the internal saphenous, median basilic, and median cephalic veins six months after the primary sore. Phlebitis, therefore, may appear within a few weeks or months of the chancre; it may precede or coincide with the roseola and the mucous patches. Le Noir and Girdwood state that the skin rash may not appear until several days after the onset of superficial phlebitis. Fournier and Loeper have recorded the fact that in one case phlebitis appeared two days before the papules in the skin. The disease does not always begin in the same way. A sensation of weight, formication, or cramp may be noticed in the limb; on the other hand, prodromata may be absent, the pain is acute from the first, and œdema appears.

In one patient the phlebitis had a sudden onset. He was returning home when he felt acute pain in the left thigh, along the course of the saphenous vein, in the popliteal space, and in the leg. Next day the whole limb was œdematous. Mauriac's patient was in hospital when he felt a sharp pain in the right calf; phlebitis had just commenced. Charcot speaks

of a syphilitic patient who felt acute pain on the inner side of the left thigh; phlebitis had just begun in the internal saphenous vein.

Fever is exceptional or slight, and throughout the disease "the symptoms," as Fournier says, "begin like those of a subacute phlebitis, which almost at once becomes of the apyretic variety, in which the affection comprises simply a plastic induration of the venous trunk."

The veins of the leg are more often affected than those of the arm. We find, in order of frequency, the internal saphenous, the external saphenous, the basilic, and the cephalic veins.

The vein is not always involved along its whole course: the phlebitis is often partial. In one of Fournier's cases induration was noticeable over the internal malleolus, and did not appear again until above the knee, whence it extended to the groin.

This segmentation has often been noted in the median cephalic and basilic veins. It is also common in syphilitic arteritis, where healthy and diseased segments alternate.

We must note the multiplicity of phlebitis in the same patient. Other diseases besides syphilis cause multiple phlebitis, but this peculiarity is, I think, nowhere more marked than in syphilis. In thirty-six cases twenty-two patients had multiple phlebitis. In my patient both legs were affected. In Fournier's patient the left leg, the right leg and arm were involved. In Fournier and Loeper's case phlebitis was present in the left leg and in the right leg and arm.

Syphilitic phlebitis is fairly often symmetrical. It is prone to relapse. A patient who thinks he is cured has a fresh attack some weeks or months after the first attack.

Phlebitis is far more common in the superficial than in the deep veins. Mauriac, however, has seen a case of deep phlebitis with thrombosis. Andry and Constantin have seen phlebitis of the popliteal vein, resembling in every way phlegmasia alba dolens.

Pain and œdema are not as a rule as severe in the syphilitic as in the other forms of phlebitis. Nevertheless, when my patient was admitted to the Hôtel-Dieu, the œdema extended from the foot to the trunk.

Recovery occurs in a few weeks, and the veins regain their elasticity. This is the reason why specific phlebitis does not leave behind it, as the other forms do, interminable œdema, which reappears on the least provocation, and obstinately resists all treatment, including massage and the cure at Bagnoles. We must not, however, suppose that syphilitic phlebitis is always of short duration; in some cases the disease lasts five or six months, owing to the multiplicity of the lesions and to the relapses which occur.

But a capital difference, which would alone suffice to distinguish the syphilitic from the other forms of phlebitis, is that it is not complicated



by embolism. In phlebitis due to puerperal or typhoid fever, to pneumonia, influenza, appendicitis, cancer, tuberculosis, gout, etc., the chief danger lies in embolism and infarcts. Nothing of the kind is found in syphilitic phlebitis, fatal embolism or infarcts having never been noted.

The diagnosis comprises two stages. We must diagnose phlebitis and prove that it is syphilitic. The signs of phlebitis include pain in the course of a vein, the transformation of the vein into a rigid and at times indurated cord, and the appearance of œdema, which may be slight, in the subjacent region of the limb. We must not, however, confuse it with syphilitic lymphangitis, which also forms a superficial rigid, painful cord, with or without reddish tracks in the skin. Lymphangitis gives rise to enlarged and painful glands. Further, the rigid cord in lymphangitis does not coincide with the course of the vein. The decision as to the specific nature demands a survey of all the causes which may engender phlebitis. If we find no such cause, and if, on the other hand, the phlebitis appears a few weeks or months after the chancre, but especially at the same time as the secondary symptoms, the lesion is specific.

I have so far considered early phlebitis. The tertiary forms are quite exceptional. They may appear as gummatous tumours of the vein, or as generalized phlebosclerosis with dilatation of the veins.

**Pathological Anatomy.**—This question has been worked out upon the living subject, because no one has died of the malady. Mendel reports a case in which Lion found a clot totally obliterating the resected segment of the vein.

Darier and Civette have published the following conclusions on the pathological anatomy of phlebitis. Among the manifestations of severe secondary syphilis we may find hard, movable nodules under the skin. Their anatomical situation is in the walls of the subcutaneous veins, their structure is that of a syphiloma, and they give rise secondarily to ordinary thrombo-phlebitis. Mercurial treatment brings about their disappearance, with an apparent return of the tissues to their normal integrity.

The recognition of the spirochæte in the lesions of the vessel wall is the specific hall-mark of syphilitic phlebitis. Benda has outlined the findings in a case of syphilitic arteritis, while Nattan-Larrier and Brindeau have given a complete description of the lesions in a case of specific arteritis and phlebitis. These authors systematically examined eighteen syphilitic placentæ, which showed such multiple vascular lesions as endophlebitis and endarteritis obliterans *aut* vegetans.

In five placentæ they found stainable spirochætæ in the vascular layers of the veins and arteries, which correspond to the tunica media. This finding shows the importance and the action of the spirochæte.

The first point in treatment is to immobilize the limb. I know that this

procedure may appear somewhat unnecessary after the statement that syphilitic phlebitis has never caused embolism. I would not, however, depend on it, because the unexpected may happen, and I therefore kept my patient at absolute rest in bed in the Saint-Christophe ward.

Specific treatment must be employed. Iodide of potassium is not of much use in my opinion, and I prefer injections of a watery solution of biniodide of mercury daily for ten days, the dose being  $\frac{1}{6}$  grain. The treatment can be suspended, and then repeated if occasion arise.

### III. ARTERITIS—ATHEROMA—ARTERIO-SCLEROSIS.

**Anatomy.**—Before undertaking the important question of diseases of the arteries, I will briefly describe their normal structure. The arteries are made up of elastic, muscular, and connective tissue, an endothelial layer, nutrient vessels, and nerves. These tissues vary in extent according to the size of the artery and its functions, just as the arterial lesions vary according to the size of the vessel.

An artery is composed of three coats : (1) The tunica externa, or adventitious coat, is composed of bundles of connective tissue, mixed with elastic fibres, and receives the vaso vasorum, which do not penetrate into the tunica media, except in pathological conditions. (2) The tunica media is the characteristic portion of the artery. In the large arteries (aorta, carotid, pulmonary arteries) the elastic tissue predominates, and the muscular elements are scanty. The elastic fibres and laminae form, as it were, a felting, whence the name *fenestrated* membrane.

These elastic elements are supported upon the internal elastic lamina, which is more important than the external one. In arteries of medium size the tunica media is less rich in elastic tissue and more rich in muscular tissue ; the elastic felting is less important ; the internal lamina persists, but the external lamina has disappeared. The smooth muscle fibres form bundles which are transverse in direction, and are inserted into the network of the tunica externa. In the arterioles the elastic tissue is only represented by the internal elastic lamina, which appears festooned in transverse sections, because the muscular fibres contract and the elastic lamina undergoes retraction. The connective tissue has disappeared. The nerves of the artery belong to the tunica media. (3) The tunica intima varies according to the artery : the endothelial layer, formed by endothelial cells, is everywhere the same ; but in the large arteries of the elastic type we find a fibro-elastic layer, which is divided into two—the one showing longitudinal, the other transverse striation (the lesions of atheroma begin in the fibro-elastic layer). In arteries of the muscular type the longitudinal layer alone persists ; in the arterioles the tunica intima is formed by the endothelium alone.

**History—Discussion.**—Since the commencement of the century, arteritis has been more or less intimately allied with questions of doctrine. Pinel, in his classification of fevers, gave the name of *angiotenic* to a transient fever which originated, as he thought, in inflammation of the arteries. He said that this inflammation was shown post mortem by the *redness* of these vessels.

Bouillaud did not agree with Pinel's narrow classification. In his opinion inflammation of the vessels and of the heart is not only a cause of fever, but represents fever taken in its widest sense. This forms the famous



**angiocarditic** fever, the anatomical proof of which Bouillaud also found in the **redness** and **phlogosis** of the lining membrane of the heart and vessels. At this time, moreover, medicine was dominated by the doctrine of inflammation, gastroenteritis according to Broussais, angiocarditis according to Bouillaud, or, in other words, gastro-intestinal inflammation, on the one hand, and cardio-vascular inflammation on the other; but it was always inflammation causing slightly different types, according to the intensity of the inflammatory process. Two renowned men had been brought to this pass by adhering to **system**.

Reaction, however, occurred. Trousseau and Rigot had already shown that the redness of the arteries, the so-called **phlogosis**, was only the result of cadaveric imbibition, and Bretonneau, in his memorable work, partly destroyed the doctrine of inflammation, and replaced it by the admirable doctrine of specificity, which has in our day been rejuvenated and confirmed by the discovery of microbes. Whilst medicine was led astray as to inflammation of the arteries and its consequences, surgeons published excellent works, and François proclaimed that arteritis causes obliteration of the arteries and spontaneous gangrene. The German school next took the field with the doctrine of embolism (Virchow), and from that time two causes of arterial obliteration were recognized—the one by thrombosis, the other by embolism.

From this time two lines of study were established: the one studied chiefly arterial degeneration, and it appears that the active process was rather neglected; atheroma was found in every case—"this vital rusting," according to Peter's happy expression; "a man is as old as his arteries," according to the dictum of Cazalis. On the other hand, the **active process** was especially studied, endarteritis and periarteritis, obliterating endarteritis and dilating arteritis, being described. Small miliary aneurysms of the brain, associated with cerebral hæmorrhage, were discovered (Bouchard, Charcot), and descriptions were given of the small aneurysms in pulmonary cavities, resulting in fulminant hæmoptysis (Rasmussen), while ectasia of the vessels in bronchial dilatation and the profuse hæmorrhage which they produced, were proved (Hanot).

Our knowledge of these lesions has gradually progressed up to the present time, when we consider chiefly the pathogenesis of arteritis and the part played by infectious diseases and their agents. I must now, however, deal with another important question, that of **arterio-sclerosis**.

In 1872 Gull and Sutton described a kind of fibroid change in the arterioles and capillaries under the name of **arterio-capillary fibrosis**. They showed that the change in the kidneys which results in atrophy is simply the extension of a morbid process which has begun in the small vessels of this organ. The doctrine of arterio-sclerosis was thus created.

What, then, is arterio-sclerosis? It is arteritis of the arterioles, and especially of the visceral arterioles. Endarteritis results in fibrous thickening of the tunica intima, in constriction of the arterioles, and in fibrous change in its walls.

Atheroma and arterio-sclerosis, which are so unlike at first sight, result from the same causes, except that the result is atheroma in the large arteries or in those of medium size, and arterio-sclerosis in the visceral arterioles. Atheroma of the large arteries is said, therefore, to be the result of arterio-sclerosis of their vasa vasorum.

The characteristic point is that the process commences in the small vessels, but as it does not remain limited to them, the process becomes extravascular, and extends to the connective tissue and the elements of the organ, which are replaced by newly-formed fibrous tissue. The change arises in the arterioles, and extends to the neighbouring regions in the form of bands and islets of fibrous tissue.

Arterio-sclerosis, when once present, shows no limit. According to some authors (Martin, Huchard, Weber), it sums up the pathogenesis of nearly all the chronic affections of the viscera.

Certain forms of **nephritis**, formerly considered as having an interstitial or epithelial origin, including senile nephritis, which has been looked on as epithelial (Charcot and Gombault), would therefore be cases of renal sclerosis of vascular origin.

Arterio-sclerosis of the coronary arteries would embrace the whole of **cardiac** pathology. The heart in Bright's disease, the senile heart, the fatty heart, hypertrophic fibrous myocarditis, and myocarditis associated with valvular lesions, would all depend on arterio-sclerosis. Valvular lesions of the heart would depend upon the primary vascular lesions, and without arterio-sclerosis of the coronary arteries angina pectoris would not exist.

The lesions of the **aorta** and the large arteries are also secondary to arterio-sclerosis. Atheroma only shows itself in arteries provided with vasa vasorum, and is the result of endarteritis and arterio-sclerosis of these vasa vasorum. The proof lies in the fact that the vasa vasorum, which correspond to the atheromatous patches, are always affected by arteritis obliterans (Martin).

In the **nerve centres** arterio-sclerosis plays a considerable part, so that tabes, general paralysis, many syphilitic lesions, etc., may have their origin in arterio-sclerosis.

The **genito-urinary** apparatus is also under the ban of arterio-sclerosis, which causes fibrous hypertrophy of the prostate and the bladder. I shall describe later the lesions of the kidneys.

The stomach and spleen present changes due to arterio-sclerosis. The



portal vein, too, may be affected by phlebo-sclerosis (Laënnec's atrophic cirrhosis).

Such are the chief local manifestations of arterio-sclerosis. In different cases it may remain localized to a single organ, such as the kidney, heart, or lung; it may attack several organs at once, or even become general and be associated or not with lesions of the large arteries, and especially with atheroma.

These views of arterio-sclerosis are of much interest, but it must be admitted that for some years arterio-sclerosis has been singularly abused. It has become all-invading, and has been held to explain everything, so that when any difficulty is experienced in finding a pathogenic or clinical interpretation, the reply is: "It is arterio-sclerosis!"

Does such a constant relation between the cause and effect really exist? Is the arterio-sclerosis always the primary lesion, and are the fibrous lesions in the viscera always secondary to sclerosis of the small arteries? When these visceral lesions are not in the immediate neighbourhood of the diseased arteriole, the partisans of this doctrine say that we must then admit either capillaritis or pericapillaritis; but then we have reached the confines of the capillary network in the innermost layers of the tissues. Why, then, may it not be admitted that the irritant sclerogenous substance, whether bacillus or toxine; whether animal, vegetable, or mineral; whether alcohol or lead, may be able to act upon the cells of the connective tissue and of the organs? The desire to reduce all visceral scleroses to arterio-sclerosis appears to me exaggerated. Reaction is taking place in this matter. Bard and Philippe, in their interesting paper, have shown, as regards the heart, that side by side with vascular sclerous hypertrophic myocarditis there is room for interstitial myocarditis, in which the arterio-sclerotic lesion is secondary. In general paralysis, according to Joffroy, encephalitis is much more probably of parenchymatous than of vascular origin. Some authors, confounding the lesions and its effects, find it a simple matter to set down to arterio-sclerosis the symptoms of "Brightism," which are really symptoms of toxicity. The result is the confounding of the vascular lesions of the kidneys with the multiple effects of intoxication from insufficiency of the urinary depuration.

After this general discussion, let us pass to the detailed study of arteritis and arterio-sclerosis. Arteritis may develop under the most varied conditions, and may have almost identical results, although its origin is very different. It is therefore necessary to divide up arteritis.

### Traumatic Arteritis.

These cases belong chiefly to the domain of surgery. Some, however, are of interest both to the physician and the surgeon, such as those which develop in the neighbourhood of superficial or of deep ulceration of the

skin, or of a viscus. In this case the walls of the artery do not escape the inflammatory and destructive process which affects the surrounding tissues, and, accordingly, two eventualities may happen. If the inflammatory process predominates, the walls of the artery become thickened and proliferate, the calibre of the vessel is diminished, the blood coagulates, and the artery is gradually converted into an impermeable fibrous cord, which may ultimately be destroyed, without untoward consequences. If, on the other hand, the ulcerative process is rapid (ulcerative or suppurative arteritis), the walls of the artery are destroyed from without inwards, before coagulation of the blood can take place, lose their resistance, and finally give rise to an aneurysm. This sometimes happens in the interior of tubercular cavities or on the surface of gastric ulcers. If, at a given moment, the arterial tension increases, the resistance of the walls is insufficient, the aneurysm bursts, and fulminating hæmorrhage occurs.

The arterial lesions which occur when an artery is obliterated by embolism are also related to traumatic arteritis. The irritation produced by the foreign body, whatever may be its point of departure and its nature, leads to inflammation of the tunica intima and to lesions analogous to those which we shall describe later.

### Infective Arteritis.

**Pathogenesis.**—This group comprises those cases which supervene during or after general diseases. They have been verified by pathological anatomy and by bacteriology, and their existence is indisputable. They are most often seen in acute diseases, such as typhoid fever, etc. (Taupin, Potain, Vulpian, Hayem); diphtheria (H. Martin); puerperal conditions (Simpson); scarlatina, measles, variola (Brouardel); malaria, rheumatism (Lancereaux, Guéneau de Mussy), etc. Among the more chronic diseases which may cause arteritis, tuberculosis and syphilis specially deserve mention.

The data acquired as regards the evolution of the tubercle bacillus show that propagation occurs both by the bloodvessels and by the lymphatics. If it is arrested in an arteriole, a colony of microbes develops at this point. The irritation thus produced in the walls of the artery, whatever be its size, suffices to cause specific inflammation. In this manner the starting-point of most tubercular granulations is explained.

Syphilitic arteritis will be studied in the next section.

As regards infective arteritis, developing in the course of the maladies enumerated above, it is probable that the arterial lesion is directly caused by the micro-organism which produces the infection, unless the toxins secreted by it can be also incriminated. These cases of arteritis would, therefore, in their mechanism resemble another class—namely, the **toxic class of arteritis**. They have, however, this distinguishing point—namely, that



in infective arteritis the number of diseased arteries and the extent of the lesions are always limited, while toxic arteritis affects a large number of vessels, and involves each vessel to a large extent.

**Pathological Anatomy.**—The following lesions are found in infective arteritis: (1) When a large artery, such as the aorta, is affected, the inner wall is red, rugose, and in places glazed, while other parts are depressed in a cuplike form, and resemble the pustule of variola (*vide* Aortitis), and the external surface shows a close network of capillary vessels filled with blood. (2) In the small arteries and in those of medium size the lesions are not visible to the naked eye, but under the microscope they are identical with those found in the large arteries. They comprise an increased thickness of the tunica intima (endarteritis), which projects into the lumen of the vessel—sometimes at one point only (parietal arteritis, Barié), at other times around the whole circumference of the vessel. When the artery is affected upon one side only, the granulation rarely projects sufficiently to obliterate the lumen, which is simply constricted. If, on the other hand, the arteritis is circumferential, the lumen of the vessel rapidly disappears (obliterating arteritis), and the circulation is interrupted in the whole area supplied by this artery, unless the collateral circulation supplies the defect. The thickening of the tunica intima depends on the multiplication of the anatomical elements, and on the infiltration of leucocytes between these new-formed elements.

Endarteritis may exist alone, but as a rule there is also some **periarteritis**. In this case the tunica externa is thickened, and pushes aside the structures in contact with it. In this case also the inflammation is characterized by the appearance of embryonic cells between the connective and elastic bundles of the tunica externa. All the elements show great tendency to undergo fibrous change.

**Symptoms.**—The symptoms of arteritis in acute diseases vary according to the calibre of the vessel, and to the organs which it supplies. 1. If the aorta is affected (syphilis, variola, puerperal state, and malaria), the severe symptoms of acute or of subacute aortitis will appear. 2. If the artery is of medium size, as in a limb (typhoid fever), the patient experiences very acute pain along the artery, or at the extremity of the limb, and the pain is increased by pressure and movement. At the same time the pulse diminishes or disappears completely, and the limb becomes cold, blue, and insensitive. A hard cord which rolls under the finger is felt along the course of the artery. If the obliteration is complete and the collateral circulation is not re-established, pustules form, livid patches appear, and the limb is affected by dry or moist gangrene. In some cases the results are not always so serious: the pulse gradually returns, the hard cord disappears, and the cyanosis only remains in the form of violet-coloured patches. Walking

may cause oedema, lasting several months, but gangrene is averted, and recovery, although slow, is the usual termination. In some cases arteritis makes very rapid and acute progress, as we shall see under Typhoid Fever.

If arteritis affects a cerebral artery (typhoid fever, syphilis), the resulting troubles depend on the functions of the area supplied by the diseased vessel. As the arteries of the motor zone are most often affected, the symptoms are nearly always very grave, and include hemiplegia, aphasia, contracture, etc. In the case of syphilitic arteritis, however, these troubles often improve and even disappear under antisiphilitic treatment, when the artery recovers its permeability, as in a case of syphilitic arteritis of the temporal arteries published by Leudet. For further detail I refer to the section on Cerebral Syphilis.

3. If the diseased arteries are of small calibre, such as those of the muscles or of the viscera, the lesions are identical with those already described. In some cases the nutrition of the invaded tissues is severely compromised. If a muscle is affected, the myositis may cause softening, and end in rupture. If the arteritis arises in the heart, for example, inflammation of arterial origin, which may give rise to all the lesions and clinical signs of myocarditis, results. As a general rule, these inflammations are transient. Lately, however, cases of chronic myocarditis (Landouzy and Siredey), encephalitis, myelitis (Marie), and nephritis, originating in infective arteritis, have been published.

### Toxic Arteritis—Atheroma—Arterio-Sclerosis.

We shall include in this group arterial lesions consecutive to pathogenic agents, to their toxins, and to vegetable or mineral poisons. The arterial lesions in alcoholism may be taken as our type. Still, plumbism, gout, rheumatism, diabetes, old age, and the infectious diseases, may give rise to them. In some cases the irritative element differs, but the inflammatory processes are identical.

They may be reduced to two chief groups :

1. As in the preceding case, the artery may be affected by **endoperiarteritis**, which diminishes its calibre and changes it into a fibrous tube. We then say that **arterio-sclerosis** is present (Gull and Sutton).

2. The tunica intima may be affected, but only in the layers subjacent to the endothelium. At this level yellowish cuplike patches are formed, having the dimensions of a lentil and filled by a fatty pulp, **atheroma**, which flows out when the endothelial covering is punctured. If the fluid as well as the fatty matter is reabsorbed, the calcareous salts only remain, and then give to the patch a rigid consistency. These atheromatous centres, which may exist in great numbers in the same vessel, may finally fuse, and give to the artery a cartilaginiform or ossiform rigidity. Accordingly, the name



of ossification of the arteries has been given to this disease, although it is not a correct term. The affected vessel loses its elasticity, bursts easily, and allows distension (aneurysm), while the irregularity of its internal surface favours coagulation of the blood (thrombosis).

These lesions, which are sometimes isolated, sometimes contiguous, occur almost exclusively in the arteries of the greater circulation. They are usually diffuse and always run a very slow course. When the whole arterial system is invaded, **atheroma** chiefly affects **the large arteries**, while **arterio-sclerosis** develops by preference in the **visceral arterioles**.

Arterio-sclerosis causes changes of two kinds in the viscera: (1) Peri-arterial inflammation which proceeds by foci, in the centres of which a diseased artery is always found (inflammatory sclerosis); (2) a lesion of degeneration, in which foci of sclerosis are also formed, but are always at a very long distance from the diseased vessel (dystrophic sclerosis, H. Martin), showing, therefore, the exact opposite of what occurs in the preceding case. Sometimes these two kinds of change are found side by side (mixed sclerosis). Of the symptoms of atheroma and arterio-sclerosis, some are due to the arterial lesions themselves, while others depend on the visceral changes which they produce. Among the former (atheroma) we will cite the tortuous course and rigid character of the arteries and the increase of arterial tension, which is shown with the sphygmograph by a sharp line of ascent, followed by a flat top and a wavy line of descent; the second sound, which is accentuated, and has a ringing character; and the frequency of purpura and gangrene of the extremities.

Among the troubles consecutive to the visceral changes we may note cerebral troubles, such as vertigo, loss of memory, giddiness, successive attacks of hemiplegia and aphasia, and failing intelligence, which may result in complete dementia. In some patients the heart is chiefly affected by arterio-sclerosis, palpitation, hypertrophy of the heart, angina pectoris, and paroxysmal dyspnoea are frequent. In other patients the digestive functions are first affected—loss of appetite and frequent indigestion. Lastly, in a fair number of cases renal changes open the scene and cause insufficiency of urinary depuration, and the numerous symptoms of Brightism, which must be carefully elucidated before the appearance of the grave symptoms of confirmed uræmia.

The treatment of atheroma and arterio-sclerosis is based on the use of iodides, milk diet, and remedies which will lower the arterial tension (Huchard).

#### IV. SYPHILITIC ARTERITIS—ARTERITIS OBLITERANS— SYPHILITIC GANGRENE—ARTERITIS ECTASIANS —SYPHILITIC ANEURYSM.

In the course of syphilis the arterial vessels may be affected alone, without any lesion of the neighbouring tissues. These cases only have a special clinical history. Syphilitic arteritis of the brain, spinal cord, aorta, etc., is dealt with under syphilis of these organs. I shall deal at present only with peripheral arteritis, and in particular with arteritis of the limbs, which forms a distinct and very interesting pathological group.

The process (either in the arteries of the limbs or in the cerebro-meningeal ones) may result in two different kinds of lesions: on the one hand, more or less complete **obliteration** of the vessel; on the other hand, **ectasia** of the vessel or **aneurysm**.

**Arteritis Obliterans—Gangrene.**—I do not know of a more typical case than that presented by Leudet (of Rouen) at the Blois Congress in 1884.

The patient was syphilitic, and suffered from painful obliterating arteritis of the anterior frontal branch of the left superficial temporal artery. This primary arteritis obliterans, limited to an arterial segment, was soon followed by symmetrical arteritis obliterans of the right side. Leudet was able to follow all its phases, including induration, obliteration of the artery, diminution and cessation of the pulse; then, under the influence of antisymphilitic treatment, he was able to follow the re-establishment of the arterial circulation, return of the pulse, permeability of the artery, and finally cure.

Syphilitic arteritis obliterans of the limbs may be followed, just as atheromatous arteritis, by mummification of the extremities, which is in every way comparable to ordinary senile gangrene. Here is a case:

An old woman came under my care for gangrene of the right leg and foot, which had come on suddenly with sharp pain two months before. The toes were blackish and shrivelled; the dorsum of the foot was livid. A large slough had invaded the lower part of the limb. The pulse was absent in the dorsalis pedis and in the tibial arteries, the skin was cold and insensible, and the picture was, in a word, that of senile gangrene. One thing, however, struck us: Over the forearm and abdomen there were irregular pigmented scars, with polycyclical outlines, and on the left upper eyelid a large copper-coloured papule, with scalloped edges. These lesions were syphilitic. I at once concluded that the gangrene of the leg was due to syphilitic arteritis obliterans. The patient was given injections of biniodide of mercury, but the treatment came too late, and amputation became necessary. The patient soon succumbed.

Examination of the amputated limb showed that the gangrene had invaded most of the posterior muscles, in the form of huge lardaceous infarcts of a waxy-yellow colour. The femoral artery was healthy; the branches of the popliteal, the tibio-peroneal trunk, and the posterior and anterior tibial arteries were obliterated in segments. At these spots the wall of the artery showed a ring-like thickening, and the lumen was obliterated by adherent thrombus. Between the segments the lesions were absent or very slight. Everywhere else, as was found later at the autopsy, the arteries were healthy and free from atheroma. Microscopical examination showed that this process



originated in the deep part of the subendothelial layer of the tunica intima, above the internal elastic limiting layer, and the organization of a thrombus had rapidly completed the obliteration.

Cases of this kind are relatively rare. Aune, however, has collected seven, and several analogous ones have been published.

Syphilitic arteritis attacks the lower limbs (Charcot, Fournier, Podres, Hutchinson) as often as the upper ones, and may be bilateral, symmetrical, or multiple, as shown by Magrez's case, in which arteritis obliterans invaded in succession the right arm and the legs, with gangrene of the left leg.

The onset is sometimes progressive, with functional troubles, loss of muscular power, and intermittent claudication; at other times sudden, with very sharp pain. Numbness, formication, coldness, cyanosis, and œdema then appear. The arterial pulsations disappear in the affected segment, and if the artery is superficial a painful cord may be felt.

The slow forms may be arrested by treatment; the permeability of the vessel becomes re-established, and the threatening symptoms disappear, although a fresh attack of arteritis may show itself in some other part. In cases of sudden onset and rapid course the obliteration results in dry gangrene. Accordingly, when a patient suffers from sharp pain, intermittent claudication, chilliness, cyanosis, and gangrene of the foot, syphilitic arteritis must be thought of, and careful search made for other stigmata of syphilis.

From the anatomical point of view our patient showed the chief characters of syphilitic arteritis. We see cases of departmental arteritis affecting a limited area of a limb. In this region the lesions do not show the same intensity at all points; one segment of the artery is respected, while another is obliterated. In the limbs, as in the brain, arteritis obliterans is chiefly segmental, and at times symmetrical (Magrez's case affecting the lower limbs, and Leudet's case affecting the temporal arteries). When these anatomical characters are clear, it is easy to distinguish between syphilitic and atheromatous lesions, which are diffuse and general; but syphilitic aortitis may also end in lesions comparable to those of atheroma (Cornil).

The **histology** of syphilitic arteritis obliterans shows nothing specific. At a late period the three coats of the artery are thickened and infiltrated, with or without miliary nodules, resembling microscopic gummata (Joffroy). The lesions frequently commence with endarteritis, rarely with periarteritis. Obliteration may occur as the result of two processes. Sometimes we find only endarteritis vegetans, which obliterates the artery more or less completely, and converts it into a fibrous cord, without any appreciable thrombus; at other times (this is the more frequent) there is thrombo-arteritis obliterans: the endothelium desquamates, or becomes necrosed, and the thrombus contributes very largely in obliterating the vessel. In both cases

the arteritis may be chronic or subacute. It appears possible to differentiate it from ordinary atheroma by the greater activity and the more rapid course in the direction of obliteration, as well as by the lessened tendency to degenerative lesions.

**Syphilitic Aneurysm.**—The second type of syphilitic arteritis in the limbs results in dilatation and aneurysm. The dilatations result from the same process of embryonic infiltration of the coats of the vessel; the middle coat, which is much infiltrated, has lost all power of resistance, and yields at one or at several points. We may see upon some vessels buds of endarteritis and aneurysmal dilatations. Here, as in the brain, these dilatations may be cylindrical and extensive, or circumscribed and aneurysmal. Aneurysms sometimes develop in several arteries (popliteal and innominate trunks, Croft); they chiefly affect the femoral, popliteal, subclavian, brachial, and radial vessels. I have seen syphilitic arteritis of the left radial artery at the wrist, which had resulted in the formation of an aneurysm.

The patient had been previously treated for a large serpiginous ulcer on the right arm. I saw him again two years later for a syphilitic cavity in the right lung. Under mercury and iodide of potassium, in large doses for a long period, all symptoms were cured. When he came to me some years later for cerebral troubles, I noticed that he wore a wrist-strap. He informed me that for some weeks past fairly firm pressure had been employed for a painful aneurysm of the left radial artery, and that an operation had been decided upon, as no benefit accrued from the compression. I asked to see the aneurysm, which was as large as a small nut, and said that this aneurysm might be the result of syphilis. I commenced treatment with large doses of mercury and iodides. The aneurysm gradually diminished in size, and three months later had quite disappeared.

Treatment is very efficacious when commenced early, before the lesions are irremediable. It consists in giving daily an injection of the solution of biniodide of mercury. Iodide of potassium may be given in addition to the mercurial treatment.

## V. ACUTE AND CHRONIC AORTITIS.

**Ætiology.**—Both acute and chronic aortitis occur. Primary acute aortitis does not, however, exist. “In the immense majority of cases the lesions of acute aortitis develop in those vessels which have previously been attacked by chronic affections.” Accordingly, these different varieties of aortitis should be described in the same chapter. Consequently, all causes which favour the development of atheroma, including old age, gout, and alcoholism, find their place in the ætiology of aortitis. **Syphilis** has a **pre-eminent** place in the pathogenesis of aortitis and aortic aneurysms. Injury, blows, and contusions may be held guilty. Infectious diseases, typhoid fever, rheumatism, tuberculosis, variola, scarlatina, and influenza may the more excite attacks of acute aortitis in proportion as the aorta has already



been the seat of chronic lesions. It is probable that bacteriological researches will finally discover the pathogenic agents of these acute attacks. Cuzzatini has found the pneumococcus in a case of aortitis set down to **cold**.

**Pathological Anatomy.**—When aortitis is frankly acute the aorta is dilated, and its inner surface is uneven. The lesion commences in the sub-endothelial coat of the tunica intima (Cornil and Ranvier). The tunica intima shows prominent patches, which vary in size from a pin's head to that of a small coin; they have been named gelatiniform patches, on account of their transparent and gelatinous appearance. These patches are formed of spherical, nucleated, embryonic cells, and some branched, flattened cells, which normally are found in the lining membrane. The patches may be a hundred times as thick as the normal membrane, and the inflammatory process, which is more active in the layers bordering on the endothelium when the aortitis is acute, especially affects the deep layer when it is chronic. These patches are sometimes of a dark brownish colour.

Small fibrinous clots may be deposited on these patches, and at times become the origin of emboli. The middle coat is but little altered, while the external coat is thickened and vascular. In some cases acute aortitis is ulcerative, or may even be suppurative.

The lesions of acute aortitis may spread around the vessel, and cause pericarditis, pleurisy, lesions of the cardiac plexus, and consecutive angina pectoris, a complication which plays a large part in the symptoms of aortitis.

**Chronic aortitis**, like **chronic arteritis**, which is frequently associated with it, is often called **atheroma**; but these two words, **arteritis** and **atheroma**, have not the same signification, for the word **atheroma**, which means a "pulp," denotes only one stage of chronic arteritis. Atheroma, "this vital rusting" (Peter) and chronic arteritis may produce such changes in the artery that these lesions have been differently called **deforming** and **nodular endarteritis**, or **arterial atheromasia**.

The lesions of chronic aortitis are characteristic. The aorta is nodular and dilated; it is rigid to the touch and hard on section; on its inner surface we find yellowish, calcareous, atheromatous patches, and dilatations of different shapes. The inflammatory process in chronic aortitis is analogous at the onset with that of the acute disease. The lesion begins in the subendothelial layer of the tunica intima, and forms patches which later undergo fatty and calcareous change—that is to say, atheroma.

These degenerations, however, are not always the result of an inflammatory process, for cases occur in which fatty degeneration of the arterial walls is the primary lesion in point of time; this lesion may affect all three coats, and may in its turn produce slow irritation and foci of chronic aortitis, so that these various lesions resemble one another, or are complete in themselves. According to some authors, these atheromatous lesions,

which are apparently primary, are the result of arterio-sclerosis of the vasa vasorum (Martin).

Whatever be their origin, the lesions of chronic aortitis are always accompanied by **atheromatous centres** and **calcareous patches**. The gelatiniform patches of the tunica intima, by their fatty change, finally form an atheromatous centre, composed of fatty detritus, cholesterine, and crystals. The fibrillary groundwork which surrounds these foci takes on a chondroid appearance, and becomes infiltrated with calcareous granules. If the wall of the vessel is intact the focus projects into the artery as an atheromatous pustule, but if the endothelial layer bursts the contents of the focus are carried away by the blood-stream; the blood enters the focus, and the blood-pressure at this point may cause dilatation and aneurysm. In some cases atheroma prepares the way for **spontaneous** rupture of the aorta, especially of its intra-pericardial portion. The blood filters between the atheromatous patches, dissects the tunica externa, and rupture occurs into the pericardium, pleura, or cellular tissue. In chronic aortitis the tunica media in turn undergoes change, and disappears in places, as the result of fatty degeneration; it loses its resistance and elasticity, while the tunica externa often becomes fibrous, so that the whole vessel is invaded; endarteritis, mesarteritis, and periarteritis are all present (Peter). The aortic orifice and valves often participate in the process of aortitis. In some cases chronic aortitis is associated with generalized atheromasia.

**Description.**—I shall first deal with acute aortitis, or at least with acute attacks which supervene in the course of a chronic process that is often latent. Acute aortitis behaves differently, according to the case; it may pass unnoticed, just as acute endocarditis may do, or it may be accompanied by a feeling of weight and oppression in the precordial and epigastric regions, or, lastly, it may give rise to the symptoms of angina pectoris.

Pain and dyspnœa are sometimes the chief symptoms. The pain is retrosternal, stabbing, continuous or paroxysmal, and radiates to the arms, neck, back, intercostal spaces, larynx, œsophagus (dysphagia), stomach (gastric crises), or liver (false hepatic colic). The dyspnœa is also continuous or paroxysmal, but the respiratory rhythm is not increased. During the attacks of pain and dyspnœa we see pulsation of the carotid and subclavian arteries, and the patient is sometimes seized with fits of coughing, and brings up bloody expectoration. On auscultation, fine râles, indicating congestion and œdema of the lung, are found over a somewhat limited area. These attacks of congestion are not solely limited to the lungs; they affect the liver, which becomes enlarged and painful, and also cause intestinal dilatation and ballooning of the belly (Rendu). Sometimes acute infective aortitis results in the formation of a purulent collection, which opens into the vessel, and presents the clinical picture of pyæmic endocarditis.



Let us now consider chronic aortitis. The signs of chronic aortitis vary with the situation of the lesion; let us take the most frequent case—namely, that of atheroma of the arch of the aorta. In the normal state the transverse dullness of the aortic and pulmonary trunks together is about 2 inches (Peter); in chronic aortitis the dullness may exceed 3 inches, according to the dilatation of the vessel.

The aorta by its expansion raises the subclavian arteries, so that we can easily feel them in the supraclavicular hollows. Auscultation may reveal differences of tone or blowing murmurs (Potain). The first aortic sound, which in the normal state depends on the sudden tension of the aorta, may become exaggerated, harsh, and blowing as the result of the atheromatous roughness of the vessel wall. The second sound, which in the normal condition depends on the closure of the sigmoid valves, may be echoing and metallic if the valves have lost their suppleness. A diastolic murmur, due to concomitant aortic insufficiency, may also be heard. The pulse is hard, while the radial artery, which often participates in the atheromasia, is tortuous and indurated. Atheroma of the aorta, like general atheromasia, is always accompanied by hypertrophy of the heart.

The clinical picture varies according as chronic aortitis comprises the whole disease, or as it is associated with a lesion of the aortic orifice, aortic aneurysm, nephritis, more or less extensive atheromasia, or local arteriosclerosis.

In short, chronic aortitis alone may remain silent for a long time, and is usually revealed by attacks of acute aortitis, while several of the symptoms which accompany it are borrowed—viz., hypertrophy of the heart, aortic insufficiency, angina pectoris, Bright's disease, etc.

Aortic aneurysm and chronic aortitis present many common signs and symptoms; among other distinctive signs there is one which has some worth—namely, that chronic aortitis does not form a tumour as aneurysm does, and consequently does not produce symptoms of compression.

Blood-letting with leeches or cupping, blisters, the actual cautery, subcutaneous injections of morphia and antipyrin, form the general plan of treatment. Iodide of potassium should be administered in large and continuous doses in acute and chronic aortitis.

## VI. ANEURYSMS OF THE THORACIC AORTA.

**Ætiology.**—Aneurysm of the aorta is very rare before the age of thirty-five years, and its ætiology is closely related to that of acute and chronic aortitis. In the enumeration of its causes heredity should not be forgotten. Injuries of the thoracic region and heavy manual labour have an influence on aneurysm, but syphilis is the chief cause.

**Pathological Anatomy.**—Aneurysm of the thoracic aorta affects, in order of frequency, first, the ascending aorta ; second, the convexity of the arch ; third, the descending aorta. The aneurysm varies in size from a small nut to that of the foetal head, and its shape is extremely variable ; it is said to be **sacciform** when the dilatation is limited to a part of the circumference, like a sac hanging from the vessel ; it is said to be **fusiform** when the dilatation is equally distributed over the whole circumference of the invaded segment. The small hemispherical aneurysms found chiefly at the origin of the aorta are called **cupuliform** ; the name **dissecting** is given to the aneurysm which results when the blood infiltrates between the internal and middle coats, and separates them to a certain extent. Two or three aneurysms in the course of the aorta are sometimes found in the same patient.

There has been lengthy discussion upon the way in which the sac forms, and, according to the part assigned to the different coats of the vessel, the classification of aneurysms into mixed internal, mixed external, etc., has been proposed, but the question should be simplified. When an artery is affected by chronic arteritis, the tunica media disappears, and the sac of the aneurysm is formed by the joined internal and external coats ; often, indeed, the walls of the sac are formed by the internal coat alone, which has been modified by the inflammation. “The newly-formed tissue which, wholly or in part, constitutes the sac is made up of flat cells, separated by a fibrillary substance, and undergoes fatty change, atheroma, and petrification ; we may also see old sacs formed of an inextensible calcareous shell” (Cornil and Ranvier).

These lesions explain the formation and development of the aneurysm ; the tunica media disappears as the result of endarteritis and periarteritis, and the resistance of the vessel becomes insufficient, so that the artery yields to the blood-pressure, and the vulnerable points permit distension. The distension of the vessel does not necessarily induce thinning of its walls, for the growth of the morbid tissue continues ; at some points, however, thinning may result and favour the rupture of the sac.

When the sac is opened, blood-clots and **laminated fibrin** are found. The clots are soft and recent, the layers of fibrin are elastic and greyish ; the older ones are found nearer the walls of the sac and are resistant, but no trace of organization is seen.

Aneurysm of the aorta is not always associated with extensive atheromasia or with arterio-sclerosis ; indeed, atheroma may be only present in the aorta and nowhere else—that is to say, the aorta may be diseased **on its own account**, independently of any other arterial lesion.

The **heart**, which some authors regard as being always hypertrophied in cases of aortic aneurysm, often preserves its **normal size**, as I have several times found.



The neighbouring tissues and organs in contact with the aneurysm undergo important changes; the **bones** show excavations: this is not the result of mechanical wear and tear, due, as was first supposed, to the movements of the aneurysm, but is the result of an irritative process, or **osteitis**, which brings about the absorption. In other cases the sac is bound down to the neighbouring organs, and spreads the inflammatory process, which leads to softening, ulceration, and perforation of the invaded tissue or organ. The fact is thus explained that the aneurysm may open into the pleura, pericardium, œsophagus, trachea, pulmonary artery, superior vena cava, or right auricle.

The communication between the aortic aneurysm and the veins gives rise to an **arterio-venous aneurysm**.

Patients suffering from aneurysm of the aorta are frequently tubercular—*e.g.*, eighteen times in forty-six cases. Various interpretations of this secondary phthisis have been given; it has been supposed that the disease is brought about by the compression of the pulmonary artery, and it has been compared to the tuberculosis that accompanies constriction of this vessel.

In one of my patients suffering from an enormous aneurysm of the aorta, death resulted from left tubercular broncho-pneumonia, which had developed consecutively to compression of the bronchus and of the left pulmonary artery by the aneurysm.

**Symptoms.**—Aneurysm of the thoracic aorta is sometimes latent, and the person affected may be suddenly carried off without any previous warning. I do not know a more conclusive case than one published by Roux of a young soldier, twenty-two years of age, who died suddenly from an aneurysm which opened into the pericardium. As a rule, events do not take this course, and the aneurysm shows itself by certain symptoms and signs; their frequency and value I shall now discuss.

1. **Pain.**—Pain may be the first symptom. Its situation and its nature depend on the nervous network affected by the tumour, so that we see spinal pain and intercostal neuralgia (lesions of the spinal nerves at their point of exit from the vertebral column), pains in the arms or in the hands, and cubital neuralgia (lesions of the brachial plexus), the agonizing pains of angina pectoris (lesions of the cardiac plexus), diaphragmatic pain and phrenic neuralgia (lesions of the phrenic nerve). These pains may be continuous, intermittent, or paroxysmal, and many persons who have aneurysm of the aorta think that they are suffering from simple intercostal neuralgia, or from angina pectoris.

2. **Dyspnœa.**—The respiratory troubles vary much. One patient may have spasm of the glottis, with attacks of suffocation; another has paralysis of the posterior crico-arytenoid muscles, which open the glottis; while a third suffers from **hiccough**, accompanied by angina and thoracic

constriction (phrenic nerve). The dyspnoea is sometimes continuous, or nearly so, being excited and increased by the least effort; inspiration is painful, and accompanied by stridor (compression of the trachea or of a large bronchus). Stridor, whether it is complete or incomplete, is a symptom of great value; when the patient is at rest, the stridor is but little marked, and only a slightly prolonged and rough **inspiration** is audible, but, as the result of effort, true stridor appears. When a large bronchus is compressed, auscultation reveals abolition or diminution of the vesicular murmur in the corresponding lung. Some patients have fits of coughing which resemble whooping-cough.

**3. Vocal Troubles.**—We find, according to the case, **dysphonia** or **bitonal** voice (Jaccoud), hoarseness of the voice, and aphonia. These vocal troubles may be intermittent or continuous, and depend on paralysis of the vocal cords, and especially of the left cord, as may be readily seen with the laryngoscope.

**4. Dysphagia.**—The trouble may be continuous or intermittent, and depends upon many causes: compression of the œsophagus by the aneurysm, paralysis or excitation of the vagus and the recurrent nerve, which supply the constrictors of the œsophagus and pharynx.

**Physical Signs.**—On examination of the thorax we find dullness which is in direct relation to the size of the tumour, and we may sometimes discover bulging in the aortic region. If the tumour is visible externally, it shows pulsation, so that we might say there are two hearts in the chest (Stokes). The pulsation of the tumour may be single or double; the first beat is due to the blood-wave which enters the aneurysm; the second beat has been differently interpreted: it may be due to the return of the blood-wave into the sac, or to the reflux of blood from the collateral arteries. Perhaps the two beats only represent the distension of the aneurysmal tumour at two different periods (Franck). The beats which occur later than the cardiac systole are **expansile**, and are sometimes accompanied by **thrill**.

Auscultation over the sac reveals **splashing** and **blowing sounds**, which may be single or double, and audible in the anterior thoracic region, and sometimes in the interscapular region. The first splashing sound is due to the shock of the blood-wave upon the walls of the aneurysm, while the second is due to the closure of the sigmoid valves. The first blowing sound is due to the roughening of the aorta from atheroma, or its compression by the aneurysm; the second sound (*souffle de retour*) is due to the return of the blood-wave into the sac, or to the aortic insufficiency which sometimes accompanies aneurysm.

The **radial pulse** presents peculiar characters, the lines of ascent and descent being practically of the same length, because the jerky movement of the arterial pulse is converted into an almost continuous movement by



the presence of an extensible sac in the course of the arterial tree (Marey). If the aneurysm is proximal to the origin of the large arteries, the radial pulse is isochronous on both sides; if the aneurysm is situated between the left subclavian artery and the innominate trunk, the left radial pulse is delayed.

This retardation of the pulse depends chiefly upon the extensibility of the walls of the aneurysm; the blood-wave is weakened and delayed. If, however, the sac of the aneurysm is but little extensible, and covered with stratified or calcified clots, it loses its extensible properties, and then the conditions change. The signs given by the expansile pulsations in the tumour, the double beats, the splashing and blowing sounds, and the characters of the pulse, all depend on the degree of extensibility in the walls of the aneurysm.

In some cases total **suppression** of the radial pulse has been noted. It may depend on obliteration of the subclavian artery by clots in the sac, or on a patch of atheroma that has developed at the orifice of the collaterals of the aorta, in which case the pulsation of the subclavian artery can no longer be felt; the suppression of the radial pulse may also depend on arterial obliteration, caused by an embolus detached from the fibrinous clots in the aneurysm, in which case the symptoms appear suddenly; it may, lastly, depend upon constriction of the artery from endarteritis obliterans, in which case the symptoms of obliteration are much more gradual.

Compression of a venous trunk (vena cava or innominate vein) may cause œdema and collateral venous circulation, as described under **Tumour of the Mediastinum**. When the circulation in the superior vena cava is interrupted, the head, upper limbs, and trunk—that is, the regions drained by the superior caval system—are cyanotic, œdematous, and show a network of dilated veins, while the abdomen and lower limbs are of normal colour.

**Condition of the Pupil.**—The presence of a tumour may cause inequality of the pupils, the one being smaller or larger than the other; if the sympathetic nerve is destroyed by the aneurysm, the pupil on the corresponding side is contracted (myosis), because the paralysis of the dilator fibres permits the free action of the constrictor fibres which arise from the common oculo-motor nerve.

If the sympathetic nerve is irritated but not destroyed by the aneurysm, the pupil on the corresponding side may be dilated (mydriasis) from over-action of the dilator fibres. In any case the light reflex is not abolished. Inequality of the pupils is an important sign of aneurysm, and may help to distinguish aneurysm from aortitis in which a tumour is absent.

Inequality, however, does not always indicate the presence of a tumour; it may be found, according to Babinski, in syphilitic persons who have neither aneurysm nor mediastinal tumour. They may have mydriasis or myosis, but the important point is that the light reflex is lost (Robertson's

sign). The inequality of the pupils is due to syphilis of the nervous system, and is associated with lymphocytosis in the cerebro-spinal fluid.

Inequality of the pupils in an aortic case may therefore have no direct relation to aortitis or aneurysm, but may be a functional effect of syphilis if the patient has that disease. Babinski has published cases in support of his opinion; Vaquez, Widai, and Lemierre have confirmed his view, and I have seen the following case at the Hôtel-Dieu :

A man, thirty-six years of age, who had had a severe attack of angina pectoris, was admitted under my care. He had been picked up moribund and brought to the hospital. Some hours elapsed before he could give us exact information. He gave a clear history of angina pectoris; for months past he had had similar attacks. His heart was hypertrophied, and a to-and-fro murmur was heard in the second right space. The attacks of angina pointed to suprasigmoid aortitis. Right pupil much dilated. If we had been unable to push investigations farther, we might have stopped at aortic enlargement.

The light reflex, however, was also absent. Syphilis of the nervous system was shown by lymphocytosis of the cerebro-spinal fluid. He had had syphilis twenty years before. The inequality of the pupils was therefore dependent upon syphilis, and it was possible to exclude a tumour. The autopsy showed the accuracy of this diagnosis: aortitis, no enlargement, no tumour of the aorta; the lesion in the nervous system was posterior spinal meningitis.

Inequality of the pupils in disease of the thoracic aorta may depend on two different causes: it may be due to interference with the sympathetic nerve by an aneurysm, or it may depend on a specific lesion of the nervous system coexisting or not with syphilitic aortitis; in the latter case we find Argyll-Robertson's sign and lymphocytosis.

Inequality of the pupil in lesions of the aorta does not therefore of necessity imply the existence of an aneurysm.

**Topographical Diagnosis.**—The existence of an aortic aneurysm will be recognized by the signs and symptoms just enumerated. Its **exact position must also be diagnosed** as accurately as possible, because it appears to me that its gravity depends more upon the situation than upon the size.

It is generally believed that the largest aneurysms are the most formidable; there is some truth in this assertion, but it is far from being the rule. Some aneurysms of **small size** are more formidable than large aneurysms. An aneurysm which grows towards the exterior may exist for many years, in spite of its great size, before compromising the patient's life, while small aneurysms, in relation with a bronchus or the trachea, may produce rapidly fatal hæmorrhage in patients who have been almost ignorant of the lesion. Careful study of symptoms will help us to localize the **exact situation** of the aneurysm.

Aneurysms which develop in the **convex** portion of the arch and those of the **anterior** portion may reach a very large size, without causing ulceration and perforation of the trachea, with which they are in no immediate relation.



These aneurysms grow forwards and upwards towards the sternum and ribs, which become eroded by rarefying osteitis. They show themselves by dullness, blowing murmurs, and expansile pulsation: signs most marked over the tumour; while their intensity is in relation with the prominence of the aneurysm, and the more or less complete disappearance of the chest-wall. Rupture into the trachea is not to be feared—at least, as long as the tumour does not affect the whole calibre of the artery, including its posterior wall, and does not cause ulcerative mischief in the direction of the trachea.

On the other hand, these mishaps are relatively frequent when the aneurysm is close to **the loop of the left recurrent nerve**. This variety merits special recognition; I have called it **aneurysm of the recurrent type**.

In this region the aorta beyond the bifurcation of the pulmonary artery is placed in front and a little to the left of the trachea, and proceeds towards

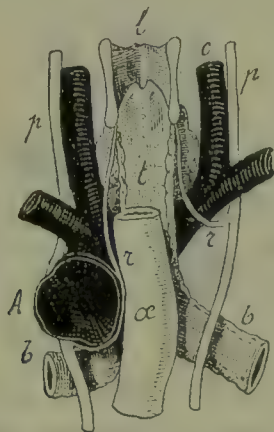


FIG. 23.—ANEURYSM OF THE AORTA, RECURRENT TYPE.

*A*, Section of the aneurysm of the aorta; *b*, bronchus; *r*, recurrent nerve; *æ*, oesophagus; *t*, trachea; *l*, larynx; *p*, pneumogastric nerve; *c*, common carotid artery.

the left bronchus, around which it passes from before backwards, and from right to left. At this point the aorta is in immediate relation with the lower part of the trachea and the origin of the left bronchus; it is contiguous to the air-passages on its concave side and its anterior segment. Accordingly, from the point of view of **prognosis**, aneurysms are here formidable, even when they are of small dimensions, because experience proves that opening of the aneurysm into the trachea or into the bronchi, with most terrible hæmorrhages, occurs **most often** in this region (Ordonneau).

How can the situation of these aneurysms be recognized? Dysphagia, oesophagism (spasms of the oesophagus), pharyngism (spasms of the pharynx), and fits of suffocation and of strangulation (spasms of the glottis), vocal troubles (want of synergy of the vocal cords), and attacks of precordial pain, are among the symptoms which help us in the topographical diagnosis of aortic aneurysm of the recurrent type.

These symptoms are nearly all due to the close relation of the loop of the left recurrent nerve, which serves as a guide to the troubles it occasions.

The importance of this variety of aneurysm will justify the following details :

The two recurrent or laryngeal nerves which arise from the vagus and from the internal branch of the spinal accessory have different relations. The recurrent nerve on the right side arises at the level of the subclavian artery, which it embraces, forming a curve with its concavity upwards. It has no thoracic portion, and therefore usually escapes in tumours of the mediastinum and in aortic aneurysms. The left recurrent nerve, on the contrary, has a thoracic portion, that corresponds to the first two dorsal vertebræ. It arises to the left of the fibrous cord, that represents the obliterated ductus arteriosus, turns round the lower and posterior portion of the arch of the aorta, which it embraces, forming a curve with its concavity upwards, and is therefore exposed to pressure from mediastinal tumours and aortic aneurysms.

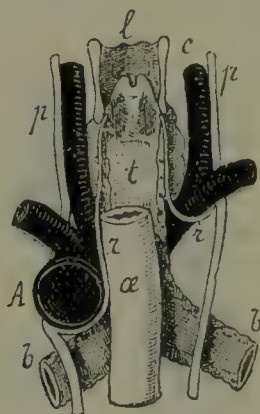


FIG. 24.—RECURRENT NERVES: POSTERIOR VIEW.

A, Arch of the aorta ; æ, œsophagus ; c, common carotid artery ; l, larynx ; t, trachea ; b, b, bronchi ; p, p, pneumogastric nerves ; r, r, recurrent nerves, one of which (the left) coasts the arch of the aorta, and ascends along the œsophagus and the trachea towards the larynx ; the other, in a like manner, coasts the innominate trunk on the right side.

The recurrent nerves give off the following branches (Hirschfeld) :

1. **Cardiac branches**, which arise from the loop of origin of the recurrent nerves, and pass either directly to the cardiac plexus or by their union to the cardiac branches of the great sympathetic and vagus.

2. **Œsophageal branches**, which arise from the left recurrent nerve, and are distributed to the coats of the upper portion of the œsophagus. The lower portions of this tube receive their nerves directly from the vagus.

3. **Pharyngeal branches**, to the inferior constrictor of the pharynx.

4. **Tracheal branches**, which arise in part from the right recurrent nerve.

5. After the recurrent nerves have passed through the inferior constrictor of the pharynx, their terminal branches supply all the muscles of the larynx, except the cricothyroid muscles, which are innervated by the external laryngeal branch of the superior laryngeal nerve.

The recurrent nerves send motor fibres to the larynx, but furnish mixed fibres to the œsophagus, pharynx, and trachea. These different organs, further, receive a direct and indirect innervation from the spinal accessory and vagus nerves. The indirect



innervation is supplied through the intermediary tract of the recurrent nerves; the direct innervation is supplied by branches arising directly from the spinal accessory and vagus.

The researches carried out by French physiologists have established the following conclusions (Cl. Bernard, Chauveau), as regards the respective part played by the spinal accessory and vagus in the complicated movements of these organs:

The vagus is the motor nerve of the œsophagus, either by its direct branches or by the intermediary of the recurrent nerve. The vagus and spinal accessory in part supply the larynx. The inferior constrictor receives fibres from the vagus through the recurrent nerve, and the upper part of the superior constrictor receives a direct branch from the spinal accessory.

Among the muscles of the larynx the larger number, including the constrictors of the glottis and the vocal muscles, properly speaking, are innervated from the spinal accessory through the recurrent nerve. The other muscles are innervated by the pneumogastric, and include the crico-arytenoid muscles, which make the vocal cords tense, and receive their nerve supply directly from the vagus by the exterior laryngeal nerve; and the posterior thyro-arytenoid muscles, which dilate the glottis, and are consequently respiratory muscles, supplied by the vagus through the recurrent nerves.

It is now easy to understand the production of the symptoms present in aortic aneurysm of the recurrent type.

1. The attacks of dysphagia and the spasms of the œsophagus and of the pharynx are explained by excitation of the recurrent nerve, some branches of which pass to the upper part of the œsophagus and to the inferior constrictor of the pharynx. They are accordingly symptoms of excitation, and not of paralysis, for dysphagia due to paralysis of a recurrent nerve would be incomplete, and would produce continuous embarrassment. There would not be paroxysmal attacks of pain, which are proper to muscular spasms.

2. The attacks of suffocation and strangulation, as well as the vocal troubles, are also explained by excitation of the recurrent nerve. For a long while these troubles, due to dyspnoea, were not clearly understood. The authorities, who considered them due to lesions of the recurrent nerve, were near the truth, though they were wrong in referring them to paralysis of the nerve and to consequent relaxation of the vocal cords. Krishaber, in 1866, in a paper "On the Occasion for Tracheotomy in Aneurysms of the Aortic Arch," was the first to give the true interpretation of this dyspnoea. This monograph, in which the author shows himself to be equally expert as an experimenter and as a clinician, deserves to be quoted *in extenso*.

The attacks of suffocation that supervene in persons suffering from aneurysm of the aorta, are not due to paralysis of the recurrent nerve, as had been supposed. The nerve, instead of being paralyzed, is stimulated by its close relation to the aneurysmal sac. The muscles of the larynx are in a state of contraction as a result of this stimulus, instead of being relaxed. The glottis is in consequence narrowed spasmodically, and the embarrassment in breathing is due to this narrowing. This statement has been confirmed by experiments and by laryngoscopic examination.

Section of one recurrent nerve in an adult animal produces relaxation of the corresponding vocal cord, with dysphonia, but the respiration is in no way affected. In a similar manner, paralysis of one recurrent nerve in man produces relaxation and immobility of the corresponding vocal cord, but suffocation does not follow. Paralysis of the recurrent nerve is sometimes seen in aortic aneurysm, and Potain has published a case in which paralysis of the vocal cord, recognized with the laryngoscope, confirmed the diagnosis of an aneurysm of the aorta.

When, on the contrary, the recurrent nerves of the animal are stimulated, the intrinsic muscles of the larynx enter into action, and as the constrictor muscles act

chiefly upon the single dilator muscle, the glottis becomes constricted, and the animal is suffocated.

A similar phenomenon occurs in man. How, then, can stimulation of **one** recurrent nerve affect both vocal cords at once, and produce spasms of the glottis? How can **unilateral** excitation produce a **bilateral** effect? Paralysis of one recurrent nerve causes paralysis of the corresponding vocal cord. Why, then, does excitation of a single nerve, such as occurs in aortic aneurysm, or in any other mediastinal tumour, produce spasm of both lips of the glottis and attacks of suffocation? Krishaber has given us an explanation of this interesting fact by studying the rôle of the arytenoid muscle.

This muscle, which is single and inserted on the two edges of the thyroid cartilages, always has the effect of bringing its two insertions together when it contracts. It is the only muscle in the economy which exerts its action simultaneously on both sides of a symmetrical organ. Accordingly, excitation of one of the recurrent nerves determines, on the one hand, constriction of the interligamentous glottis by the action of the lateral crico-arytenoid muscles and of the thyro-arytenoid muscle on the corresponding side, and, on the other hand, the complete occlusion of the respiratory glottis by the bilateral action of the arytenoid muscle. Respiratory and vocal troubles result from this action.

These experimental results have been verified with the laryngoscope in a patient during an attack of suffocation caused by an aortic aneurysm. The whole left cord (side on which the recurrent nerve was stimulated) was drawn over to the middle line. The right vocal cord remained in its normal position, as regards its anterior two-thirds, but the space between the arytenoid cartilages—that is to say, the respiratory glottis—was completely closed. Extreme embarrassment of respiration resulted from this spasm of the glottis, and the voice was altered in character. The alteration of the voice was due to the tetanization of one vocal cord, to the forced approximation of the arytenoid cartilages, and to the respiratory distress. In another case the left vocal cord was parietic, while the voice was hoarse and bitonal. During the attacks of suffocation, however, the laryngoscope showed that the two cords were in a condition of spasm, and almost completely obliterated the glottis. The spasm affected **both** vocal cords, although the left nerve (as was verified at the autopsy) was alone affected by the aneurysm.

This research explains the attacks of suffocation, strangulation, dysphonia, aphonia, pharyngism, and oesophagism which often accompany aneurysm of the recurrent type. These symptoms may appear alone, in succession, or in combination. It is difficult to explain why the presence of a tumour in the region of the recurrent nerve produces at one time paralysis and at another time excitation. It would seem, theoretically, that the nerve would be excited at first, while its fibres are still intact, and paralyzed later, when the fibres are in part destroyed by the pressure of the tumour. Nothing of the kind, however, occurs. In some cases the paralytic stage is not preceded by a phase of excitation, and in other cases almost complete destruction of the recurrent nerve has been found post mortem, although the patient has shown no recurrent symptoms. It is also difficult to say why the symptoms of excitation, suffocation, and strangulation come on in the form of intermittent attacks, when the determining cause is in continuous action. Such an occurrence is fairly common in troubles of the nervous system, which frequently assume an intermittent form, although the provoking cause is continuous.

However this may be, the symptoms allow the localization of an aneurysm in the neighbourhood of the left recurrent nerve—that is to say, in the region where aneurysm is most grave, because it so often opens into the trachea and the bronchi. The aneurysm may not be as large as an egg or a nut, but it is very serious when situated in this region.



**Termination.**—Aortic aneurysm takes several years to develop ; recovery is unfortunately the exception, and death occurs in various ways. In about half the cases death supervenes without rupture of the aneurysm : the patient dies from pneumonia, for suppurative pneumonia is frequent in aortic aneurysm, from pulmonary phthisis ; from asphyxia, consecutive to compression of the trachea and bronchi ; from complications caused by compression of the pulmonary vessels or of the vena cava ; or sometimes from attacks of angina pectoris, as in two cases which I have recently seen.

When death results from rupture, the opening involves, in order of frequency, the trachea, the bronchi, the pleura, the pericardium, the lungs, the œsophagus, and, very rarely, the skin.\*

When an aortic aneurysm opens into the trachea or a bronchus, the resulting **hæmoptysis** is sometimes so severe and sudden that death occurs in a few minutes. I have seen sudden death on two occasions. In other cases, however, the aortic aneurysm, whatever the point of rupture, may cause a series of small hæmorrhages, that lasts for days and weeks before inducing death. In a certain number of cases the patient, for several days and on several occasions, brings up bloody sputum, which is the prelude of fatal hæmoptysis.

Rupture into the pleura is fairly frequent ; the blood may filter slowly into the pleura producing a pleural hæmatoma, the origin of which is sometimes misunderstood. Thoracentesis is performed once or twice, and when the patient dies, we are surprised to find post mortem an effusion of blood into the pleura consecutive to the opening of an aortic aneurysm.

Rupture into the **pericardium** is not rare, as Godart has collected forty-seven cases.

\* The relative frequency of these perforations is as follows (Ball and Charcot, "Dictionnaire des Sciences Médicales," tome v., p. 546) :

|                                               |    |    |    |    |    |    |
|-----------------------------------------------|----|----|----|----|----|----|
| Right auricle                                 | .. | .. | .. | .. | .. | 1  |
| Pericardium                                   | .. | .. | .. | .. | .. | 8  |
| Pulmonary artery                              | .. | .. | .. | .. | .. | 2  |
| Pleura {left                                  | .. | .. | .. | .. | .. | 11 |
| {right                                        | .. | .. | .. | .. | .. | 8  |
| Anterior mediastinum                          | .. | .. | .. | .. | .. | 8  |
| Left lung ..                                  | .. | .. | .. | .. | .. | 6  |
| Trachea ..                                    | .. | .. | .. | .. | .. | 3  |
| Bronchi {left                                 | .. | .. | .. | .. | .. | 3  |
| {right                                        | .. | .. | .. | .. | .. | 1  |
| {both                                         | .. | .. | .. | .. | .. | 3  |
| Æsophagus ..                                  | .. | .. | .. | .. | .. | 3  |
| Duodenum ..                                   | .. | .. | .. | .. | .. | 1  |
| Peritoneum ..                                 | .. | .. | .. | .. | .. | 2  |
| Subperitoneal tissue ..                       | .. | .. | .. | .. | .. | 5  |
| Cellular tissue of the anterior thoracic wall | .. | .. | .. | .. | .. | 2  |
| Rupture externally ..                         | .. | .. | .. | .. | .. | 4  |
| Death without rupture                         | .. | .. | .. | .. | .. | 54 |

Rupture of the aneurysm through the **skin** takes place under different conditions ; the perforation may occur externally, or may be subcutaneous and cause wide effusion of blood.

**Diagnosis.**—Aneurysm of the thoracic aorta and chronic aortitis, with atheroma and general dilatation of the aorta, have many symptoms in common : pain, dyspnœa, attacks of angina pectoris are seen in both cases, as well as rupture of the aorta and consecutive hæmorrhage. An aneurysm, however, forms a prominent tumour, which gives rise to distinctive signs ; the tumour alters the character of the radial pulse, gives the sensation of two hearts beating in the chest, and determines compression of the trachea with stridor ; compression of the veins with œdema and varicose dilatations ; and compression of the recurrent nerve with the symptoms already described.

**Tumours of the mediastinum** also cause pressure symptoms, but they do not present a double impulse beat, the murmurs of aneurysm, or the inequality and want of synchronism in the two pulses.

It is also necessary to make the diagnosis from **arterio-venous** aneurysm, for the aorta may communicate with the superior vena cava, the auricles, the pulmonary artery, and the right ventricle ; but diagnosis is sometimes very difficult, for the symptoms are, as a rule, simply an exaggeration of those already produced by compression of a venous trunk—*i.e.*, œdema, cyanosis, collateral circulation, and tendency to coma. On auscultation a continuous blowing murmur, with systolic reduplication, may be heard.

Lastly, the diagnosis between aneurysm of the aorta and that of the innominate artery must be made. In the latter case the distinctive signs are as follows : Prominence of the right clavicle ; bulging and dullness in the right clavicular region ; pulsation and murmurs in this region ; compression of the veins on the right side, with stasis in the right jugular vein, cervical and tracheal pains, which are more marked on the right side.

**Treatment.**—The **treatment** of aneurysm of the aorta is general and local. Local treatment of the sac by electro-puncture has given good results, and this means, which is still on trial, has appeared to be of some efficacy. A definite opinion as to injections of gelatine cannot yet be given (Lancereaux). Mercurial treatment will be considered under Syphilitic Aneurysms.

## VII. SYPHILIS OF THE AORTA—SYPHILITIC AORTIC ANEURYSMS.

At the Hôtel-Dieu\* I have devoted three lectures to this important question ; they will serve as my material for this section.

**Discussion.**—Syphilitic infection may cause numerous lesions, including subacute and chronic aortitis, atheromatous and gummatous degeneration,

\* " Syphilis de l'Aorte," *Clinique Médicale de l'Hôtel-Dieu*, 4<sup>me</sup>, 5<sup>me</sup>, et 6<sup>me</sup> leçons, 1897.



lesions of the aortic orifice and sigmoid valves, large aneurysms, sacciform aneurysm of the recurrent type, small aneurysms which may be multiple and cupuliform, coronaritis obliterans, and coronaritis with miliary aneurysms.

Aortic syphilis does not always show itself by diffuse lesions; in many cases the lesions are localized to a segment of the vessel, forming a kind of **segmentary aortitis**, which is somewhat analogous to syphilitic segmentary arteritis. Distinct clinical types result, and show a special group of signs and symptoms. It is therefore not enough to describe as a whole the syphilitic changes in the thoracic aorta; it is necessary, as far as possible, to divide them into certain forms, and I shall make an effort to do so in this section.

It must, however, be understood that the aortic lesions which we are about to study, are syphilitic in nature, and no doubts must be left in the mind as to this fact.

The recognition of syphilitic arteriopathies is of recent date. Not long ago savants doubted the action of syphilis upon the arteries, and authorities are still found whose opinions differ from those which I shall now state as to the action of syphilis on the aorta. That syphilis may attack the arteries, just as many infectious diseases do, need cause no surprise, and it is clear that syphilitic arteritis must enter into the list of infectious diseases of the arteries. The proof is, however, wanting, both as regards the pathogenic agent of syphilis, which we do not know,\* and also as regards the character of the arterial lesions which often have no special nature; but, on the other hand, we find conclusive proofs and arguments in the efficacy of specific treatment, and also in other considerations.

When a patient suffering from tertiary lesions is at the same time affected by syphilitic arteritis, whether it be obliterating or dilating, in parts which are **accessible to the sight** (limbs and face), and when under specific treatment he recovers from the tertiary lesions and from the arteritis, it is evident that the arteritis in question was of a syphilitic nature.

When an individual who is still young, and has no other cause for arteritis or for atheroma, contracts syphilis, and **in the early months** of his infection is affected by cerebral arteritis, which sometimes results in obliteration of the artery, with all its consequences, or at other times in aneurysm, with rupture (see section on Cerebral Syphilis), a direct relation between the syphilis and the lesion of the artery cannot be denied.

Now, syphilis, which has a marked predilection for the cerebral arteries, and also injures the peripheral arteries—*i.e.*, the temporal, radial, popliteal, femoral, or innominate trunks—has no reason for sparing the aorta, and, in fact, it does not do so. When aortitis, with aneurysm, occurs in a young man about six years after syphilitic infection, and no other causes capable of explaining the aortitis can be found, it is rational to consider the lesion of the aorta as due to syphilis (case of Kalindéro and Babès). When an individual is seized four, six, or ten years after infection with agonizing pains, due to aortitis, which is cured by specific treatment, it is natural to consider both the angina pectoris and the lesion of the aorta as due to syphilis (cases of Hallopeau,

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\* Cf. p. 495, where mention is made of the spirochæta.—TRANS.

Rumpf, Vincenzo Vitone, and personal case). When obliterating arteritis of the Sylvian artery occurs in a syphilitic patient, and yields to specific treatment, it is rational to attribute this cerebral arteritis to syphilis, and if this individual some years later suffers from aortitis with aneurysm, it is also natural to attribute the aortic lesion to syphilis (personal case). When aortitis, with its train of symptoms, appears in an individual suffering from pustulo-crustaceous syphilides, it is very difficult not to doubt that there are two tertiary manifestations, which occur simultaneously or in succession, affecting the skin on the one hand and the aorta on the other (personal cases). When syphilitic perforation of the roof of the palate is seen in a woman suffering from aortic aneurysm, it is only right to admit that in such a patient the tertiary manifestations of syphilis have attacked successively the aorta and the roof of the palate (Jaccoud's case). When aneurysm of the aorta supervenes in a syphilitic patient who has lesions of the parietal bone, liver, and testis, it may be affirmed that the aortic lesion is also of syphilitic origin (Buehle's case).

These arguments appear to me to be decisive. The aorta is not immune to syphilis. It may be added that these effects are generally tardy. In this respect there is some difference between syphilis of the cerebral arteries and syphilis of the aorta. In short, while syphilis may affect the arteries of the brain at a very early stage—even five or six weeks after infection—I do not think that such an early onset has ever been seen as regards the aorta. When I recall, one by one, my cases of syphilitic lesions of the aorta, I find that these lesions have appeared only at a very advanced stage. In three of my patients at the Hôtel-Dieu, syphilis of the aorta supervened from fourteen to twenty years after the chancre; in two of my patients at the Necker Hospital, the aorta was infected only at an advanced period; in one patient at the Saint-Antoine Hospital the first signs of aortic aneurysm appeared eighteen years after infection; in one of Duguet's patients the aortic lesions supervened twenty years after infection. Mauriac considers that aortic lesions appear, as a rule, about twelve years after the chancre, and I place among the earliest the case of Kalindéro, in which syphilitic aneurysm of the aorta came on seven years after, and the cases of Rumpf and Vincenzo Vitone, in which syphilitic aortitis supervened four years and six years after infection. These facts are of importance, and serve to emphasize the fact that, if cerebral arteritis is to be feared during the first year of infection, there is no fear of aortitis for many years.

**Division.**—We are now clear as to the existence and the time of appearance of syphilitic lesions of the aorta; let us discuss in detail each of these lesions and their localizations. From the pathological point of view they differ but little from the lesions of common aortitis: atheroma is found in its different forms and stages; the same deformities of the vessels and the same tendency to dilatation and to aneurysm are seen. There is nothing, either in the anatomical or in the histological picture, that can serve to differentiate the lesions of chronic syphilitic aortitis from those of aortitis due to other causes. An interesting point, however, is that the aorta is sometimes the seat of true gummata, which may give rise to small cupuliform aneurysms, regarded by some authors as characteristic of syphilis.

These various lesions, including thickening, induration, atheroma, gummata, dilatation, aneurysms, etc., are sometimes diffuse and indefinitely distributed over the thoracic aorta; at other times **they are localized to a clearly defined region**, and present the following anatomical and clinical types:



1. Suprasigmoid syphilitic aortitis.
2. Syphilis of the aortic orifice with incompetence.
3. Large aneurysms of the aorta.
4. Aneurysm of the recurrent type.
5. Small multiple cupuliform aneurysms.
6. Obliterating coronaritis and miliary aneurysms of the coronary arteries.

This classification is far from being final, and does not include diffuse aortitis of the thoracic trunk, but it is perfectly applicable to a fairly large number of cases in which the aortitis is localized to a circumscribed region of the aorta.

### 1. Suprasigmoid Syphilitic Aortitis.

In this type the lesion is confined to the first portion of the ascending aorta, thus forming a kind of segmentary aortitis, that may be called **supra-sigmoid**.

In this form we find pain and angina (*angor pectoris*). The pain, which may be slight or violent, transient, continuous or paroxysmal and acute, with suffocation and a sense of constriction in the chest, radiates like the pain of angina pectoris, and has its maximum in the sternal or in the precordial region, which is painful on pressure.

In a patient with these symptoms the attention is at once called to the aorta. On percussion, no enlargement of the vessel can be made out; on auscultation, no murmur is heard. Under such circumstances, and in the absence of any appreciable lesion, every possible supposition is considered, including neuralgia of the cardiac plexus, smoker's heart, hysteria, cardialgia of tabes—in short, all the causes that are capable of producing cardiac neuralgia; while sufficient consideration is not paid to syphilis, because it may date back ten, twelve, or fifteen years, and, it may be added, because this suprasigmoid aortitis has not yet been sufficiently recognized. For my part, I consider it one of the most frequent manifestations of aortic syphilis. The situation and nature of the pains which it excites are sufficient to attract attention and lead to the diagnosis.

These pains should cause no surprise, and no elaborate theories are necessary to explain them. Let us not forget that syphilitic arteritis is sometimes extremely painful. One of Leudet's patients had very severe pain in the temporal arteries, which were affected by syphilitic arteritis; one of my patients suffered much pain in the radial artery, which was the seat of a syphilitic aneurysm; persons suffering from syphilis of the cerebral arteries (basilar and Sylvian arteries) have at times fearful headache due to the arteritis. The same remark applies to syphilitic aortitis. Why should it not be extremely painful when the aorta adjoins the nerves of the cardiac

plexus, which is always ready to produce the syndrome of angina pectoris ? The agonizing sternal or retrosternal pain which radiates to the neck and left arm may be the result of syphilitic aortitis, especially when the supra-sigmoid segment is affected.

The following cases prove the truth of this view :

About fifteen years ago Potain and myself saw a woman, still young, with angina pectoris. The pains had been coming on for some weeks past, and had finally become very severe. They were continuous, yet broken by agonizing paroxysms, like those of angina pectoris. The aortic orifice was healthy ; the aorta showed no dilatation. She was too young for atheroma ; tobacco, hysteria, and tabes, were out of the question. Treatment proved unsuccessful, though an ice-bag gave some relief. A tertiary ulcer appeared on the right thigh. Specific treatment was at once employed ; the ulcer was soon cured, and the pains of angina pectoris completely disappeared. I ask, What could this case be, except an attack of syphilitic aortitis which had left the aortic orifice intact ?

Some years ago, with Duplay and Ramond, I saw a gentleman suffering from serpiginous ulceration, which had destroyed part of the right thigh. He was finally cured. Five years later, very sharp pain in the cardio-aortic region. Auscultation revealed no lesion of the aortic orifice. I thought of suprasigmoid aortitis. Remembering the serpiginous ulceration, I prescribed specific treatment, which he did not take. Some months later Ramond was called out to see him. He was suffering from most terrible precordial pain, and died in a few hours.

Hallopeau has published a very interesting memoir on this subject, in which he describes syphilitic angina pectoris :

A man of thirty-six years of age, ten years before had had benign syphilis, which was properly treated. He was taken ill one night with acute precordial pain, radiating to the left shoulder. During the next few days the pain recurred several times in the twenty-four hours, and radiated as far as the left elbow and the end of the last three fingers. The symptoms of angina pectoris disappeared completely under treatment with mercury and iodide.

In the monograph to which I have just alluded, Hallopeau has collected the three following cases :

The first, reported by Rumpf, concerns a man, twenty-nine years of age, who, six years before, had had chancre, followed by secondary symptoms, when he felt violent pain in the precordial region, radiating into the back and the left arm. The pain came back in the form of attacks, and was accompanied by a feeling of thoracic constriction and palpitations. The attacks at first were some days apart ; later they occurred daily. Auscultation revealed no abnormal sign at the aortic orifice, proving that the supra-sigmoid aortitis had not encroached upon it. The age of the patient excluded atheroma, and syphilis could alone be held guilty. Treatment confirmed the diagnosis, and recovery finally occurred.

The other two cases are given by Vincenzo Vitone. One of his patients, thirty-four years of age, was, four years after infection, taken ill with headache and vertigo, and later with crises of angina, suffocation, and precordial pain, which radiated to the left arm. These attacks occurred several times a day. On auscultation no lesion of the aortic orifice was found, doubtless because the aortitis was confined to the suprasigmoid segment. The condition was due to syphilis, because subcutaneous injections of mercury arrested the attacks.



The other patient suffered from attacks of angina pectoris, which recurred several times a day, although on auscultation it was impossible to discover the slightest lesion of the aorta or of the heart. In this case too the angina pectoris, which certainly depended on suprasigmoid aortitis, was cured by subcutaneous injections of calomel.

These cases prove that syphilitic aortitis may be extremely painful, and, just as arteritis in cerebral syphilis shows its presence by headache, so aortitis shows its presence by the symptoms of angina pectoris, from the most mild to the most severe forms. As long as the lesion remains limited to the suprasigmoid segment of the aorta, the valves are not invaded, and the orifice remains healthy; the symptoms of angina pectoris are the only evidence of the lesion, but they are quite sufficient to indicate the diagnosis.

I cannot lay too great stress on suprasigmoid aortitis, which is a favourite localization of syphilis of the aorta. In a patient suffering from angina pectoris without atheroma, lesions of the aortic orifice, or dilatation of the aorta, we are too often inclined to diagnose neuralgia due to tobacco, hysteria, or arthritis, and to mistake the true nature of the disease; we accuse the patient of smoking or of drinking too much, cut off his tobacco, tea, and alcohol, and then consider our treatment sufficient. We are wrong. We are not sufficiently familiar with the idea that aortitis may arise ten, twelve, or fifteen years after infection, at a time when the patient himself no longer thinks of it, and we do not remember that aortitis, when it is confined to the suprasigmoid segment, may excite no other symptoms than those of angina pectoris. It is the more important not to make a mistake, because syphilitic aortitis, when taken in time, is curable; if it is left to itself, it may lead to invasion of the sigmoid valves and coronary arteries, dilatation of the aorta, formation of cupuliform aneurysms, or rupture of the vessel.

## 2. Syphilis of the Aortic Orifice—Aortic Incompetence due to Syphilis.

After having described suprasigmoid aortitis, let us consider the changes caused by its extension to the aortic orifice and the sigmoid valves. To give an idea of this process I have only to sketch the history of one of my patients at the Hôtel-Dieu:

A strong policeman, who was forty-five years of age, had for some time experienced such acute cardiac pain that he was unable to work. He could not wear his tunic buttoned, because the least pressure over the sternum was unbearable. Moreover, crises of angina pectoris, with all their train of symptoms, appeared on the least movement, sometimes even without any apparent cause.

For some time life had been intolerable. He could not clean his room, and could only walk very slowly from one tree to another, as policemen are wont to do on their beat, without being seized with angina and suffocation, which pulled him up short. Percussion of the sterno-costal region caused fairly sharp pain. Auscultation showed that the angina pectoris was associated with aortitis and aortic incompetence.

Having made the first step in the diagnosis, it was necessary to find the cause of

the aortic lesion and of the angina. He was not suffering from arterio-sclerosis, in the true sense of the word, and had no renal disease, for he had no albuminuria and no symptoms of Bright's disease. He was an aortic case, with incompetence of the orifice and hypertrophy of the heart.

The aortic incompetence was consecutive to a lesion of the aorta, and not to one of the heart. The sigmoid valves had been invaded from the side of the endarterium, and not of the endocardium. This distinction was established by the fact that aortic insufficiency, consecutive to lesions of the endocardium, and most commonly seen in rheumatic cases, is rarely painful, and may pursue its course for a long while without causing symptoms of angina pectoris. On the other hand, aortic insufficiency consecutive to lesions of the aorta is preceded or accompanied by symptoms of angina.

It was not sufficient, however, to diagnose aortitis and aortic insufficiency; it was also necessary to know what was the cause of these lesions. He was free from any infectious disease which might cause aortitis, but seventeen years before he had had a chancre, followed by enlargement of the inguinal glands, mucous patches in the mouth, etc. I was justified, therefore, in making a diagnosis of syphilitic aortitis with valvular incompetence. The treatment confirmed the diagnosis. I gave fifteen injections (oily solution of biniodide of mercury), and later a fresh series of twelve injections. The improvement was so marked that six weeks after admission he returned to duty. He came back every year for treatment with mercury, and the acute symptoms never reappeared.

This case is a typical one of aortic insufficiency, associated with syphilitic aortitis. The lesion of the orifice was not improved by the treatment; the diastolic murmur did not diminish, because the lesion of the orifice was irremediable; but, at any rate, the treatment was very effective, as regards the symptoms of pain and dyspnoea, so that we may hope that the lesion will not be fatally progressive, and that it may be arrested.

For seven years I heard nothing of him, but in September, 1905, he came to the Hôtel-Dieu, when I inquired as to his health.

"Sir," said he, "for seven years I have had no pain. I do my own work, and also do day-work for others. My health is grand, and there is not a better worker for miles round. I dig all day, and in summer I do harvesting from four in the morning till eight at night."

Such was his statement. Seven years ago he had been under treatment for such severe angina that death appeared imminent. Fortunately, the angina was due to syphilis, and injections of biniodide of mercury worked a wonderful cure. The valvular lesion will always remain as marked as it was seven years ago, but we may say that it has become harmless. He has gone back to Ardèche to work. Perhaps I shall find him still in good health in years to come.

### 3. Syphilitic Aneurysms of the Aorta.

My opinion is that aneurysms of the aorta are in most cases of syphilitic origin. I recognize more than ever that syphilis has a marked predilection for the aorta. Too much importance cannot be given to early diagnosis. All treatment depends upon it.

Syphilitic aortitis may result in dilatation of the vessel and in aneurysms of every size. They may assume considerable proportions, as in a man suffering from aneurysm of the arch.

The first warnings were of two years' duration. He felt acute pain in the left cervical region and the left arm, without any symptom of angina pectoris. He had



so little distress that he could, without the least breathlessness, ascend several stories, while carrying heavy weights. The pains which he experienced were not like those seen in cases of aortitis of the preceding type. It must be added that the localization of the lesion was quite different; and while, in the other patients, the suprasigmoid aortitis reacted on the branches of the cardiac plexus in the form of angina pectoris, here the aneurysm of the terminal portion of the arch left the cardiac plexus intact, but affected the branches of the brachial plexus.

When this cervico-brachial neuralgia appeared, he consulted Fournier, who diagnosed syphilitic aneurysm of the aorta. He had had a chancre, followed by secondary symptoms. In 1884 he was treated for syphilitic endarteritis obliterans of the left Sylvian artery at the Saint-Antoine Hospital. At this time, after violent headache, right hemiplegia with aphasia, appeared. I saw the patient some days later, and at once prescribed mercurial inunctions and iodide of potassium. The aphasia and hemiplegia finally ended in complete recovery.

Ten years later he suffered from aortitis of the same nature, which ended in aneurysm. These details were known to Fournier, who at once recognized the mark of syphilis, which, ten years apart, affected the Sylvian artery and the aorta. The proof that the aortitis was really syphilitic is that mercury and iodide of potassium, prescribed by Fournier, cured the cervico-brachial neuralgia, which no other treatment had been able to assuage. The aneurysm underwent no change, for it was the result of two lesions which do not retrocede, but the extreme pain caused by the aneurysm yielded to the specific treatment. He came to see me for the neuralgia, which had again become intolerable. Under the influence of specific remedies the pains and insomnia disappeared.

A patient, thirty-six years of age, was admitted into the Saint-Antoine Hospital for an enormous syphilitic aneurysm of the aorta. At the age of fifteen he had contracted syphilis, which was followed later by tertiary ulcerations, with indelible scars and painful exostoses. Sixteen years after the chancre, pains appeared in the right shoulder, and were more intense at night. Later they spread over the whole arm and right side of the chest. Six months later, œdema of the chest-wall on the right side and venous dilatation. Millard, finding pulsation to the right of the sternum, diagnosed a syphilitic aneurysm of the aorta, and prescribed Gibert's syrup, with iodide of potassium. The œdema and the collateral circulation disappeared, but the severe pains soon returned, and the patient was taken into my ward. At the right of the sternum, in place of the upper ribs, which had disappeared, I found a large aneurysm. The pains in the arm and shoulder were so sharp that the patient could get no rest. It was clear that he had suffered from a series of syphilitic troubles, ending in aneurysm of the aorta. I ordered mercurial inunction and iodide of potassium. The pains diminished, the nights became better, but no evident change in the aneurysm could be seen. He died suddenly from fulminating hæmoptysis.

The autopsy revealed enormous dilatation of the ascending aorta and of the arch, the walls of which were thickened. The sac was studded with small secondary aneurysms; some were hollowed out like a cup, while others projected in the form of nodes, due to the stratified layers of fibrin which filled the cup and were raised above the wall of the artery. Indeed, the sac contained a crop of small aneurysms, due to circumscribed gummata, as has been described by Letulle, Kalindéro, and Babès.

These tumours may occur at any point of the thoracic aorta. The lesions found post mortem show slight differences. In some cases the process is disseminated over the aorta, which is dilated, thickened, and studded with atheromatous patches at different spots; in other cases the aortitis is limited to a segment upon which the aneurysm develops later, and the vessel is almost normal in the rest of its extent.

#### 4. Aneurysm of the Aorta of the Recurrent Type.

Let us now consider the lesion which I have named aneurysm of the **recurrent type**. I have employed this name because the aneurysm develops in the region of the aorta contiguous to the loop of the left recurrent nerve, and because its most prominent symptoms depend upon the close relation of this nerve.

The anatomical, clinical, and experimental details of this variety of aortic aneurysm have been described in the preceding chapter, to which I refer the reader.

Dysphagia, attacks of pharyngism, œsophagism, suffocation and strangulation, and vocal troubles, localize the aneurysm in the neighbourhood of the recurrent nerve. This situation is especially serious, because rupture into

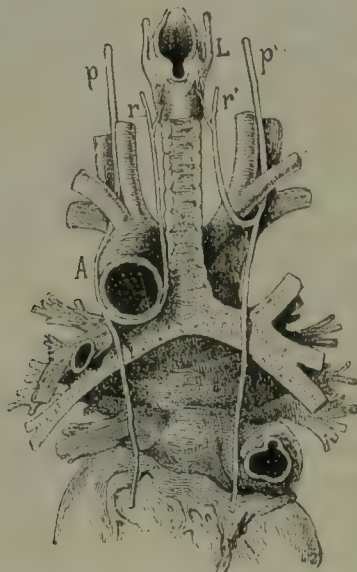


FIG. 25.—ANEURYSM OF RECURRENT TYPE.

*L*, Larynx ; *p*, *p'*, vagus nerve ; *A*, aorta ; *r*, *r'*, recurrent nerves.

the trachea and bronchi most frequently occurs here. The aneurysm may not be larger than a nut, but it is very serious when situated in this region, as the following cases prove.

The first of these cases is taken from Ordonneau's thesis.

A man, fifty-eight years of age, came into the Hôtel-Dieu at Nantes for violent pain in the neck and upper part of the chest. His respiration was embarrassed, and inspiration was characteristic of spasm of the glottis. The voice was hoarse ; swallowing was painful. As he felt better, he left the hospital, but returned soon after in a much worse state. He was cyanosed, the aphonia was complete, the dysphagia was more severe, and the attacks of oppression were terrible. As he was syphilitic, we thought of laryngeal syphilis, with consecutive œdema. Tracheotomy was performed. He succumbed during the night.

At the autopsy the larynx was found free from disease. At the lower part of



the trachea, on its left side, there was an aneurysm of the arch of the aorta, no larger than a nut. The aneurysm was adherent to the trachea, and was skirted laterally by the left recurrent nerve. The portion of the trachea which was not in contact with the aneurysm showed on its inner surface an ulcer a few millimetres in diameter. At this point the aneurysmal sac was partly formed by the trachea, and was so thin that it would certainly have opened into the air-passage in a short time.

This case is an exact reproduction of the statements made above. The syphilitic aneurysm was of small dimension, and did not compress the trachea or the œsophagus. The dyspnœa and dysphagia, therefore, were not produced by pressure on these tubes. Its existence was revealed by aphonia, dysphagia, spasms of the glottis, and asphyxia, due to the close relation of the recurrent nerve. Even if the patient had survived these complications, he was threatened by fatal hæmorrhage, for the aneurysm was on the point of bursting into the trachea.

Savard has published a similar case :

A soldier, forty-four years of age, who had contracted syphilis, was many years after taken ill with vocal troubles and dyspnœa. Later the aphonia became complete, and the difficulty in breathing was complicated by terrible attacks of suffocation and sucking-in. As auscultation of the chest, the heart, and the aorta, gave negative results, the possibility of syphilis affecting the bronchial glands was thought of, and iodide of potassium prescribed. The situation, however, continued to grow serious, and on the second day after admission he died from hæmoptysis in less than a minute. The autopsy revealed an aneurysm of the size of a nut, forming a diverticulum, attached to the aorta. The aneurysm, which was adherent to the left bronchus and to the trachea, compressed the left recurrent nerve. After the trachea and bronchi had been opened, Savard found at the origin of the left bronchus a perforation, establishing communication between the bronchus and the aneurysm. The hæmoptysis had taken place through this opening. This case, which is absolutely typical, proves the gravity of aneurysms, even of small size, when they are near the loop of the recurrent nerve. It further proves that these aneurysms may reveal themselves by none of the usual signs, and may only betray their presence by the signs special to aneurysms of the recurrent type.

When the aneurysm develops in this region, it almost completely escapes our methods of investigation, especially if it is of small size ; it causes no expansile pulsation, no double impulse, no dullness, and no murmurs ; its presence is not always shown by pain. In some cases nothing can be seen, heard, or felt, but yet we can arrive at the diagnosis of the aneurysm by the symptoms I have indicated.

Kalindéro and Babès have well brought out the rôle of gummatous aortitis and circumscribed gumma in the pathogenesis of syphilitic aneurysm of the aorta. Among their cases there is one which absolutely conforms to the recurrent type.

A physician, twenty-nine years of age, had contracted syphilis seven years previously. Treatment had for a long time kept him free from any manifestations of syphilis, when, six years after the infection, he was taken ill with dysphonia, cough, and severe dyspnœa. The physicians at Bucharest, after laryngoscopic examination, were of opinion that the trachea and recurrent nerve were being compressed by a small aneurysm or by enlarged glands. In a few months the laryngeal symptoms disappeared, the

attacks of suffocation were averted, he resumed his work, and his health appeared perfect. One day, without any warning, he died from **fulminating hæmoptysis** in a few minutes. Before his death, however, he had still the time and the astonishing presence of mind to write a few lines to Babès, begging him to make a post mortem and preserve the specimen.

The autopsy demonstrated the existence of patches of syphilitic aortitis and of a sacciform aneurysm as large as a nut, which had developed in the concavity of the arch of the aorta. The aneurysm rested on the left bronchus, and pushed the lower end of the trachea slightly to the right side. The aneurysm had opened into the left bronchus by a perforation 5 millimetres in diameter.

It is easy to reconstruct the different stages in this case. We have a young man who contracts syphilis. Seven years later syphilitic aortitis supervenes, and is chiefly localized to the aortic segment which is in relation with the loop of the left recurrent nerve. At this point an aneurysm of small size develops, and shows its presence by symptoms due to the proximity of the recurrent nerve—viz., dyspnoea, spasm of the glottis, and vocal trouble. After this warning everything becomes normal, but the aneurysm pursues its insidious course, without pain, pulsation, or any visible signs. One day rupture occurs, and is accompanied by fulminating hæmoptysis.

I have seen a similar case, which shows how aneurysm of the recurrent type behaves.

I saw, with Dr. Woelcker, a gentleman, fifty years of age, who for some months had experienced suffocation, with vocal troubles and difficulty in swallowing. Swallowing was painful and almost impossible during the attacks, while the aphonia was complete, and the suffocation so marked that the patient repeatedly thought that he was dying. Between the attacks the voice remained hoarse, deglutition was difficult, and the breathing was far from being normal. Examination of the cardio-aortic region yielded negative results. As the patient was **syphilitic**, I thought of some mediastinal lesion, and aneurysm of the recurrent type came into my mind. I told his family of the possibility of fulminating hæmoptysis. In order to gain further information, however, I asked for a laryngoscopic examination by Dr. Bonnier.

When Woelcker and Bonnier arrived at the house to examine the patient, the servants told them that he had just died of fulminating hæmoptysis. This catastrophe verified the diagnosis, and I have not the slightest doubt that this patient, like those of Kalindéro and Savard, died from rupture of the aneurysm into the left bronchus or the trachea.

Such cases prove that aneurysm of the recurrent type has its special symptoms: dysphonia, aphonia, and attacks of suffocation, of strangulation, of pharyngism, or of œsophagism, with or without precordial pain, are therefore found more or less in every case. These symptoms may occur in succession or in combination. They appear in the form of attacks, and though they may last only a short while as a kind of warning (Kalindéro's case), they are none the less of the highest importance. They allow us to localize the aortic aneurysm in the neighbourhood of the recurrent nerve—that is to say, in a region which is especially serious, and in which opening of the aneurysm into the trachea or into the bronchi most usually occurs. It would therefore be wrong to consider that the largest aneurysms are the most formidable. An aneurysm which develops towards the exterior, and sometimes reaches considerable size, as in the case described above, may last



for several years, in spite of its size, before compromising the patient's life; while small aneurysms which are in relation with the bronchus or with the trachea may cause fulminating hæmoptysis in persons who scarcely suspect the existence of an aortic lesion. By careful study of the symptoms, which I have tried to put clearly, and by laryngoscopic examination, we can as accurately as possible define the topography of aortic aneurysm of the **recurrent type**.

### 5. Cupuliform Aneurysms—Gummata of the Aorta.

Let us now consider syphilitic aortitis with **circumscribed gummata**, which result in **multiple cupuliform aneurysms**, varying in size from a pea to a nut. Several of these small aneurysms may be counted at intervals in the course of the thoracic aorta; we might, indeed, speak of an eruption of small aneurysms. These aneurysms sometimes develop upon an aorta which is neither deformed nor dilated; at other times they are set on the walls of a large aneurysmal dilatation. According to several authors, these small aneurysms are absolutely special to syphilis. The subjoined cases will give an exact idea of this variety.

On November 9, 1892, a man, forty-three years of age, was admitted for precordial pains, with angina and suffocation, which used to come on suddenly after a walk, or after some rather sudden movement, or, indeed, without apparent cause. On several occasions the crisis had been so violent that the patient thought he was dying. The pain started in the precordial region, and radiated into the left arm and hand. The duration was variable. The first crisis had been short, but the later ones came on in rapid succession, and then lasted several hours, practically without any respite. The patient did not dare to walk or to go upstairs: he dreaded lying down, because he was afraid of a reappearance of the crisis.

He evidently had angina pectoris, and it behoved us to search for the cause. When he uncovered for examination, we noticed above the left clavicle a large and absolutely characteristic pustolo-crustaceous syphilide, and learnt that he had been syphilitic for ten years. Percussion and auscultation of the cardio-aortic region gave negative results. The aorta was not increased in size, and the aortic orifice was absolutely healthy. The patient had neither lead-poisoning, gout, diabetes, tabes, nor hysteria, and was not a smoker. As tertiary syphilis was in full activity, it was permissible to diagnose syphilitic aortitis.

On the evening of admission he refused to lie down, so much did he dread a crisis. He was seized with a terrible attack of angina pectoris. He was undressed and put to bed, but he had scarcely lain down, when he jumped up in agony, said that he was dying, and passed away in a few moments. At the autopsy my first care was to examine the aorta. It was not dilated, but on the surface we saw four small aneurysms, at intervals, from below upwards. The first aneurysm was of the size of a large pea, and situated at the level of the right auricle. The second aneurysm, of the same size, was 4 centimetres above the preceding one, in front and to the right of the aorta. The third aneurysm, of the size of an almond, formed a prominence, about a centimetre higher up, at the junction of the ascending and horizontal portions of the aorta. A fourth aneurysm, of the size of a cherry, was situated on the anterior part of the aorta, at the origin of the innominate trunk. The aorta was opened, and the integrity of the aortic orifice and sigmoid valves was proved. The vessel was thickened

and studded with patches of gummatous and atheromatous aortitis. The first and third of these aneurysms were excavated in the form of a thin transparent cup, which would soon have ruptured. The second and fourth aneurysms formed a hard prominent projection, composed of fibrinous clots, adherent to the wall. On histological examination of the thinnest portion of the aneurysm, the tunica media had almost disappeared. The coronary arteries were incised and carefully examined. Their walls showed arteritis at different points, but their orifice was absolutely free, and the lumen was not obliterated.

He had been attacked by acute syphilitic aortitis, which had rapidly caused multiple aneurysms. The aortic orifice and the sigmoid valves had been spared, and death had supervened in an attack of angina pectoris, consecutive to aortitis. Let us remark, in passing, that death was not attributable to cardiac ischemia, consecutive to obliteration of the coronary arteries, for the heart was not ischemic, and the coronary arteries were permeable at every point.

A man, fifty-eight years of age, was admitted for agonizing dyspnoea, which had begun about two years previously, and had soon been followed by precordial pain, radiating into the left arm. Although the pain and the dyspnoea were present together, the latter was the chief trouble. On auscultation of the heart, I found a diastolic murmur of aortic insufficiency, and a systolic one of mitral insufficiency. I therefore made the following diagnosis: Angina pectoris, consecutive to aortitis which had encroached on the aortic orifice, with aortic and mitral incompetence. We had still to discover the cause of this aortitis, and as, after reviewing the probable causes, I could only find one—syphilis, which the patient had had nine years before—it appeared to me natural to put the aortitis down to syphilitic infection. I prescribed mercurial inunctions and iodide of potassium. A few days later the patient was seized with a terrible attack of dyspnoea and pain, which during the preceding days had recurred at closer and closer intervals, and he succumbed in a few minutes.

At the autopsy our attention was first directed to the aorta. It was dilated with patches of chronic aortitis. We counted **seven** small aneurysms at intervals, from below upwards. The first aneurysm was as large as a plum-stone, and situated in front and to the right of the aorta, on a level with the apex of the right auricle. The second aneurysm, of the same size, was situated above the preceding one. The third aneurysm was smaller, and situated higher up. The fourth was situated on the anterior part of the aorta, near the innominate trunk. The fifth aneurysm occupied the anterior part of the aorta, near the left carotid. The sixth was situated close to the left subclavian artery. The size of each of these last three aneurysms was equal to a cherry-stone. Lastly, a seventh aneurysm, of the size of a walnut, occupied the ascending and lateral portion of the aorta.

Some of these aneurysms were cupuliform; others were covered by fibrinous clots, which projected, like nodes, into the lumen of the vessel. Histological examination of the more affected parts revealed complete disappearance of the tunica media. The coronary arteries were carefully examined, **but were not obliterated either at their orifice or in their course.** We found only some atheromatous patches 1 centimetre from the orifice of the posterior coronary. The heart muscle was much hypertrophied, especially in the left ventricle. Bands of fibrous tissue were found on histological examination.

These two cases are very instructive. They prove that aortic syphilis may take the form of circumscribed gummata, with or without other lesions of the vessel. In these cases, as in most syphilitic lesions of the ascending aorta, the angina pectoris was so severe that both our patients died from it. They died, although there was no inflammation of the coronary arteries,



proving that obliteration of the coronary arteries is far from being the only cause of fatal angina pectoris.

The multiplicity of syphilitic aneurysms was shown by Jaccoud in 1886 in his clinical lectures at the Pitié, where he quotes several well-known cases.

These include: the case published by Vallin, of a man forty-five years of age, who was syphilitic, and died suddenly from hæmorrhage into the pericardium. At the post mortem four aneurysms were found on the thoracic aorta.—The case of Orlebard, of a syphilitic patient, twenty-nine years of age, at whose autopsy three aneurysms of the thoracic aorta were found, the first being situated above the posterior sigmoid, the second a little higher, and the third below the origin of the innominate trunk.—Malecot's case of a man, fifty-eight years of age, who was both alcoholic and syphilitic; post mortem three aortic aneurysms were found: one at the origin of the aorta, the second on the arch, and the third just above the diaphragm.

Nalty's case (Verdié's thesis) deals with a man who had had syphilis five years before. One day he noticed a beating at the root of the neck. Six months later a pulsating tumour appeared in the same region, below and a little to the right of the sterno-clavicular articulation. This tumour was an aneurysm. As the patient had tertiary ulcers on the thigh and knee, it was reasonable to ascribe the aneurysm to syphilis, and treatment was prescribed. After transient improvement the situation became worse, and the patient died. At the autopsy two aneurysms were found—one at the level of the innominate artery, the other in the aorta. The latter compressed the trachea behind, and in front projected into the sterno-clavicular region. Besides these two large aneurysms there were upon the aorta several small aneurysms, characterized, says the author, by erosions of the walls of the vessel and gummatous projections. Further, numerous gummata, varying in size from a pin's head to a cherry-stone, were scattered over the surface of the heart and the endocardium.

Jonas has reported the case of a man, thirty-three years of age, who had had syphilis for nineteen years, and three months before was attacked by cardiac and aortic troubles. He died, and at the autopsy enlargement of the whole ascending part of the aorta was found. Its surface was irregular and partly calcified, and 4 centimetres above the aortic orifice eight or nine aneurysms, varying in size from a pea to that of a filbert, were counted.

In syphilitic aortitis, which has been carefully studied by Brault and Letulle, the whole of the arterial wall is infiltrated at a circumscribed spot by embryonic lesions. "Round cells, united at different spots into confluent nodules, have a suspicious appearance. We are generally right at some period or other, if not in recognizing, at least in suspecting, the formation of miliary gummata."

Kalindéro and Babès are still more explicit on the presence of syphilitic gummata in the aorta. According to these authors, the multiple small aneurysms are due to gummata of the walls of the aorta. These authors say that, in addition to aneurysm of the aorta due to sclerous aortitis, we see small circumscribed aneurysms, due to the development of syphilitic gummata in the walls of the vessel.

Syphilitic lesions of the aorta are, like every form of aortitis, liable to secondary infection. Kalindéro and Babès have brought this point out

clearly. They found that microbes invade the inflamed vessel. I quote the result of their researches : " The most internal layer of the tunica intima is a little more easily stained and more uniform than in the normal state, and contains a sheet of microbes in the form of diplococci, or short, very straight bacilli, which stain well with aniline dyes, or, better still, with hæmatoxylin. Nests or larger nodules containing these microbes are found at different places."

### 6. Syphilitic Coronaritis.

Syphilis of the coronary, just as of other arteries, shows two chief forms : we may find obliteration or dilatation with aneurysm.

Letulle has recorded a case of obliterating syphilitic coronaritis:

A woman, thirty-nine years of age, who was syphilitic, with right hemiplegia and aphasia, died from cerebral softening, following obliterating endarteritis of the left Sylvian artery. Syphilitic lesions were found in several of the organs. Letulle found the right heart seamed with fibrous bands, and atrophied in its whole lower half. On section, most of the coronary arterioles in the right myocardium were affected by obliterating endarteritis.

Balzer has published a case of syphilitic miliary aneurysms of the coronary arteries.

The patient was a man, fifty years of age, who showed a tertiary syphilitic ulcer, which had destroyed the septum and subseptum of the nose and part of the upper lip. He died of phthisis, and the autopsy revealed the following lesions: The anterior coronary artery was the seat of about thirty miliary aneurysms, placed in a row on the anterior surface of the ventricle, over the aortic and pulmonary infundibula. These aneurysms were as large as a pin's head, and involved the arterioles of the pericardium. After being detached from the wall of the heart and stained with picro-carmin, they showed every known type, being sacciform, fusiform, and dissecting. The rupture of one of these aneurysms set up fatal hæmorrhagic pericarditis.

Haushalter, at the post-mortem examination of a syphilitic patient who died suddenly while eating a meal, found coronaritis obliterans, characterized histologically by masses developed in the tunica vascularis. The masses exactly resembled gummatous lesions in appearance.

**Treatment.**—Mercury is the sovereign remedy for syphilitic aortitis; it may be given in conjunction with iodides. I prefer oily or watery injections of biniodide of mercury. An injection containing  $\frac{1}{6}$  grain of biniodide is given daily for a fortnight. The treatment is stopped for a time and then repeated. Iodide of potassium may be prescribed during the intervals when mercury is not being given.

Under the influence of this treatment (which is described in the Appendix), we often witness the disappearance of the acute pains of syphilitic aortitis, and of the headache due to cerebral arteritis; we can also master cervico-brachial or intercostal neuralgia, which sometimes accompanies the growth of aneurysm. The dyspnoea, vocal troubles, dysphagia, spasms of the glottis, and attacks of oppression, resulting from an aneurysm in the



neighbourhood of the recurrent nerve, may disappear, or, at any rate, become less severe.

The efficacy of the treatment is proved by the cases which I have quoted. My patient at the Hôtel-Dieu, in whom angina pectoris was most severe, was completely relieved by the treatment, and was able to resume his duties as a policeman. My other patient, in whom the aneurysm formed a large thoracic tumour, and who had intractable cervico-brachial neuralgia, was completely freed from his pain by the treatment which Fournier had already prescribed at the Saint-Louis Hospital, and which I continued at the Hôtel-Dieu. The patient whom I saw with Potain had syphilitic angina pectoris, in which treatment had failed; he was completely relieved by mercury and iodides. The patients of Hallopeau, Rumpf, and Vincenzo Vitone also suffered from syphilitic angina pectoris, and were cured by mercurial preparations, given either by the mouth or by injections. The patient whom I had at the Saint-Antoine Hospital, and in whom Millard had already diagnosed syphilitic aneurysm of the aorta, lost his dyspnoea, brachial neuralgia, and oedema under treatment with mercury and iodides. In one of Jaccoud's patients treatment brought about an improvement, which lasted two years, "and would perhaps have been final if it had not been abandoned too soon."

In Nalty's patient the treatment produced a very notable diminution in the dyspnoea and cough, and for some time the pulsations of the aneurysms were less intense, and the tumour seemed to diminish in size. In short, dyspnoea, cough, and especially pain, improve or disappear under the influence of early treatment.

When such a result has been obtained, we ought not to remain satisfied. Syphilis is naturally obstinate, and does not yield easily; let us beware, therefore, of considering as a cure what is most often a momentary improvement. Treatment may have mastered the pains or other symptoms which accompany aortitis at its onset, as well as at a more advanced period, but this is not sufficient. Even when these symptoms do not reappear, and, much more so if they do, let us begin treatment again for a second or a third time, the duration of such treatment being from fifteen to eighteen days on an average, and the periods that divide the treatment being some weeks, or at most some months.

If we are thoroughly imbued with these principles, and have the opportunity of attacking syphilitic aortitis at its onset, we can obtain therapeutic successes; we can judge of this by the results I have just given. If, however, the aortic lesions are already old, and an aneurysm has already appeared, are we to abstain from all specific treatment? Such is not my opinion, and although we cannot count here on success, we have at any rate the satisfaction of being able to improve the patient's condition, to avert imminent danger, and to arrest the invasion of the lesions.

### VIII. DIAGNOSIS BETWEEN ANEURYSM AND NERVOUS PULSATION OF THE ABDOMINAL AORTA.

Aneurysm is far less common in the abdominal than in the thoracic aorta. Lebert, in 69 cases of aortic aneurysm, found—Ascending aorta, 24 ; arch, 27 ; descending aorta, 9 ; abdominal aorta, 9. The enlargement in most cases is near the coeliac axis ; the structure is similar to that of the thoracic aneurysm. The only special point is the frequency of rupture into the subperitoneal tissue, and, as a result, a diffuse aneurysm.

The abdominal tumour has practically the same causation as the thoracic aneurysm, but its localization gives rise to certain special symptoms. The pains may simulate those of lumbar neuralgia ; they may be acute and paroxysmal. Some patients cannot lie down in bed, and are compelled to assume a sitting posture. The pains may be due to pressure on the nerve trunks, or to erosion of the vertebral bodies. The weakness of the legs and the pain make walking difficult. The aneurysm is shown on palpation by a tumour, and by pulsation more readily felt in thin persons. When we press the abdominal wall back, we can feel a pulsating tumour in the subumbilical region, either in the median line or to one side, usually the left. A diastolic murmur is audible on auscultation. The femoral pulse is later than the radial pulse. Rupture usually takes place into the retroperitoneal tissue. Arterio-venous aneurysm is extremely rare. Aneurysm of the abdominal aorta has given rise to numerous errors in diagnosis ; aneurysm may somewhat resemble the epigastric pulsation found in aortic insufficiency, the transmitted pulsation seen in adherent pericardium, and the pulsation of the aorta transmitted by tumours of the pancreas, the stomach, or the small omentum. Abdominal aortitis may also show some resemblance to aneurysm. The usual mistake, however, lies in confusing aneurysm of the abdominal aorta with the so-called nervous pulsation of the vessel. “ Violent pulsation along the course of the abdominal aorta is found in some subjects, while material lesions are absent.” I have frequently seen this nervous pulsation in persons who were afraid that they had an aneurysm, and have pointed out this fact to my class at the Hôtel-Dieu.

Nervous pulsation of the aorta is usually seen in hypochondriacal, neurasthenic, or hysterical persons, in cases of flatulent dyspepsia, and in anæmic women during menstruation or pregnancy. The sufferer usually discovers the pulsation, and comes to the physician, who recognizes, on inspection and palpation, the pulsation in question. On depressing the abdominal wall the hand feels a violent pulsation, which is most marked when the patient is lying down. On auscultation a shock seems to lift the head, and pressure on the vessel with the stethoscope produces a systolic murmur ; no pain is felt.



The pulsation is periodic ; it may last for months, appearing and disappearing suddenly. The condition is not serious, though the sufferer grows alarmed from the violent pulsation, loses sleep, and becomes neurasthenic. Correct diagnosis is therefore important. "Laënnec and Bayle both admit making an error ; the former, profiting by his error, has left us an interesting description of this phenomenon."

In my own cases the following diagnostic points have given me help ; the beats are just as forcible in aneurysm as in nervous pulsation, but in the former case we find a tumour.

Aneurysm is frequently accompanied by lumbo-abdominal neuralgia ; similar pain is not seen in nervous pulsation. Aneurysm runs a slow and progressive course ; the symptoms appear gradually, and the pulsation does not at once reach its maximum ; nervous pulsation, on the other hand, may appear from one day to the next, cease, and reappear periodically.

Nervous pulsation, though important, has been scarcely noticed in the text-books, but it was known to Hippocrates, who states that the son of Eratolos had marked pulsation after an attack of dysentery. The symptom was well known to Bailleu, Morgagni, and Lancisi. Its pathogenesis is still a question of theory. The important point is correct diagnosis and proper treatment.

Two women under my care at the Hôtel-Dieu for nervous pulsation of the aorta were afflicted with flatulent dyspepsia and dilatation of the stomach. I prescribed lime-water before and after meals, and a cold douche every morning. Rapid improvement followed, and the patients left the hospital freed from all symptoms. Valerian, bromides, and hydrotherapy are indicated when the nervous condition appears to dominate the situation.

## IX. CARDIO-AORTIC LESIONS IN TABES—TABETIC ANGINA PECTORIS.

**Discussion.**—Patients suffering from tabes may show lesions of the heart and aorta. This fact, which now appears commonplace, was unknown twenty years ago. Vulpian, in 1879, was the first to note the coexistence of cardio-aortic lesions and locomotor ataxy.

A patient, who for six years had suffered with lightning pains in the lower limbs, also had aortic insufficiency and stenosis. He died, and, on the one hand, the spinal lesions of tabes, and, on the other hand, aortic mischief, were found. The sigmoid valves, which were thickened and shrivelled up, had caused insufficiency and constriction of the orifice. In another of Vulpian's cases, a woman, fifty-one years of age, suffered from tabes, accompanied by a double lesion of the aortic orifice, with dilatation of the aorta and symptoms of angina pectoris. She died, and at the autopsy the medullary lesions of tabes and aortic insufficiency were found. The aorta was very dilated and incrustated, especially at the arch, with numerous patches of atheroma.

This coexistence of tabetic and cardio-aortic lesions had not escaped Vulpian. "I cannot state," says he, "that the relation of cause and effect exists, but I feel that I must call your attention to this coexistence, and Charcot, at the Salpêtrière, has many times in my presence insisted on the frequency of aortic lesions in ataxic patients."

Charcot and Bouchard published in 1866 an analogous case.

Their patient, who was subject to severe attacks of dyspnoea, was affected with the lightning pains of tabes, as well as an aortic lesion with a to-and-fro murmur. He died, and at the autopsy tabes and cardio-aortic lesions were found. Heart much hypertrophied; aorta dilated and incrustated with atheroma; sigmoid valves shrivelled up, incompetent, and studded on their free edge with small warty vegetations.

In 1890 Grasset, who had collected twenty-four cases, including two personal ones, discussed the pathogenesis of cardio-vascular troubles in tabes, and implicated the reaction of the lightning pains upon the heart.

Letulle has published two cases of aortic lesions in tabes, and puts them down to general arterio-sclerosis, which produces both morbid conditions. In 1881 Jaubert collected twelve fresh cases. Truc, in 1883, brought forward six other cases, and Teissier, in 1884, described among atrophic lesions perforation of the sigmoid valves, and compared it to perforating ulcer of the foot. Raymond, in the *Dictionnaire des Sciences Médicales*, thus sums up the question: "The most probable hypothesis regards the aortic and mitral lesions of tabetics as an accidental complication induced by early senility, or developed as the result of such causes as rheumatism, alcohol, and syphilis, which commonly intervene in such cases."

Syphilis occupies the chief place in the pathogenesis of cardio-aortic lesions in tabes. We know how skilfully Fournier sought to prove that tabes is a syphilitic or a parasymphilitic lesion. By the same reasoning it was very natural to suppose that syphilis produces simultaneously the medullary and aortic lesions of tabes. Bouveret had already published the case of a syphilitic patient, who was attacked at the same time by aortic constriction and early tabes; antisymphilitic treatment determined rapid improvement. Schultze, in 1892, saw two cases of aortic insufficiency in syphilitic patients with tabes, and did not hesitate to set down the tabes and the aortic lesion to the account of syphilis. The same opinion has been expressed by Marie.

It is certain that we possess a large number of cases in which aortic and mitral lesions have appeared in the course of confirmed tabes. They may be distinct or associated; aortic lesions, however, are three times as frequent as mitral ones. Nordmann notes twenty mitral as against fifty-one aortic lesions. As regards my personal experience, I have several times seen aortic,



but never mitral, lesions. Rendu has stated the same opinion. Nordmann's statistics are as follows :

|                                                   |   |                                                                                                                                     |
|---------------------------------------------------|---|-------------------------------------------------------------------------------------------------------------------------------------|
| In 55 cases of aortic lesions in tabetic patients | { | Aortic insufficiency, 38.<br>Aortic stenosis, 7.<br>Aortic insufficiency and stenosis, 6.<br>Aneurysms of the arch of the aorta, 4. |
| In 20 cases of mitral lesions in tabetic patients | { | Mitral insufficiency, 10.<br>Mitral stenosis, 4.<br>Insufficiency and stenosis, 6.                                                  |

The present view is that the aortic lesions are, like tabes itself, of syphilitic origin. I do not profess to decide the question of pathogenesis, and shall content myself with the following statement :

If syphilis is indeed the cause of tabes and of aortic lesions at one and the same time, why do the aortic lesions never appear after tabes ? Why does not syphilis cause in some cases tabes, and, later, aortic lesions, and in other cases aortic lesions, and, later, tabes ? I ask, further, why do not people with syphilitic aortitis (and they are numerous) become tabetic ? There is in every case an anomaly which clashes with my opinion. Further, if syphilis really induce, at one and the same time, tabes and aortitis, why does syphilis in a tabetic patient reserve all its blows for the aorta, and why does it spare the other vessels, notably the cerebral arteries, which are so often affected in syphilis ? This is another unexplained anomaly. Lastly, if the aortic lesions of tabetic patients are syphilitic, why are they usually so indolent, while syphilitic aortitis, like angina pectoris, is nearly always painful ? I give these ideas, and although they may not be of such a character as to solve the present question, they at least throw some doubt on the interpretation of the aortic lesions given by the most reliable authors. They tend to take from syphilis the preponderant rôle which has been given to it, and if this rôle is true in tabes, the question of cardio-aortic lesions in tabetic patients appears to me of small import.

**Symptoms.**—The clinical course of the cardio-aortic manifestations in tabes present some peculiarities, and the most prominent fact is that they are often unknown to the sufferers. I cannot do better than compare them from this point of view with the joint troubles in tabes, which, in spite of the marked disorganization of the joint, are usually painless, so that patients can perform every movement without feeling the least pain. This relation between the articular and cardio-aortic lesions is a novel argument in favour of the theory which tends to consider these lesions as trophic troubles. Grasset had clearly seen that the aortic lesions of ataxic patients do not show themselves by the usual symptoms ; they must be looked for, or they will often pass unnoticed. Jaubert, Rendu, Albespy, and Nordmann note the singular tolerance of cardio-aortic lesions in tabes. I have proved the truth

of this assertion, and I have several times found, to my surprise, marked aortic lesions after a long and careful examination of a tabetic patient, who in the course of his interrogatory had in no way drawn my attention to his aorta. These lesions are not, however, always latent. One patient has palpitation, another pain and dyspnœa, but these symptoms are rarely alarming or end in syncope and the agonizing pains of angina pectoris. The cardio-aortic lesions are well borne by the patient, and scarcely seem to make the prognosis of tabes worse. Tabetic patients rarely die from heart trouble.

Some tabetics show true attacks of angina pectoris without aortic lesions. A tabetic patient who formed the subject of one of my clinical lectures\* had violent angina pectoris, without appreciable lesions of the aorta. I thought that this was a case of neuralgia of the cardiac plexus, comparable to those visceral crises which are constantly met with in tabes.

This idea of angina pectoris, which is independent of lesions of the aorta and coronary arteries, and supervenes as a visceral crisis of tabes, appears to me established by undoubted proofs, and is admitted by several writers. These crises would appear to be related, says Leyden, to neuralgia of the cardiac nerve, or neuralgia cordis, according to Romberg's expression. Leyden quotes the following cases in support of his opinion :

A man, thirty-eight years of age, who suffered from locomotor ataxia, had for three or four weeks attacks which began with violent pain in the region of the heart, and lasted about half an hour. During the attack the heart beat as though it would burst, but auscultation revealed no lesions. The heart sounds were pure, the aortic orifice was healthy, the radial artery was normal, and there was no trace of arterio-sclerosis. Another of Leyden's cases is that of a tabetic who suffered at first with laryngeal, and later with cardiac crises, characterized by terrible angina, with pain radiating into the left arm. During the attacks the patient thought that he was going to die. Leyden takes care to remark that on auscultation he found neither murmurs nor abnormal sounds, which shows that this case was one of visceralgia in the true sense of the word.

Berbès had reported an analogous case in a man, fifty-one years of age, suffering from tabes, who was taken ill with typical angina pectoris.

The cardiac crises were preceded by laryngeal crises. In this case, too, it was a question of visceralgia angina pectoris, for no lesions of the heart and aorta were found on auscultation.

Landouzy has published a similar case.

A woman, thirty-nine years of age, who was a proved case of tabes, but not of syphilis, suffered from frequent gastric crises. She was at times seized with attacks of angina pectoris, accompanied by palpitation, precordial distress, and dyspnœa, and a tearing sensation, which started in the sternum and radiated into the left arm. This case was also one of visceralgia, for the patient showed neither signs nor symptoms of aortic lesions.

Groedel has reported the case of a man, forty-one years of age, who, in the course of

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\* Dieulafoy, 1897. "Angine de Poitrine tabétique," 7<sup>me</sup> leçon (*Clinique Médicale de l'Hôtel-Dieu*).



tabes, was seized with violent attacks of angina pectoris. He was awakened one night with a feeling of angina and precordial pain. During the attack his face was pale and covered with sweat, and his pulse was small and rapid. Although these attacks were repeated several times during the year, it was impossible to discover any aortic lesion on auscultation.

Debove has discussed tabetic angina pectoris in detail.

In one patient the attack of angina pectoris was always preceded by girdle pains. The pain appeared in the precordial region, at the level of the third rib, radiated to the left arm as far as the end of the little finger, and was so acute that it seemed to the patient as though "everything stopped still inside him." After this first attack he was advised to give up tobacco. Although he did not smoke during this period, the attacks did not cease. Auscultation revealed no lesion of the heart or of the aorta. Debove accordingly considered these attacks "as a visceral crisis, analogous to those which may affect other organs in tabes; in fact, as the visceral crisis, which is angina pectoris." Further, the patient in question suffered later from crises affecting the anus, testis, and urethra.

I am of the same opinion as Debove and Leyden, and, in opposition to Huchard, I allow the existence of a cardio-aortic visceralgia, which may be fatal; it is a **neuralgia cordis**, independent of aortic lesions and coronaritis. Tabes, which is essentially a painful disease, excites violent crises in the lower limbs, the thorax, the face, and the stomach, in which case the crises resemble the pains of an ulcer; in the kidney, where they simulate renal colic; in the rectum, the testis, the urethra, and the larynx. Why, then, should we deny to the nerves of the cardiac plexus what is so readily conceded to the nerves of other regions and organs?

It is the more rational to admit visceralgic angina pectoris in tabetic patients, as the same patient often has visceral crises in other organs; thus my patient had had gastric, urethral, and rectal crises when he was seized with angina pectoris. In Debove's case the crises of angina pectoris were preceded by girdle pains, and were followed by rectal, testicular, and urethral crises; Landouzy's patient suffered from gastric crises when angina pectoris supervened; one of Leyden's patients had terrible laryngeal crises before angina pectoris appeared. Cardio-aortic visceralgia is, therefore, neither a strange nor isolated fact in the history of tabes; it belongs to the group of other visceralgias, and may precede, follow, or alternate with them.

If, however, it is reasonable from clinical proof to admit tabetic angina pectoris apart from any lesion of the aorta and coronary arteries, have we also undoubted anatomical proofs? We have such proofs, as is shown by the following case, which Vulpian published in the *Revue de Médecine* in 1885:

A man, thirty-three years of age, was taken ill with the first symptoms of tabes at the age of twenty-nine, and from the first had gastric crises. In the course of tabes he experienced complete attacks of angina pectoris, which Vulpian described minutely. Vulpian excluded hysteria, of which he found no stigmata. He, therefore, admitted in this case the existence of tabetic angina pectoris, independent of any cardio-aortic lesion, as examination of the heart and aorta had proved the integrity of these organs.

The patient died of phthisis, and the diagnosis was confirmed. The spinal cord showed the classical lesions of tabes, but **the heart and aorta were absolutely healthy.** Cardiac neuralgia in tabes is, therefore, an accomplished fact.

Histological researches have also confirmed this opinion; peripheral neuritis explains the neuralgic pains, the real cause of which has till now passed unnoticed. Oppenheim found degeneration and atrophy of the pneumogastric nerve in a case of tabes, accompanied by gastric crises and by angina pectoris, with pain radiating into the left arm. Grocco and Fusari found changes in the cardiac plexus, in the vagus, and the laryngeal nerves, and in the abdominal sympathetic and the cœliac plexus, in a patient who had had attacks of angina pectoris, with gastric and laryngeal crises. Pitres and Vaillard admit, as regards the case in question, that the visceralgic crises of tabetic patients, including those of angina pectoris, may be due to neuritis of the corresponding visceral nerves.

We may, therefore, say that tabetic patients may be affected by cardio-aortic neuralgia, which reproduces the syndrome of angina pectoris, just as they are affected by visceralgic crises of the stomach (gastric crises), kidneys (renal crises), urethra (urethral crises), bladder (vesical crises), testis (testicular crises), and rectum (ano-rectal crises). All these crises form part of the painful symptom-complex of tabes, and are made up of acute lightning pains in the organ and limbs.

To sum up: A tabetic patient may show, on the one hand, aortic lesions with or without angina pectoris, and, on the other hand, angina pectoris without aortic lesions. In the former case the aorta is atheromatous and dilated, while the aortic orifice is damaged, and, a remarkable fact, these aortic lesions, which in other cases, as, for example, in syphilis, are so painful, are usually slight and painless in tabes. Hence an apparent paradox results—viz., that the tabetic patients who have terrible attacks of angina pectoris are generally those who have not aortic lesions. They are affected by neuralgia or neuritis of the cardiac plexus.

The **treatment** of angina pectoris in tabes must now claim our attention. Mercury and iodide should never be omitted if the patient is syphilitic, for it is not impossible that he may have suprasigmoid aortitis. Further, there is a series of therapeutic measures which I recommend as regards local treatment—the application of the cauterizing point to the thoracic region, or, better still, an issue which is allowed to suppurate; this issue, which is an old therapeutic measure, and has been too much neglected in our time, gives excellent results. I also recommend an ice-bag day and night over the heart. To avoid the weight of the ice-bag we should suspend it from a hook, and to avoid erythema we must protect the skin of the precordial region with lint or oiled silk. Injections of morphin or heroin may be given in doses of  $\frac{1}{8}$  grain; aspirin, and antipyrin, are of much service.



With these various therapeutic measures we may also employ suspension, as in my case, for it has a beneficial effect upon the pains of tabes; prudence and moderation must, however, be observed, for a crisis of angina pectoris arising during suspension would have dire results.

## X. ANGINA PECTORIS.

**Description.**—The cardiac neuralgia which is called **angina pectoris** (Heberden) is a painful affection that supervenes in the forms of attacks. The patient is **suddenly** seized with an attack, which may come on without appreciable cause, or as the result of emotion, fatigue, slight exercise, or a somewhat hearty meal. In the region of his heart, along the left border of the sternum, he feels a stabbing pain, which radiates to the neck, the epigastrium, or the thorax. As a rule, the pain affects the left arm, the hand, and the last two fingers (area of the ulnar nerve), while the skin of the hand becomes pale. When the attack is violent, the pain is accompanied by an appalling sensation of constriction and dyspnœa, and the patient, who is bathed in a cold sweat, feels as though gripped in a vice, or squeezed by an enormous weight. Suffocation and syncope appear imminent, and the unfortunate sufferer, who can neither speak nor move, remains quite conscious, and experiences the inexplicable feeling of impending death. “It is as though there were a momentary cessation of life” (Elsner). The attack lasts some seconds or some minutes, and then disappears, leaving the following traces: numbness of the left arm, swelling of the testis (Laënnec), urgent desire to urinate, eructation of gas, vomiting, and marked lassitude.

The attack, however, is not always of the classical form just described. The pain which starts from the heart, radiates in the most diverse manner; it may not be limited to the left side, but may invade the arm and hand on both sides, spread up the neck as far as the articulation of the jaw, descend towards the epigastrium, and surround the trunk like a girdle, or pass down into the groin as far as the testis. The pain may follow an inverse course, starting in the hand and rapidly travelling up towards the chest like an aura. This fact led Trousseau to suppose that **angina pectoris** is sometimes a manifestation of epilepsy—that is, an **epileptiform neuralgia**.

During the attack, and in spite of the imminence of suffocation, auscultation reveals no abnormal sounds; the heart beats are normal or slow; they may be irregular, if the angina is associated with cardio-aortic lesions; in some cases they are hurried.

In some patients the symptoms are of a different character, and the attack of pain is sometimes replaced by attacks of dyspnœa, with or without palpitation. The dyspnœa is at times so marked that the painful element

is of secondary moment. This agonizing and paroxysmal dyspnœa somewhat recalls the severe dyspnœa of uræmia.

In many cases angina pectoris is not acute; the patient constantly experiences for weeks or months continuous or intermittent pain in the left arm, with a feeling of discomfort and oppression in the precordial region. This chronic condition is sometimes interrupted by acute attacks. Loss of appetite, salivation, constipation, and ballooning of the belly are common during the attacks.

**Ætiology.**—Angina pectoris and its varieties may be the result of many causes. In some cases it is a neuralgia which is independent of any appreciable lesions, though sometimes complicated by neuritis; it is most often associated with lesions of the **aorta** and **coronary arteries**. In order to simplify the question of pathogenesis, I would thus state the case:

In the first variety angina pectoris is the result of some lesion of the aorta and coronary arteries. The **coronaritis** usually affects both coronary arteries. The lesions of atheroma, endarteritis, and arterio-sclerosis of the coronary arteries produce stenosis, and may end in obliteration. **Ischemia** of the heart, symptoms of angina pectoris, and sudden death may be the result. All cases of aortitis, whether acute or chronic, infective, syphilitic, gouty, or atheromatous, favour this variety.

Angina pectoris resulting from aortic lesions without affection of the coronary arteries belongs to the second variety. Under Syphilitic Aortitis I have given the report of two patients who died from angina pectoris; the autopsy demonstrated the presence of aortic lesions, and the complete permeability of the coronary arteries. This proves that ischemia of the heart, consecutive to coronaritis obliterans, is not indispensable to a fatal attack of angina pectoris, although some authors hold a different opinion.

Cases of angina pectoris without aortic lesions and coronaritis are placed in the third variety, and comprise neuralgia and neuritis of the cardiac plexus, which are similar to the neuralgias of the facial, sciatic, and other nerves.

Tumours of the mediastinum which are close to the cardiac plexus may excite the neuralgia or the neuritis which provokes angina pectoris. In these cases inflammation or **neuritis** of the cardiac plexus, with or without participation of the phrenic nerve, has been found at the autopsy (Lancereaux, Peter).

In the form of neuralgia angina pectoris is associated with epilepsy, hysteria, cerebro-cardiac neuropathy (Krishaber), diabetes (Vergely), and Brightism (personal case); it is also caused by the abuse of tobacco (Beau), tea, or coffee. The gouty diathesis may show itself by angina pectoris, just as it does by migraine or by attacks of asthma. Rheumatism may also lead to cardiac neuralgia; angina pectoris sometimes appears during an attack



of acute rheumatism, while at other times it is **essentially** rheumatic, though the patient has not yet suffered from articular rheumatism (Viguier).

In the preceding chapter I have quoted, among others, Vulpian's remarkable case of a tabetic who suffered from angina pectoris: post mortem Vulpian found no trace of any aortic lesion; the case was one of cardiac visceralgia. Tabetic patients have crises of cardiac neuralgia, just as they have neuralgic crises, affecting the stomach, rectum, urethra, etc.

According to some authors, every **true** angina pectoris is due to constriction of the coronary arteries and consecutive **ischemia of the heart**, while the other varieties are **false** angina pectoris.

These authors, therefore, would make two great classes of angina pectoris. In the one they would place angina pectoris with lesions of the coronary arteries—that is, the true and fatal form; in the other class they would place cases of angina pectoris, neuralgia, or neuritis without lesions of the coronary arteries—that is, the false and not fatal form.

This division is, however, in my opinion, too absolute. I am ready to admit that stenosis and lesions of the coronary arteries are the chief cause of the disease in many cases, but I cannot subscribe to a classification which would divide angina pectoris into two well-marked categories, the one comprising true angina, which is usually grave and too often fatal, while the other concerns false angina, which is looked upon as an almost negligible quantity. I agree that a patient who has coronaritis obliterans may be harder hit than one who has not, but this is no reason for saying that the latter is a false angina; indeed, so little is this form a false angina that the attacks may be acute, painful, and fatal. At the Saint-Antoine and Necker Hospitals I witnessed two deaths from angina pectoris; the autopsy was made with every possible care. Both patients had chronic aortitis; in one of them the aortitis was syphilitic and accompanied by small multiple aneurysms, but in both the coronary arteries were quite free. We cannot, then, speak of true and false angina, but we must admit cases which are more or less formidable, but which may all prove fatal. As regards prognosis, how can we affirm that the coronary arteries are or are not affected during life? I think, therefore, practically speaking, that every patient who suffers from angina pectoris may die of it.

Finally, whatever is the primary cause of angina pectoris, whether it be ischemia of the heart or not, whether the initial lesion affects the aorta alone, or the aorta and the coronary arteries, or the coronary arteries without the aorta, the cardiac plexus, with its afferent and efferent nerves, is the starting-point of the symptoms. The whole cardiac plexus, including the branches of the vagus, the great sympathetic and the ganglia, may take part. The richness of this plexus, the importance of its nerves, and its numerous anastomoses, explain both the gravity and the diversity of the symptoms.

The radiations of pain, or *synæsthesiæ*, modify the symptomatic expression of the attack, which, according to circumstances, shows itself by pain, dyspnoea, or syncope, or by the whole symptom-complex. The cardiac plexus, by its pneumogastric or its sympathetic branches, holds the symptoms of angina, asphyxia, precordial pain and syncope under its sway. The reaction on the brachial plexus, and especially on the ulnar nerve, explains the pain in the hand and the little finger. When the attack is more generalized, the diversity of the pains is in relation with the nerves affected by the neuralgia, and in this way we can explain the sensations of strangulation and œsophagism (fibres of the pneumogastric), the thoracic constriction and pain at the insertion of the diaphragm (phrenic nerve), the pallor, sweating, prostration, and chilling of the extremities (radiation to the great sympathetic) (Peter).

**Course.**—The first attacks of angina pectoris are generally slight or transient, and do not as yet show the severity which they acquire later. In some cases, however, the first attack may be fatal. I was present at the death of one of the most distinguished physicians at the Saint-Louis Hospital, who died in his first attack. Some individuals (though this is rare) remain free after a single attack; as a rule, the crises, which appear at first at long intervals, occur at closer intervals, and finally come on every week or every day. Sometimes the patient has some respiratory distress, precordial constriction, numbness of the arm, or aching pain in the region of the aorta; this condition may last for weeks and months, quite apart from the attacks. The attacks are frequently periodic, and return at a fixed hour, whether the neuralgia is or is not symptomatic of an organic lesion; the periodicity is not, then, a distinctive sign in favour of pure neuralgia.

The chances of cure are greater, and the prognosis is more favourable when angina pectoris is not associated with lesions of the coronary arteries or of the aorta; the prognosis is certainly less grave when the disease is only a simple neuritis (hysteria, smoker's heart). Angina pectoris, however, is always formidable, for sudden death and fatal syncope are only too frequent.

**Diagnosis.**—The diagnosis of angina pectoris is sometimes difficult, because of the variations in the symptoms. Pleurodynia of the precordial region is characterized by superficial pains, which are limited to the muscles, and do not radiate beyond the invaded parts. Cervico-brachial and thoracic neuralgias are limited to the course of the diseased nerves; they show painful fixed points, and do not arise with the suddenness of angina pectoris. It must not be forgotten that angina pectoris is often only an epiphenomenon or an advance sign in lesions of the aorta and of the mediastinum; it is frequently the first warning of aneurysm of the aorta, and in such a case it is imperative to diagnose the cause. At other times, instead of appearing in the complete form, the angina is only indicated by



one of its symptoms, such as numbness in the left arm, pain limited to the little finger, or a sensation of constriction and agony in the region of the heart. These defaced forms should be recognized.

Neuralgia of the **phrenic nerve**, whether primary or consecutive to diaphragmatic pleurisy or to acute pericarditis, shows itself by a complex of symptoms (situation of the pain, dyspnœa, hiccough), which permits the distinction from cardiac neuralgia; but neuralgia of the phrenic nerve is sometimes associated directly (neuritis) or indirectly (reflex radiation) with neuralgia of the cardiac plexus. There is in these cases an "association of pains which depends on the innervation of the circulation by the cardiac plexus and on that of the respiration by the phrenic nerve" (Peter).

**Hysterical** angina pectoris merits special mention, and the diagnosis must rest on the following considerations: The attack usually occurs at night; hystero-genic zones are found in the precordial and sternal regions, and the aura begins with hyperæsthesia at these spots. Hysterical angina pectoris ends in tears, and is provoked by moral causes, while angina, which is not hysterical, is brought on by physical causes (fatigue, walking, strain).

**Treatment.**—The first indication is to eradicate every cause (emotion, fatigue, hearty meals, and excess of tobacco) that is capable of producing an attack; the second indication is to treat the disease (gout, rheumatism, or syphilis) of which the angina is sometimes only a manifestation.

During the attack morphia and ice appear to me to be the best treatment. One or more subcutaneous injections of morphia are given, and ice-bags are applied to the precordial region. We may add inhalations of chloroform or of ether to these methods. Local blood-letting and the application of leeches or cupping to the painful region often give good results.

Aspirin renders much service. Thirty grains of aspirin, divided into two cachets, are given every two hours. I must also mention nitrite of amyl in capsules; inhalation of a few drops at times brings about immediate improvement.

Blisters, issues, or the cautery may be applied over the painful region between the attacks. In a case in which painful symptoms had persisted for a long while, I ordered ice-bags, which were changed day and night, to the precordial region for several weeks; in this case the skin must be carefully protected by means of lint or flannel to avoid erythema. In the case to which I have alluded the patient found such relief from the permanent application of ice that she went about without removing the bags.

The question of angina pectoris, when associated with syphilitic lesions of the aorta, has been dealt with in one of the preceding sections, so I shall not refer to it again; but the physician should remember the possibility, and give injections of mercury if necessary.

Every individual who is subject to angina pectoris, whatever its cause, must give up tobacco, tea, alcohol, and coffee; he must avoid emotion, hearty meals, and violent exercise. He will also experience benefit from treatment which lowers the arterial tension (Huchard).

## XI. ARTERIAL TENSION—HYPERTENSION— HYPOTENSION.

The study of arterial tension has become the order of the day, and clinicians attach special importance to the variations which the tension undergoes in different morbid conditions.

Professor Potain was one of the first to invent a practical method of estimating the arterial tension; his researches have given interesting results, but they have been chiefly confined to the pressure in the peripheral vessels, such as the radial artery. Mosso, Bouloumié, and others, have recently drawn attention to the blood-pressure in the smallest vessels, and have described the changes in the "arterio-capillary tension."

**Technique.**—I shall describe here Potain and Bouloumié's instruments for measuring the arterial or arterio-capillary pressure, because they are the most practical. The first apparatus, called the "sphygmomanometer," is composed of a dial manometer and an indiarubber tube, which ends in a small ampulla filled with air. The ampulla is applied over the radial artery so as to obliterate the pulse; the number indicated by the needle expresses the arterial pressure in centimetres of mercury.

The second apparatus has, in addition, a rubber finger-stall, with which the end of the finger is compressed till the anæmia is complete. The needle indicates the pressure produced by the reappearance of the circulation in the pulp of the finger.

It is well to take the blood-pressure apart from digestion, and in the recumbent position.

The mean reading in a normal person varies from 14 to 18 for the radial pressure, and from 11 to 13 for the arterio-capillary pressure. The tension is usually higher at night than in the morning; it is affected by food, emotion, and temperature. It is not as high in women as in men, and increases in proportion to the age.

As a rule, though it is not an absolute one, the heart-beats are slowed in proportion to the increase of the blood-pressure.

Alimentary products and certain drugs (adrenalin, ergotin) modify the blood-pressure in a more or less lasting manner; the most interesting variations, however, occur in pathological conditions.

Most diseases influence the arterial tension; some cause a rise, others a



fall. The limit of hypertension and of hypotension is somewhat artificial ; we speak of hypertension when the reading exceeds 18 for the radial and 14 for the digital pressure ; of hypotension when the figures are below 14 and 10.

### Hypertension.

Transient hypertension may occur in many morbid states : eclampsia and cerebral hæmorrhage, lead-poisoning, tobacco-poisoning, acute vascular affections, aortitis, pericarditis.

Lasting hypertension depends upon two factors—Bright's disease and arterio-sclerosis. Potain in particular has called attention to the arterial hypertension in Bright's disease ; it may be as high as 24, 26, or even 30 centimetres, and it is so common as to be an indication of the highest importance.

Atheroma and arterio-sclerosis are most often accompanied by notable increase of blood-pressure, and it is possible that in some cases the hypertension precedes the usual manifestations of arterial atheroma (Huchard).

Apart from these two factors, diabetes and certain forms of dyspepsia may perhaps play some part. Bosc and Vedel have admitted in some subjects a kind of hereditary predisposition to hypertension, and hydropathic hypertension has been described, though its nature is somewhat indefinite ; the tendency is, however, to attribute it to hypersecretion of the suprarenal glands and of the pituitary body.

**Symptoms.**—The first symptom of hypertension is a rise in the arterial tension to 20 centimetres of mercury or more, and in the capillary tension to 15 or 18.

The radial pulse is hard ; the temporal artery is tortuous ; the heart-beats are violent, and the second sound in the aortic area is ringing ; a white line made with the nail on the abdominal wall lasts only one or two seconds.

In addition to these physical signs, Vaquez has grouped certain functional symptoms into a syndrome : convulsions, which appear in eclampsia as well as in uræmia and saturnism, and resemble general or partial epilepsy ; amaurosis without change in the fundus oculi, but with headache and increased ocular tension ; homonymous hemianopsia ; progressive changes in vision, which follow the variations in the arterial tension ; transient aphonia, which appears and disappears suddenly ; vertigo ; pulsation in the arteries of the neck ; hot flushes ; sweating ; pallor of the face ; heaviness in the limbs. Some writers would set down to hypertension the dead fingers, and the cryæsthesia I have noted in Brightism, the repeated attacks of pulmonary congestion, the crises of angina pectoris, and certain troubles of a neurasthenic nature, mental depression, transient attacks of mania, and obsession. Slight jaundice may exist in persons whose tension is high. Sudden death is sometimes seen, and lumbar puncture or post-mortem examination may show scattered hæmorrhages in the meninges. Hypertension, therefore, has

a varied symptomatology, which has been gradually enriched by signs formerly attributed to the diseases which it accompanies. The pathogenesis of hypertension has been much discussed. It is often due to peripheral vasoconstriction, which may follow true lesions or spasmodic troubles, or may be the result of toxic or mechanical phenomena. An undeniable part is played by the sympathetic nerves, the lesions of arterio-sclerosis, and poisoning due to lead or tobacco.

In arterio-sclerosis we may ask whether hypertension, as Huchard says, is the cause, and not rather the consequence, of vascular lesions; in renal impermeability we do not know whether it results from retention of toxic products or simply of chloride of soda, or from increase in the mass of the blood.

The theory of Vaquez deserves special mention. He has found changes in the suprarenal glands, and has set down hypertension as a manifestation of the vasotonic hypersecretion of these glands. Sézary has shown, however, that, in chronic nephritis, the predominant factor is not the suprarenal over-activity, but, especially, the obstacle to the circulation of the blood, caused by the renal sclerosis.

The prognosis is serious. In addition to the immediate complications already noted, we must not forget that the overwork to which the arteries are subjected may lead to permanent hypertrophy of the muscular coat, and even to atheroma (Huchard).

**Treatment.**—Prophylaxis consists in avoiding tobacco, spiced foods, meat diet, and alcohol. Curative treatment comprises lacto-vegetarian diet and rigid hygiene; we must give drugs which lower the tension and dilate the vessels—*e.g.*, nitrites and iodides, purgatives, diuretics; in severe cases, bleeding is useful.

Dechlorination and carbonic-acid baths are often of service.

### Hypotension.

Hypotension may be transient or lasting. In the former case it appears in most infectious maladies, in many forms of poisoning, and in acute cardiovascular affections; in the latter case it is seen in Basedow's disease, in myocarditis and chronic diseases of the pericardium and the heart, and in chronic tuberculosis; finally, it is one of the classical symptoms in Addison's disease. The syndrome of arterial hypotension is not so complete as that of hypertension. The manometer reading may be as low as 8 or 10 centimetres. The pulse is small and, as a rule, rapid; the line made by the nail remains white for a long while. We may also find sluggish peripheral circulation, cyanosis, coldness of the extremities, oedema, passive congestion of the liver, and oliguria. This syndrome is typical in Addison's disease, and may be called the syndrome of hypotension or asystole.



The pathogenesis is readily summed up : when vascular, it is due to some obstacle at the periphery ; when toxic, it may be put down to the action of the toxines upon the vessels and the vasomotor system. The part played by the tubercle bacillus and its products in these cases is well known. The pathogenesis of hypotension in Addison's disease is of great interest, because it is proof of the insufficiency of the suprarenal secretion. It is possible that similar insufficiency exists in acute suprarenalitis, and in infectious disorders and that we must give to it its share in the production of the hypotension which is often found.

Rénon and Azam have recently put forward a similar theory as to insufficiency of the pituitary secretion in the infections.

The treatment consists in the exhibition of cardio-vascular tonics, when the low tension is dependent on some cardiac lesion.

In Addison's disease good results have followed the use of suprarenal extract and of adrenalin. Opothérapie has been employed in some infectious diseases ; capsular extract has appeared to be of some slight use. Rénon and Debille have given their patients pituitary extract.

## PART III

### DISEASES OF THE DIGESTIVE SYSTEM

#### CHAPTER I

#### DISEASES OF THE MOUTH

##### I. ERYTHEMATOUS STOMATITIS.

**Description.** — Inflammation of the mouth is called stomatitis (στόμα, mouth). Erythematous stomatitis (buccal catarrh) is the most frequent variety; it is met with at all ages. Its usual **causes** are dentition, the eruption of the wisdom teeth, dental caries, artificial teeth, abuse of tobacco and highly-spiced foods, drinking of hot liquids, accumulation of tartar, and oral sepsis. The various micro-organisms in the mouth play a large part in its causation. Erythematous stomatitis is also associated with digestive troubles, and often accompanies catarrh of the stomach. Patients with Bright's disease sometimes have stomatitis, which has a tendency to ulcerate. It is not yet decided whether this is due to the elimination of toxic principles or to the association of the microbes in the mouth.

Stomatitis is ushered in by pain, which is aggravated by heat, cold, contact with food, and movements of mastication. The mouth is hot, dry, and clammy; the breath is offensive, and the sense of taste blunted.

The inflammation of the mucous membrane is often limited to a specified region, such as the cheeks, the tongue, the roof of the palate, or the gums. The inflamed mucous membrane, which is red, dry, and shining, is covered, according to the region, with papillæ, or swollen glands. On the edges of the gums we find opalescent epithelial patches. Where the mucous membrane is lined with loose connective tissue, as on the cheeks or on the lips, swelling and œdema are seen. In places, desquamation of the epithelium causes erosions and superficial ulcers, which are very painful. The reaction in the submaxillary glands is but slight.

The course and duration of the disease depend on the cause. Stomatitis may be kept up or revived by dental caries, slow eruption of the wisdom teeth, or accumulation of tartar. Chronic gingivitis is frequent in diabetic patients; the teeth often fall out in consequence. Erythematous stomatitis



readily yields to mouth-washes of borate of soda, or to pastilles and lotions of chlorate of potash. Frequent cleansing of the mouth with antiseptic solutions, such as thymol (1 in 100), or chloral hydrate (1 in 100), may be prescribed. The cause of the stomatitis must be carefully looked for.

## II. MERCURIAL STOMATITIS.

**Ætiology.**—Whether mercury is introduced into the system by the skin, the respiratory organs (mercurial vapours), the mucous membranes, or the digestive system, the drug is partly eliminated by the salivary glands, and its action on the mucous membrane produces an inflammation called “mercurial stomatitis.”

Gilders, fitters, looking-glass makers, miners who live in the midst of metallic vapours, and hatters who handle nitrate of mercury, are especially liable to poisoning. Mercurial preparations given in syphilis by inunction, by subcutaneous injections, or by the mouth, may cause stomatitis, which varies in severity according to the susceptibility of the individual. One patient can tolerate repeated inunctions of mercury, whilst another contracts stomatitis after a single inunction of mercurial ointment. Mercurial stomatitis is almost unknown when hypodermic injections of the biniodide are used. I have during the last few years given more than ten thousand injections of biniodide of mercury, and I have not noticed the least accident.

**Pathogenesis.**—The nature of mercurial stomatitis has been much discussed. It was formerly regarded as purely toxic, but it is now considered a septic condition (Galippe). It seems to me difficult not to admit that the condition is most often infective, the mercury leading to more rapid multiplication of the microbes in the mucous membrane when previous gingivitis and dental changes have occurred.

**Description.**—Mercurial stomatitis may be **acute** or **chronic**. The inflammation usually starts behind the last molar tooth of the side upon which the patient sleeps (Ricord). The symptoms are most marked in the lower jaw and in the neighbourhood of decayed teeth. From the first the patient complains of a metallic taste in the mouth, of irritation, and of heat and pain at the angle of the jaws. The breath rapidly becomes foetid; mastication is painful; the gums are soft, swollen, and bleed easily. The mouth, which is at first dry, is soon filled with saliva. These cases have been called the **stomatites d'alarme** (Fournier).

When the above symptoms alone are present, mercurial stomatitis yields to treatment in a few days; we see, however, severe forms, which were more common when it was customary to provoke and keep up salivation in the treatment of syphilis. In these grave forms (Fournier) the inflammation invades the alveolar periosteum. The teeth are laid bare and

loosened. The inside of the cheeks becomes swollen, and shows the mark of the teeth, while the tongue becomes much enlarged. The inflamed parts are reddish, and show ulcers which are covered by a greyish pultaceous fur. The saliva flows from the mouth day and night in such quantity that it may amount to 6 or 8 pints in twenty-four hours. The saliva contains mercury in small quantities, and bleaches gold. Deglutition becomes difficult, the breath is horribly foetid, the fever is high, diarrhœa appears, and the patient sinks into profound anæmia.

We may see still more terrible forms, in which the œdema extends rapidly to the pharynx and the sublaryngeal tissues, and reaches the submaxillary region. The general induration and the enormous swelling of the tongue make deglutition impossible, and threaten the patient with asphyxia. Salivation, fever, and insomnia wear out the sufferer, and at times bring about a fatal termination. When mercurial stomatitis reaches the **chronic stage**, the acute symptoms disappear, but the swelling of the mucous membrane and the ulcers persist. The teeth gradually fall out, and the jaws may become necrosed. In some cases it is chronic from the first. The acute symptoms are absent, the ptyalism is insignificant, and the ulceration of the gums is not marked; but the alveolar periosteum is attacked, and the teeth are laid bare, and finally fall out. This exceptional form has been observed amongst the miners of Almaden (Roussel).

**Pathological Anatomy.**—The lesions of the mucous membrane, formerly considered as due to common stomatitis, are said to have a certain specific character. In several patients Delbanco has noted hypertrophy of the sebaceous glands in the mouth, which form yellowish grains about the size of a pin's head. Histological examination has proved that they are enlarged acinous glands, identical with the sebaceous glands of the skin.

**Diagnosis.**—The diagnosis rests on the ætiology, which permits us to distinguish the mercurial from the other forms of stomatitis with severe ptyalism—*e.g.*, uræmic stomatitis.

**Treatment.**—The treatment may be summed up as follows: Limit the inflammation by means of topical emollients, give chlorate of potash, and cauterize the ulcers with nitrate of silver, tincture of iodine, perchloride of iron, or hydrochloric acid. Chlorate of potash has the advantage of being in part eliminated by the salivary glands, and thus forms a kind of permanent mouth-wash. It may even be given with the mercury as a preventative. Mercurial preparations should never be prescribed without careful examination of the teeth and gums. In severe cases, where the nutrition is threatened by the difficulty in swallowing, liquids can be given by the œsophageal tube. Some authors have recommended iodide of potash, which seems to facilitate the elimination of mercury. Antiseptic solutions may be of some use when the infectious element is predominant.



### III. ULCERO-MEMBRANOUS STOMATITIS.

**History.**—The disease which to-day is described under the name of **ulcero-membranous stomatitis** has for a long time been confounded with other affections of the mouth under the misleading names of stomacace (*στόμα*, mouth; *κακός*, bad), aquatic chancre, scurvy of the mouth, membranous stomatitis, etc. When the Vendean Legion was in garrison at Tours in 1818, Bretonneau described, under the name of “**diphthérie buccale**,” certain membranous ulcerations of the mouth among the men, and he considered himself the more certain because some of the men had at the same time contracted pharyngeal and laryngeal diphtheria. Bretonneau’s doctrine was opposed. In 1853 Rilliet and Barthez rejected the theory of **buccal diphtheria**, which they replaced by the name of “**ulcero-membranous stomatitis**”—a disease which has nothing in common with diphtheria; and in 1859 Bergeron, observing an epidemic of this disease among the soldiers in the Saint-Martin Hospital, gave to it the name of **specific ulcerous stomatitis**. This difference of opinion does not condemn the doctrine of Bretonneau. It seems to prove that the patients observed by Bretonneau were suffering from a twofold epidemic—ulcero-membranous stomatitis and diphtheria. Bretonneau did not understand the former, but he was correct in creating “**diphthérie buccale**,” and it would be a grave error to reject this manifestation of diphtheria, which has been confirmed by recent bacteriological researches.

**Description.**—Ultero-membranous stomatitis commences like the other forms of stomatitis. After three or four days of malaise or of fever, sometimes even without prodromata, the symptoms of inflammation appear—a feeling of burning and dryness, redness and swelling of the mucous membrane. The ulcers, however, are the specific stamp of the disease.

They are usually limited to the **left side of the mouth**. The first ulcer generally affects the mucous membrane of the mouth on a level with the last molar teeth. The ulcers are found, in order of frequency, on the gums, cheeks, and lips, especially the upper lip; more rarely on the tongue, roof of the palate, and tonsils. The ulcers on the gums are often vertical, and may occupy the whole edge of the gum; those on the cheek and lips are more commonly oval. At first the lesion is a prominent violet-coloured disk, which soon becomes soft. The surface is pulpy, yellowish, or greyish, and is really a slough composed of the elements of the mucous membrane. The slough is slightly adherent; it becomes detached, and leaves in its place an ulcer, which bleeds easily, and has a greyish base, with irregular and ragged edges. The ulceration spreads by the progressive elimination of the foetid detritus which lines its cavity, and large ulcers sometimes result from the confluence of smaller ones. The surrounding parts are more or less oedematous. During the second week the process of repair commences, the bottom of the ulcer becomes clean, the greyish membranes are detached, leaving a reddish granulating surface, and cicatrization follows.

To sum up, ulcero-membranous stomatitis is a specific, curable, and more or less superficial gangrene of the mucous membrane of the mouth. The ulcers, however, do not in themselves comprise the entire lesion.

Softening and swelling of the mucous membrane of the gums, which becomes greyish and bleeding, has also been observed. Some authors have, confounded this stomatitis with scurvy. It is also common to meet with the symptoms and the lesions of sore throat; ulcers may develop at the back of the throat, producing an ulcero-membranous tonsillitis. It is evident that the disease is not simply a stomatitis; it is an ulcero-gangrenous inflammation of the whole bucco-pharyngeal cavity.

In the ulcerative stage the local symptoms become acute: pain severe, mastication impossible, deglutition difficult, breath horribly foetid, saliva profuse and streaked with blood, yet much less profuse than in mercurial stomatitis. The submaxillary and retromaxillary glands are often swollen, and remain so until cicatrization of the ulcers occurs. The inflamed glands do not suppurate, but in scrofulous subjects they may persist indefinitely. The general symptoms of fever, and gastro-intestinal and nervous troubles are more marked in children than in adults.

Its course is not influenced by concurrent diseases (Bergeron). Its duration is variable. The patient recovers in a week or ten days with proper treatment; in the opposite event the disease may be prolonged for weeks and months. Complete recovery is the rule. In grave cases, however, the alveolar edge of the maxilla becomes necrosed; the teeth are laid bare, and fall out without showing any trace of caries.

**Diagnosis—Ætiology.**—The situation, course, and characters of the ulcero-membranous patches make it impossible to confound this disease with other forms of stomatitis. In “*diphthérie buccale*” pain and salivation are practically absent. False membranes are found on the gums, the lower lip, and the labial commissures. These membranes are at first whitish, and then grow darker; when they are removed, the mucous membrane is found to be intact, or only exulcerated, which is in striking contrast with the ulceration just described. Clinical examination is, however, generally insufficient, and bacteriological investigation is necessary; it may reveal unsuspected diphtheria.

**Syphilitic** mucous patches and **tubercular** ulcerations of the mouth have such clearly defined characteristics that a mistake in diagnosis is impossible.

Ulcero-membranous stomatitis often assumes an **epidemic** character. It is contagious, but does not appear to be inoculable; there are, however, positive cases of inoculation. It attacks children of from four to ten years of age, and especially adults, when it finds conditions favourable for development, such as those presented by bad hygiene, insufficient food, etc. This explains the epidemics among soldiers, sailors, or in hospitals and in prisons. Bacteriological researches concerning this variety of stomatitis and the tonsillitis which often accompanies it, have always revealed the



presence of two micro-organisms—a spirillum and a fusiform bacillus (Vincent). Up to the present it has not been possible either to cultivate this bacillus or to reproduce the lesion experimentally. For the time being it is impossible to assign a specific rôle to them. “Their constant presence, however, in almost pure culture in the exudation is sufficient presumption to regard them as the causes of the disease.” Prophylaxis demands the removal of persons from all causes of contagion and from the scene of the epidemic. For an adult, chlorate of potash should be administered daily, and if necessary the ulcers should be touched with nitrate of silver, or, better still, with small tampons of absorbent wool, moistened with a solution of sublimate (1 in 1,000).

#### IV. THRUSH

Thrush is also called white mouth or creamy stomatitis. At first sight it resembles a whitish and creamy layer, which lines the mucous membrane of the mouth at different points. This layer is formed by the union of disks, which look like curds of milk, and are composed of a seed-bed of projecting whitish grains.

The description, however, would be very incomplete if it were limited to the oral variety. The mouth is doubtless its seat of election; nevertheless, recent researches have proved how common it is in other regions. Although it is here described among the diseases of the mouth, I must mention the other manifestations of the disease. I shall therefore trace the life-history of thrush, and shall then mention the modifications seen according to the region affected.

**Life - History — Pathological Anatomy.**—Under the microscope a particle of thrush shows two distinct elements: the one is the stroma, in which you find neither pus nor fibrin, formed by pavement cells of every age, mostly degenerated and granular; the other, the specific element, is formed of interwoven filaments and of rounded corpuscles. These filaments were formerly considered as a mycelium, and the rounded corpuscles were looked upon as the spores. At that period thrush was described as a vegetable, belonging to the mushroom family, a cryptogram of the genus *Oidium aphthophyta* (Gruby), *Oidium albicans* (Robin), *Oidium syringospora* (Quinquaud). The researches of Audry have shown thrush to be a yeast. He classes thrush amongst the saccharomycetes, describes its yeast-like form, gives the results obtained from cultures on solid (potatoes) and liquid media (Koch's broths), and proposes to call the parasite *Saccharomyces albicans*. More recent researches tend to show that thrush is not a true saccharomyces. A case of pharyngeal thrush, caused by a yeast comparable to the industrial yeasts, has been published, and proves once more that “a naturally inoffensive

micro-organism can on a prepared medium become pathological, and give rise to a characteristic disease." Cultures of thrush on sterile carrot give snow-white colonies in forty-eight hours. Microscopic examination reveals the *saccharomyces* in the form of rounded cells, in groups or chains, covered by an envelope which does not stain. In broth the microphyte, after a few days, forms long filaments and oval cells; on a solid medium, however, the culture no longer shows filaments, but only rounded corpuscles. True spores only appear in a mineral fluid containing sugar.

As an accessory element we often find an alga in the shape of little rods without any trace of articulation, known as the *leptothrix*; it has nothing to do with thrush, and exists in many morbid conditions of the mouth.

The fungus, when inoculated into the veins of an animal, causes mycotic lesions, which are very interesting (Klemperer, Linossier and Roux, Grasset). Experimental oïdian infection chiefly attacks the kidneys (Roger, Noisette), which are riddled with little white granulations (Roger). The serum of animals, vaccinated against *Saccharomyces albicans*, causes agglutination of the parasite, which covers itself with a thick cuticle (Roger), behaving like Eberth's bacillus, with this difference, that in the case of the bacillus the agglutinative reaction is one of infection (Widal).

We may now consider the distribution and the behaviour of thrush.

Thrush is usually found in the mouth. Its appearance is announced by a peculiar state of the mucous membrane, which I shall describe in a moment. The relations between thrush and the mucous membrane are variable, according as the thrush is epithelial or dermic (Parrot). In **epithelial** thrush the filaments go down into the epithelial layer, the pavement cells are separated by a pile of cells, and the thrush rises in the shape of tufts. In **dermic** thrush the filaments go down as far as the derma of the mucous membrane, and the papillæ show nuclear proliferation. The epithelial variety of thrush is only found on the *velum palati* and on the roof of the palate.

In the **pharynx** thrush may be primary, and precede that in the mouth, but this is very rare indeed.

Thrush is very common in the *œsophagus*, where it forms yellowish or brownish patches; the elements go down deeply, through the *tunica mucosa*, as far as the muscular layer. The connective tissue shows abundant proliferation at the affected points.

In the **stomach** the thrush, covered by a thick layer of mucus, appears in the form of greyish papillæ. It adheres firmly to the walls, which it penetrates deeply. The superficial portions of the gastric glands are destroyed, but the *culs-de-sac* are dilated, and resemble gourds full of spores (Parrot). The vessels show thrombosis, due perhaps to the penetration of the filaments.



The **cæcum**, with its acid reaction, offers conditions favourable to the development of thrush.

The **lower vocal cords** offer a good soil for the propagation of thrush, because they are provided with pavement epithelium, whilst thrush does not develop on mucous membranes with columnar epithelium, no doubt because the cilia prevent the parasite from gaining a hold. Parrot has found nodules of thrush in the infundibula of the lungs.

It is not exceptional to find thrush on the vulva, vagina, anus, or the prepuce, and on the nipples of wet-nurses.

Thrush is also a pyogenic agent. Grasset has found it in an abscess of the gums, and Charrin in a submaxillary abscess. In man, as in animals, it may produce a general infection in the lungs, the spleen, the kidneys, and the brain (Schmorl, Zenken, Ribbert, Pineau).

**Ætiology—Pathogenesis.**—Thrush often appears within the first two weeks of birth. It may develop as a purely local affection, favoured by dirty bottles and acidity of the milk; in this form it presents no grave features. These rare cases excepted, thrush is a secondary affection, indicative of general ill-health. Thrush is more general at the two extremes of life, and any cause of organic decay favours its development. In the new-born it is associated with digestive troubles, enteritis (Seux), bad hygiene, defective feeding, and in the state of malnutrition which Parrot has called **athrepsia**. In the adult and the aged it accompanies cachexia, phthisis, cancer, chronic diarrhoea, prolonged suppuration, etc. It is likewise associated with acute diseases (pneumonia, pyelo-nephritis, cystitis, typhoid fever, and the puerperal state). Thrush is contagious, but the soil must be favourable to its development. It was very common in children's hospitals, but is much less so to-day, owing to the rigid observation of prophylaxis and antisepsis.

Gubler has rightly attached great importance to the acidity of the medium. This assertion remains true, though we know that thrush may grow in alkaline media. Thrush cannot be cultivated in the saliva (Roux and Linossier); consequently dryness of the mouth and absence of saliva favour its development, as is seen in cachexia, infectious fevers, hectic fever, etc.

**Description.**—The **mouth** is the seat of election in thrush. The patches develop on the tongue, on the inside of the cheeks, and at other points of the mucous membrane, and it is easy to detach them by rubbing. The appearance of thrush in the mouth is preceded by a peculiar state of the mucous membrane, which is dry, shiny, and painful. It becomes red and glossy and desquamates, whilst the papillæ of the tongue stand out; the little whitish islands then appear, and unite to form the patches of thrush previously described. Thrush is often discrete and localized to the tongue, which appears covered by more or less irregular creamy patches. In other cases

the thrush is confluent ; it spreads over the inside of the cheeks, soft palate, tonsils, and pharynx, and may assume a yellowish or greyish tint, which at first sight resembles diphtheritic concretions. The reaction of the saliva is acid.

In the adult, mastication and deglutition become difficult and painful. The new-born infant cries, takes the breast with difficulty, and at last refuses it. The development of thrush in young children coincides with digestive troubles, vomiting, and diarrhœa, which in grave cases are accompanied by rigors, erythema of the buttocks, and cutaneous ulcerations. The enteritis may precede the thrush, or their development may be simultaneous. The gravity of the case is, however, subordinate to the general condition of the patient. If thrush is purely a local symptom, the prognosis is benign, and restoration to health is not far off. Some cases of secondary thrush are not of too unfavourable a prognosis. In a marasmic infant or in a cachectic individual the appearance of thrush is of evil omen, and though it may be possible to cure the local mischief its appearance is almost always the index of approaching catastrophe.

To sum up, as regards evolution and prognosis, thrush presents certain differences in children and in adults. In the adult, and much more so in the aged, thrush nearly always indicates a fatal issue. In a young child thrush often has the same serious signification ; in some cases, however, it is primary, and is simply a variety of stomatitis easily cured.

**Diagnosis—Treatment.**—The diagnosis is easy. In default of microscopical examination, which immediately removes all doubts, thrush must not be confounded with the flakes of curdled milk which may be removed by gentle friction, leaving the mucous membrane underneath absolutely healthy. Thrush should not be confounded with diphtheritic stomatitis, where the exudation occurs, not in projecting isolated grains, but in whitish adherent patches, which are quite different from thrush.

Alkalis must be given, as thrush does not flourish so well in alkaline media. After detaching the patches, the affected parts are painted several times a day with glycerine and borate of soda in equal parts. In very young children we must find a good wet-nurse, or give milk of good quality. If dyspeptic or intestinal troubles accompany thrush, two or three teaspoonfuls of lime-water in the milk should be prescribed, or a drop of Sydenham's laudanum in a little syrup and water. Cleanliness is imperative as regards the feeding-bottle, the breast of the nurse, and the mouth of the child.



## V. APHTHÆ.

**Description.**—Aphthæ are sometimes described under the name of “aphthous stomatitis,” a term which is not strictly correct, because the stomatitis is merely secondary. The name “aphthous fever” would be more suitable. In benign cases, which are the rule, prodromata are absent. The mucous membrane of the lips, the tip and edges of the tongue, the mucous membrane of the cheeks and palate, show red spots, on which vesicles, like those of herpes, develop. The vesicles become filled with a milky liquid, and are surrounded by an area of swollen mucous membrane. They break, and by the second or third day ulcers are formed.

The ulcers are circular; their size varies from a pin’s head to a lentil; some may be more extensive. The floor of the ulcer is greyish, and formed by a mass of degenerated cells. Its edges are irregular, fixed, and clean-cut. The ulceration rarely lasts more than a week, and heals without leaving scars.

The lesion produces burning pain, which becomes very acute at the moment of ulceration. The breath is foetid, and salivation is marked; as mastication and sucking are painful, a child refuses the breast, and an adult must be satisfied with liquid nourishment. The submaxillary glands are rarely enlarged. The general symptoms are trifling in slight cases; when the aphthæ are discrete, the symptoms comprise digestive troubles, accompanied or preceded by slight fever. They are, however, more marked when the aphthæ are confluent, and the name aphthous fever is then applicable. In this confluent form, which may be epidemic, fever and general symptoms precede the eruption.

The ulcers attack the roof of the palate, the velum palati, the tonsils, and the pharynx, and unite to form large ulcerated areas. In some cases ulcers are seen on the limbs, especially on the hands. The fever is high, the digestive troubles (vomiting and diarrhoea) are severe, and the disease, especially in children and old people, assumes an adynamic form which may be fatal.

**Ætiology.**—The ætiology is not well known. The disease is observed at all ages, sometimes in an epidemic form; in some individuals the coincidence of aphthæ, with certain eruptions—herpes, impetigo, and eczema—has been noticed, and a diathetic origin has therefore been suspected. We now take a different view of the question. In the seventeenth century, physicians spoke of contagion, and recent researches tend in effect to show that aphthous fever is an infectious disease of microbic origin. Clinically, there are striking analogies between the disease in man and aphthous fever in cattle. Numerous cases seem to prove that the disease may be transmitted from animals to man, and unboiled milk seems to be the most usual mode of contagion.

It is certain that in bottle-fed children the eruption is symmetrical on both sides of the median raphe of the palate.

**Treatment.**—When the disease is slight, the treatment consists in washing out the mouth with soothing lotions, or with a 5 per cent. solution of salicylate of soda (Hirtz). Chlorate of potash should be given, and healing will be hastened by weak applications of nitrate of silver. Purgatives are generally indicated. I obtained excellent results from a milk diet in a hospital patient who had had aphthæ for several years.

## VI. GANGRENE OF THE MOUTH—NOMA.

Under the name of **noma** it is customary to describe a form of gangrene which runs a special course and is of microbic origin; it occurs, especially in children, between the ages of three and five years, and forms a definite morbid entity.

**Description.**—This terrible disease commences insidiously and without pain in the mucous membrane of the cheek, and more especially of the left cheek. The mucous membrane assumes a cyanotic tint; a pimple, filled with ruddy serum, forms, breaks, and leaves in its place a greyish ulcer, which at this period is odourless. The ulceration spreads rapidly in depth and in extent. It assumes a greyish-black tint, and the breath becomes extremely foetid. In the fortunate but rare cases the process then stops; the base of the ulcer grows clean, granulations form, and cicatrization takes place. When the disease pursues its course, the ulceration extends. The ulcer becomes putrilaginous, black, and surrounded by an inflamed zone; from the third to the seventh day an indurated nodule forms in the deep tissue of the cheek, and indicates the seat of the gangrene and the region which it is about to invade. The lip and the cheek are œdematous. The skin of the cheek grows shiny and purple, while the saliva is sanious and foul-smelling.

At a given moment the cutaneous eschar appears; it is dry and appears depressed. The gangrene affects the entire thickness of the cheek, and may invade the lips, the nose, and the eyelid. The dead tissues then fall out in pieces, and leave in their place an excavation, which communicates with the mouth, and allows foetid liquid, drinks, or gargles to pass through. The bones of the face are not always respected. Necrosis attacks the bones of the neighbouring regions (maxilla, vault of the palate). In some cases the gangrene does not limit itself to the face, but develops simultaneously in the lungs, the vulva, the pharynx, the œsophagus, and the extremities.

The **general symptoms**, which are slight at first, become exceedingly grave when the skin is invaded. The fever may be intense; prostration and adynamia follow the febrile stage. Diarrhœa becomes incessant; the loss of



weight is considerable. In **noma** the lymphatic glands of the neck are scarcely enlarged. Death generally occurs between the fifth and fifteenth days, and in case of cure (which only happens in one case out of five) the patient shows scars, fistulæ, and sometimes hideous deformities.

**Ætiology.**—Noma is seen at all ages, but it is most common in children of from two to five years of age. The disease is always secondary, and it is remarkable that local lesions of the mouth, such as stomatitis, have almost no effect on its development, whilst general diseases, such as measles, scarlatina, typhoid fever, diphtheria, and scurvy, are favourable to its inception. Noma, however, has become very uncommon, owing to antiseptic measures which were previously unknown. Bacteriological researches have not yet given conclusive results.

The **treatment** consists in washing out the mouth with a solution of boric acid (4 in 100) and applying the thermo-cautery daily, care being taken to sustain the strength of the patient by tonics and stimulants.

## VII. SYPHILIS OF THE LIPS.

**Chancre.**—**Chancre of the lips** is so common that in Nivet's statistics concerning 338 chancres of the mouth and throat labial chancre accounts for 260. Not a single year passes without my seeing four or five cases. This chancre, like those of the oral cavity, may result from direct contact or from indirect contagion by means of infected articles—*e.g.*, spoons, glasses, pipes, etc. Chancre is likewise seen on the lips of the new-born infant when the nurse's nipple is infected. The chancre is here, as elsewhere, single; nevertheless, multiple chancres are not rare. Chancre of the lip commences as a trifling lesion. The patient fancies that it is a crack, a scratch, or a pimple. The comparison with the crack and the scratch is particularly applicable to chancres of the commissures and median part of the lip. In a few days its development is complete, and it then assumes various forms. Some chancres are papilliform, like a small slightly ulcerated tumour, with a reddish floor, which is indurated and bleeds easily. Other chancres, again, especially on the commissures of the lips, present the appearance of a flat, superficial, smooth ulcer, which is flesh-coloured, or covered at times by a diphtheritic layer. It affects the angle of both lips. Movement and rubbing make it bleed; at first sight it might be taken for a patch of eczema. Chancre of the lips is often papular, and forms an indolent tumour indurated at its base. It usually affects both mucous membrane and skin. It is covered by a crust, especially on its cutaneous segment. This crust, common to all chancres of the skin, is brownish; it may be removed after preliminary softening, and leaves exposed a red eroded surface, which bleeds on being rubbed. At first sight these chancres resemble a furuncle or an epithelioma.

All these chancres are alike in that they are painless, indurated at their base; and accompanied by painless satellite glands. The adenopathy is unilateral in lateral chancres and bilateral in median chancres; it is submental in chancres of the lower lip, and submaxillary in chancres of the upper lip, gums, tongue, and cheek. The labial chancre lasts from four to six weeks; it disappears without scarring, but leaves an induration, which in turn disappears. The **diagnosis** of chancre must be made from labial herpes, eczema, furuncle, and epithelioma. The indolence and the parchment-like induration of the base of the chancre, with the early and painless adenitis, are sufficient for diagnosis.

**Secondary Lesions.**—Syphilides of the lips appear as small opaline-tinted erosions (erosive variety), and if the syphilide attacks the skin, as, for instance, the commissures, it is covered with little crusts. These syphilides are very common in children suffering from hereditary syphilis. Syphilides are exceedingly contagious.

**Tertiary Lesions.**—The gumma, which is rare on the lips, has, however, a special predilection for the upper lip. It may be superficial or deep and intramuscular; it reaches the size of a lentil or of a cherry. The more numerous the gummata, the more is the lip deformed. The gumma goes on to the formation of an ulcer, with prominent encrusted edges and greyish, sloughy floor.

The sclerous syphiloma is seen on the lips. I had a very remarkable case of this kind under my care. It has a predilection for the lower lip; nevertheless, both lips are, as a rule, invaded. In its first period it causes general, and sometimes considerable, hypertrophy of the lip, which looks somewhat like a strumous lip. It has been likened to a horn or to a tapir's lip. The induration of the tissues is uniform, and sometimes mammillated. It does not terminate, like the gumma, in softening and ulceration. It ends when untreated in atrophy. The lip grows thin, and the mouth is contracted. Tertiary labialitis is not accompanied by pain and glandular reaction. The lips, which are rigid and deformed, partially lose their functions (articulation of sounds, mastication, deglutition).

## VIII. SYPHILIS OF THE TONGUE.

**Chancre.**—Chancre is rare on the tongue; it is more common on the tip than on other parts. Sometimes it forms a bleeding ulcer, with a red or greyish floor; at other times an ulcerated tumour inserted in the tongue. The base of the chancre is always indurated, and the adenopathy is unilateral or bilateral, according as the chancre is on the side of the tongue or in the median line. Chancres of the tongue are not indolent, like those of other regions; they are sometimes very painful, on account of the movements of



mastication, and on account of their incessant contact with saliva, tobacco, drink, and food.

**Secondary Lesions.**—Mucous patches of the tongue may appear as erosions, ulcers, nipple-like projections, and smooth patches. Syphilides on the edge of the tongue readily ulcerate; those which occupy the dorsal surface of the organ, when they are not properly treated, ulcerate, and form fissures, cracks, or furrows, with hard and swollen edges. Lingual syphilides are sometimes papular, especially on the back of the tongue, where they form bosses and nipple-like projections (toad's back). Some form vegetations, deform the tongue, and simulate a cancerous growth.

Yet another variety of syphilide is seen on the tongue—viz., smooth patches, which are, as it were, varnished. They are not erosive, but it may be said that at the site of the patch the mucous membrane is shaved off (Fournier) like a mown field (Cornil). These patches, which are fairly regular in contour, are only found at the back of the tongue, and border on the neighbouring regions where the villi are intact. This variety very closely resembles eczema, and a differential diagnosis becomes therefore necessary. Eczema of the tongue has received the most diverse names: lingual pityriasis (Rayer), geographical tongue (Bergeron), glossitis exfoliatrix marginata (Fournier), areal eczema, or desquamative marginal eczema (Besnier), and lingual psoriasis.

Eczema generally commences at the edge of the tongue. At first we see a small circle, or several little finely desquamating circles, with festooned or polycyclical outlines. Fully developed eczema is characterized by a raised patch, with a red or rose-coloured base. The patch or patches are surrounded by a border, or by yellowish and greyish ribbons, which are the remains of the normal mucous membrane. The eczema may remain marginal or may attack the whole tongue, but in unequal degree. When the eczema is general, the dorsal surface of the tongue is red and smooth; some greyish islands, in the shape of arabesques, remain, and are the remnants of the normal mucous membrane. This eczema has an acute course; it may last some days or several weeks. It always remains localized to the tongue, and does not attack the velum palati, floor of the mouth, or cheeks, like leucoplasia. The submaxillary glands are never enlarged. Sometimes the eczema is accompanied by such symptoms as pruritus, burning, and pain; sometimes these symptoms are insignificant. Such is the lingual eczema of those suffering from arthritis, gout, and dyspepsia. It is, however, certain that this eczema often appears, with the reminders, on the tongue of those suffering from recent or old syphilis. Parrot and Kaposi consider it a symptom of hereditary syphilis; syphilis is an important factor, and it might be considered a secondary **parasyphilitic** manifestation.

**Tertiary Lesions.**—1. **Gummata** may be superficial, in the dermis, or

deep, in the muscles. Intramuscular gummata are nowhere so frequent as in the tongue. The gummata always affect the upper surface of the tongue, and vary in size from a pea to a nut. When they are multiple, the tongue is mammillated, being, as it were, crammed with nodules (Fournier). It extends beyond the teeth, and is almost too large for the mouth. It becomes elephantiasis-like, and hinders articulation, mastication, deglutition, and respiration.

At times there is only one gumma, the rest of the tongue being free. At the Hôtel-Dieu I have had a patient whose single gumma\* formed a tumour of the size of a small nut in front on the right half of the tongue. His speech was spluttering; mastication and deglutition were extremely painful. He could not close his mouth, and the saliva drooled away. I put the patient upon injections of biniodide of mercury. The symptoms improved rapidly, and cure was complete in eight weeks. Such a gumma simulates other tumours of the tongue, *e.g.*, tubercular cold abscess, interstitial sarcoma, lipoma, and hydatid cyst.

2. **Ulcerations** may result in the circumscribed or diffuse gumma. It seems that tertiary ulceration may appear without having been preceded by any gummatous swelling. Gummatous ulcers are hollow, deep, with clean-cut edges and a sloughy floor, which is greyish, yellowish, or greenish, sanious, and sometimes covered with fungoid growths yielding a slight resemblance to cancer. These ulcerations are neither bleeding, like epitheliomata, nor purulent, like tuberculomata. Mastication and deglutition are very painful, or almost impossible.

A patient under my care for syphilitic ulceration had the front of the tongue eaten away. The pain was so severe that she kept the tongue quite still. She had great difficulty in drinking a cup of milk. She spluttered instead of speaking. She drooled saliva, and was unable to rinse her mouth or to clean her teeth. Contact with cold air was painful. Insomnia and want of food had led to a loss of 20 pounds in a few weeks. This proves once more that pain is not the appanage of tubercular ulcers, for syphilitic ulcerations of the tongue and throat are at times exceedingly painful. The ulcer was cured with injections of biniodide of mercury.

After a variable time the syphiloma, if treated, cicatrizes, but the tongue often remains furrowed.

In some cases the gummatous ulceration, if not treated, may become stationary, and last for months and years, without extending beyond its first limits. Fournier has seen ulcers which were one or two years old. He quotes "a very curious case of a tertiary lingual ulcer, dating back no less than twenty years, during which time the patient had submitted to no treatment whatsoever. And note this, as a proof of what specific medication can do in such cases—this ulceration had lasted twenty years. Mercury was

\* Dieulafoy, "La Grosse Gomme Solitaire" (*Clinique Médicale de l'Hôtel-Dieu*, 1903, 9<sup>me</sup> leçon).



administered, and in four days the ulceration commenced to get better ; in four weeks it was cured."

Syphilitic ulcers of the tongue may in exceptional cases become phagedænic or serpiginous.

Fournier mentions a woman who had thrice had tertiary ulcers at the back of her throat ; on the fourth occasion the tongue was invaded at the base by two large, deeply excavated ulcers.

A patient had a tertiary ulcer on the right half of the tongue. The ulcer ate in deep, grew larger, assumed a gangrenous aspect, and was stopped only at the end of three months. Fresh ulceration appeared three months later. The hole became gangrenous, and putrilaginous shreds were detached from it. "The tongue vanished in a kind of deliquescence." It took several months to cure the patient.

3. The **sclerous syphiloma** has a marked preference for the tongue ; it may be superficial or deep. The superficial sclerous glossitis attacks the mucous membrane ; the affected parts are, as it were, *depapillated* (Fournier), and contrast with the normal mucous membrane, covered with papillæ. The affected mucous membrane is sometimes of a vivid red, at other times whitish. Between the fingers the mucous membrane feels as if doubled over a cartilaginous plate ; its sensitiveness is lessened, and sometimes quite lost. In **deep sclerotic glossitis** the tongue is enlarged ; its dorsal surface is divided by more or less deep furrows into lobes and lobules. The tongue is, as it were, tessellated (Fournier), and in some cases the median furrow may attain a depth of 1 centimetre. Ulceration, unless provoked by exterior causes (alcohol, tobacco, friction against the teeth), is not seen. The disease runs a very slow course, without pain and glandular enlargement, when there is no ulceration. The tongue, which is as rigid as a piece of wood, gradually loses its functions ; articulation of words, mastication, and deglutition are badly performed. The gummatous and sclerotic forms are often associated ; the condition is then a sclero-gummatous glossitis. Syphilitic glossitis may appear as nacreous, laminated patches of a smooth or mammillated form ; these are called mucous patches. What is to be understood, in a general way, by **oral leucoplasia**, a name invented by Vidal, which, with advantage, has replaced the faulty term of psoriasis ? The name "oral leucoplasia" (white patches, opaline patches, or smoker's patches) is given to the silvery, laminated, and indurated patches which are found in smokers, or in gouty and syphilitic persons. Oral leucoplasia affects not only the tongue, but may also attack the lips, the cheeks, and the velum palati.

The patches are formed of thick, white epithelial scales, with superficial induration of the mucous membrane. These patches, which take years to develop, may become fissured and painful. Buccal leucoplasia may be cured or be prolonged indefinitely, or finally end in epithelioma. Debove stated this fact, which is generally admitted (Vidal). In such a case the patch assumes a papillomatous aspect, or rather is accompanied by painful deep-

seated induration ; the lesion reacts on the glands, and we have a buccal epithelioma. No difference exists between oral leucoplasia in people who are or are not suffering from syphilis. Kaposi admits leucoplasia of a syphilitic nature. Besnier considers syphilis as a predisposing cause. It is probable that we have here one of those lesions in which syphilis causes an important difference (parasymphilitic lesions). At the Hôtel-Dieu I had a patient suffering from sclerotic syphiloma of the tongue with leucoplasia. Both lesions were cured by injections of biniodide.

**Diagnosis.**—The diagnosis must be made when the syphiloma presents the form of a non-ulcerating tumour, of ulceration, or of sclerotic glossitis. I have remarked previously that the lingual gumma at times causes a firm or soft tumour, when the diagnosis is not always evident at first sight. This variety of lingual syphiloma closely resembles the lingual tuberculoma, or the intramuscular tubercular abscess, also called the tubercular gumma. Each presents similar localization, size, and appearance to both sight and touch. The existence of concomitant pulmonary tuberculosis is a probable, but not a certain, sign ; puncture of the tumour and examination of the liquid are sometimes necessary for the diagnosis (Marion). Interstitial **sarcoma** of the tongue may simulate gumma so closely that specific treatment may be needed to decide the point. Lingual **lipoma**, too, somewhat resembles submucous lingual gumma. Vergely says : “ If antisymphilitic treatment is prescribed, the rapid change in the gumma clinches the diagnosis.”

Let us now consider the ulcerative syphiloma. The tertiary ulcer must not be confounded with dental ulcers. The latter occupies the edge of the tongue, is generally elongated, and disappears if care is taken to file off or remove the offending tooth. **Tubercular** ulceration of the tongue differs from tertiary syphilitic ulceration. The edges are more jagged and less clean cut ; the ulcer is less crateriform ; it suppurates, and the adjacent mucous membrane is frequently studded with yellow tubercles. Koch's bacillus is often found in the scrapings from the ulcer.

**Epithelioma** of the tongue and tertiary syphilis have common characteristics which at times make the diagnosis difficult. Fournier, however, says that the syphilitic lesion commences with an internal induration, and the epithelioma with an external tumour. Syphilis causes deep ulceration, while epithelioma ulcerates on the surface. Syphilis forms multiple lesions, but epithelioma is solitary. Syphilitic lesions rarely bleed and secrete but little ; epithelioma bleeds easily and secretes much. The pains of syphilitic ulceration are less spontaneous and less radiating ; the ulcerous syphiloma is not accompanied by adenopathy (unless from secondary infection), while the epithelioma causes marked enlargement of glands. Treatment, so beneficial in syphilis, has no effect in epithelioma. Histological examination may prove the presence of epithelioma.



Do not let us forget that epithelioma may be grafted on a syphilitic ulcer of the tongue, just as on an ulcer of the stomach. In 1907, a patient came into the Hôtel-Dieu with gummatous ulceration of the tongue. Under mercurial treatment the improvement was rapid, and the epitheliomatous change took place during cicatrization.

Sclerous syphilitic glossitis must not be confounded with smoker's glossitis. The smoker's tongue may be indented and uneven, with greyish nacreous islets, especially at the tip; these characteristics recall lingual syphilis, and hence the diagnosis is at times difficult.

Syphilis of the mouth and of the tongue must be treated with injections of biniodide of mercury, in daily doses of from 1 to 2 centigrammes and more. The future will prove whether we may substitute Ehrlich's treatment, with 606, for mercury.

## IX. SYPHILITIC PERFORATION OF THE ROOF OF THE PALATE.

Perforation of the roof of the palate is not rare in tertiary syphilis, if I judge of its frequency by the number of people who come to hospital **with a hole in their mouths**. I quote here from my clinical lecture\* on this question :

A patient came to the Hôtel-Dieu for a "corroding disease" in his mouth. He had had syphilis twenty years before; ten years later rhinitis, followed by dacryocystitis. When he blew his nose, sanious fluid and thick crusts came out. Breath was foetid. The oral trouble appeared insidiously during the course of the chronic nasal syphilis. He found one day that he could not inhale the smoke properly, and the smoke passed through the nose. This was the first hint of any perforation. A little later the symptoms of perforation became evident: the voice became nasal, food and drink regurgitated into the nose. The perforation increased in size, and he noticed a "small hole" in the roof of his palate. He used a piece of gutta-percha to stop the hole. The disease growing worse, he came under my care. The breath was foetid; his speech was rendered almost unintelligible by the perforation; rhinitis not cured; nasal secretion abundant and muco-purulent; deglutition difficult; saliva running from the mouth; food retained in the nasal cavity by the gutta-percha, and putrefying there.

On removing the plug, the stench was horrible, and the mucous membrane bled profusely. After extraction of the foreign body, examination of the perforation and of the excavation was possible. On the buccal side a hole about the size of a florin occupied the centre of the palatine vault. It gave access to a conical excavation, the bottom of which was formed by the vault of the nasal fossæ. The edges of the perforation did not look like cicatricial tissue. The active progress of destruction, which had been going on for seven years, was evident.

Examination of the nasal fossæ showed the extent of the mischief: vomer almost completely destroyed; perpendicular plate of the ethmoid laid bare; cartilaginous septum almost intact, but much displaced to the right; mucous membrane of the nose swollen and suppurating.

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\* "Syphilis Naso-Buccale" (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 4<sup>me</sup> leçon).

I at once prescribed mercurial treatment (oily injections of biniodide of mercury). After six injections the rhinitis grew better; after fifteen injections the nasal mischief was arrested, and the ozæna disappeared. The injections were then suspended for about ten days and renewed. Collin made an obturator, so that the patient could talk, sing, drink, and eat as well as ever.

In another case I witnessed the process. A woman under my charge told us that a year before she had a so-called "severe cold in the head"—that is, syphilitic rhinitis. She could only breathe with her mouth open, because the nose was blocked; she lost her sense of smell, and the breath became foetid. The rhinitis went on eight or nine months, and three months ago her attention was drawn to the roof of her palate. She touched it with her tongue, and felt an abscess in course of formation. Later she noticed at the same spot a small piece of bone. This was a sequestrum. On swallowing, liquids came back through the nose, and though the perforation was not yet complete, since the sequestrum was still *in situ*, a fissure had established communication between the nose and the mouth.

Examination of the palate showed the process in full evolution. In the median line, in the centre of a reddish granulating ulceration, the sequestrum stood out; it was formed by a fragment of the maxilla, to which part of the vomer was ankylosed. There was, therefore, between the mouth and the nose only a simple fissure, filled in part by the sequestrum and the granulations of the neighbouring parts. Symptoms were absent, but when the communication was established the symptom-complex became clear: nasal voice, defective pronunciation, reflux of liquid and solid food through the nose, etc. Examination of the nasal fossæ showed that the cartilaginous septum was almost entirely eliminated, and, in the absence of treatment, the dissolution of the nose was close at hand.

I had in the Hôtel-Dieu a patient nicknamed "The Cornet-Player." Two years before he had syphilitic rhinitis, when the following incident took place: For several weeks he had felt on the roof of his mouth a small painless projection, which, however, gave no trouble, until one evening, when he was playing his cornet, the symptoms of perforation suddenly appeared. He could get no sound from his cornet, because the air passed through the nose. His nasal voice amused his friends. He took a glass of beer, but the liquid came back through the nose.

Next day he told us the history of the previous evening. We found in the roof of the palate a small perforation. His voice was nasal and his pronunciation defective. Fluid came back through his nostrils. I sent for his cornet, but he could not make a sound. The expired air passed through a hole in the palate. A gutta-percha obturator was put into the perforation; the symptoms immediately disappeared. In this case, too, the perforation supervened suddenly in the course of syphilitic rhinitis.

The following case also presents many interesting points:

"For the last seventeen years," said my patient, "I have gone about with a hole in my mouth. I have made obturators in caoutchouc and in gutta-percha as well as I could, but they are very imperfect. What can you do for me?" At the same time he removed the so-called obturator. His speech was at once transformed into a kind of unintelligible mumbling. I examined his mouth, and saw on the roof of the palate an enormous hole, large enough to take in a hazel-nut. The perforation had commenced seventeen years before as a small opening in the course of syphilitic rhinitis. The ulceration had gradually destroyed a part of the roof of the mouth, and yet during this long time the general health was not interfered with, and the syphilis did not manifest itself elsewhere. The necrosis was arrested by means of oily injections of biniodide of mercury. Berger made an obturator, and since then phonation, mastication, and deglutition have been performed to perfection.



**Description.**—The necrotic process shows itself first on the floor of the nasal fossæ, and the general rule is that syphilitic rhinitis almost always precedes the perforation of the roof of the palate (I am speaking of the roof, and not of the velum). It was believed for a long time that perforation of the roof proceeded from the mouth towards the nose. This is an error. The perforation proceeds from the nose towards the mouth. This view is held by Fournier and Duplay, and I agree with them entirely.

Besides, if we consider syphilis of the nasal fossæ as a whole, we see that it is the centre from which syphilitic lesions of the neighbouring parts start out. In one patient, syphilitic rhinitis ends in necrosis of the nasal bones, and the bony framework collapses. In another, nasal syphilis attacks the lachrymal bone and the nasal process of the superior maxilla. Dacryocystitis, with or without abscess, is the result. In a few, happily rare, cases nasal syphilis attacks more dangerous regions—the upper wall of the nasal fossæ and the cribriform plate of the ethmoid and the sphenoid bones. Syphilitic osteo-periostitis readily spreads into the cranial cavity, and may cause meningo-encephalitis, abscess of the brain, phlebitis of the sinuses, or “naso-cranial syphilis,” as Fournier calls it.

Perforation of the roof of the palate is also a consequence of nasal syphilis. “In consequence of tuberculo-ulcerous syphilides, or of gummatous periostitis of the floor of the nasal fossæ,” says Fournier, “a larger or smaller segment of the superior maxilla is denuded, and becomes necrosed. An eliminative peripheral phlegmasia occurs, and an abscess is formed under the mucous membrane lining the inferior surface of the diseased bone. This abscess points in the mouth as a small hemispherical tumour. At a given moment it opens spontaneously, or is opened by the surgeon. The orifice soon enlarges, and then a part of the necrosed segment appears bare on the roof of the palate. Lastly, the necrosed portion separates, and in a moment, to the great surprise of the patient, a more or less extensive perforation of the palate is formed, with two major troubles, which are its necessary consequence—alteration of the voice and reflux of solid and liquid foods introduced into the mouth.”

Two distinct phases then occur in this syphilitic process. In the first phase, which is often slow and insidious, the process is nasal. The lesion betrays itself by the symptoms of chronic coryza, with ozaena, crusts, mucopurulent secretion, and the formation of sequestra, easily distinguishable with the probe. This phase is followed by the phase of perforation. This perforation usually occupies the median line of the roof of the palate a little in front of the palate bones. It follows “the partial necrosis of the maxilla, or of the two maxillæ and the vomer at their point of meeting.” The perforation may be round or oval. At first it is the size of a pin’s head; later it may be as large as a florin or more, because it may invade a portion of the

roof of the palate. On the side of the mouth the lesion in the palate is also very indolent. My patient noticed nothing until the smoke from his cigarette came out of his nose, and revealed the perforation. The cornet-player felt no pain in his mouth when the perforation suddenly occurred.

At the moment of its formation the perforation does not always look like a hole; it may be only a simple fissure, as in the second case. "The hole" only exists when the sequestrum has been eliminated. This elimination may be slow, the fragment crumbling gradually; it may be sudden, the fragment being evicted as a whole, as in the cornet-player. The elimination of the sequestrum usually leaves but a small perforation, which is at this time perfectly curable, either spontaneously or by specific treatment.

As a rule, a syphilitic perforation left to itself tends to enlarge. The ulcero-necrosing process, which is slow in its course, recalls both rarefying osteitis and phagedæna. In two or three years the perforation attains the size of a halfpenny; in four or five years it is as large as a florin. It is somewhat surprising that syphilis persists in a certain region, and slowly pursues its ravages for ten or fifteen years, whilst it leaves the rest of the organism free. The same process is seen in other regions. We are not sufficiently familiar with these elective localizations of syphilis, and we too often make mistakes, because we believe syphilis to be incapable of lasting for ever in one spot, and of respecting the remainder of the economy.

The symptoms vary according to the size of the perforation. If the perforation is a simple fissure or a small opening, reflux of a few drops of fluid through the nose and slightly nasal timbre of the voice are its only symptoms; if the perforation is larger, nasal voice, defective pronunciation, and reflux of drink and food through the nose are the consequences. These symptoms recall paralysis of the *velum palati*; in both cases we find the same difficulties of deglutition and pronunciation.

Besides the nasal sound which renders the voice unnatural, certain words or letters cannot be pronounced. Thus "b" and "p" are pronounced "m," for the following reasons: Under normal conditions the letters "b" and "p" are formed by the column of expired air, which suddenly separates the lips and makes them vibrate. When the roof of the palate is perforated, the column of expired air loses its strength, because it is divided into two parts: one part separates the lips, and only succeeds in pronouncing "m," while the other passes into the nasal fossæ, and renders the voice nasal. The patient can partially remedy this condition by pinching his nose. Deglutition is rendered very difficult by the perforation of the palate, because the solids and liquids which are passing between the tongue and the palate, and are being pushed from before backwards, enter the hole and come back through the nose. Perforation of the roof of the palate betrays itself by



other symptoms, such as the impossibility or difficulty of sucking, of whistling, of blowing, or of inhaling the smoke of a cigarette.

**Pathogenic Diagnosis.**—Perforation may be also caused by tuberculosis and by lupus, but it is extremely rare. “Out of twenty palatine perforations,” says Fournier, “nineteen may be ascribed to syphilis.” I go even further, and I believe that out of forty perforations syphilis can claim thirty-nine. Yet this considerable disproportion, notwithstanding, the diagnosis, must be made. Tubercular perforations of the velum palati are more common than those of the roof. The latter alone demand our attention here.

Case observed by Quenu :

A patient suffering from phthisis complained of the passage of liquids through his nose. On examining the mouth, an antero-posterior ulcer was observed on the middle line of the roof of the palate, behind the incisors. The surface was covered with a yellowish-grey detritus. In the centre there was a small perforation. On the posterior part of the roof of the palate the swollen mucous membrane was covered with the tubercular granulations. Post mortem, the roof and the velum of the palate were removed. The mucous membrane was destroyed ; the bone formed the bottom of the ulcer. The perforation was due to the destruction of the fibro-mucous tissue which closed the anterior palatine canal. This perforation, like the bony canal, which is single on the side of the mouth, bifurcated into the two tubes which opened on either side of the nasal septum. On the nasal side the perforations were rounded.

Caussade has sent me the following case :

He had under his care a tubercular patient who complained of a smarting in the roof of the palate and of sharp pains on contact with food. On examining the mouth, the general pallor so frequent in tuberculosis was noticed. On the roof of the palate, to the left of the middle line, and, as it were, grafted on to the cicatrix of old lupus, there existed an irregular ulcer of the size of a florin. Around this ulcer, which had a sanious floor and loose edges, were some yellowish granulations. A probe readily entered the nasal fossæ, proving the existence of a small perforation. Tubercle bacilli were found in the purulent liquid bathing the ulcer ; they were also present in the yellow granules.

To sum up : the jagged, loose edges of the ulceration, the sanious floor, the yellow peripheral granules, and the presence of the bacilli in the pus, distinguished the tubercular from the syphilitic ulceration.

There is a perforating disease of the mouth which must be distinguished from syphilitic perforations. What is to be understood by **perforating disease of the mouth** ? Fournier thus names the trophic lesion, which is especially seen in tabetics ; it is comparable with perforating ulcer of the foot. Baudet has collected seven cases. This trophic trouble, which as a rule causes no pain, runs the following course : progressive absorption of the alveolar arches causes the teeth to fall out spontaneously. The falling out of the teeth is followed by absorption of the alveolar border. “Perforating disease” follows this bony absorption ; it commences with ulceration of the mucous membrane, and burrows down into the tissues until it perforates the bone. This perforation only affects the superior maxilla (though

trophic troubles are met with in both maxillæ). It may be unilateral or bilateral; it never occupies the centre of the roof of the palate, but the periphery, especially at the site of the first molars which have fallen out. The perforation is elongated in its antero-posterior direction, and may allow insertion of the finger; it establishes communication between the mouth and the nasal fossæ or the maxillary sinus.

Perforating disease differs from syphilitic perforations of the palate in the trophic troubles which precede it—viz., casting of the teeth and absorption of the alveolar borders of both maxillæ. As I have just stated, it never occupies the centre, but the periphery of the roof of the palate; the edges are usually insensitive. It is a trophic trouble almost always accompanied by positive or aberrant symptoms of tabes.

**Treatment.**—Treatment is in part prophylactic. As the perforation is preceded by syphilitic rhinitis, we should recognize and treat the rhinitis. In a syphilitic patient chronic coryza is always open to suspicion. Anyone tainted with syphilis who catches “a persistent cold in the head,” with purulent mucus and crusts, must be watched closely, because the rhinitis may end, for want of specific treatment, in ozæna, in destruction of the cartilages and the bones, and in perforation of the palate. With all the more reason must treatment be commenced without loss of time in a syphilitic patient who, during a chronic coryza, feels a swelling in the roof of the palate. This swelling is the prelude of a process which will terminate in perforation; the lesion, if taken in time, may be confined within limits.

The perforation at its commencement is always of small size, perhaps as large as a pin's head. At this moment it is sometimes curable by mercury, with or without the addition of iodides. When the perforation is large, we have only two means of remedying it—surgical intervention or a well-made obturator. In either case the patient must be at first put on specific treatment to limit the necrotic process if it is still active. This treatment consists in mercury, with or without iodide of potassium. As regards mercurial preparations, I give the preference to injections of an oily solution of biniodide of mercury.

## X. TUBERCULOSIS OF THE MOUTH.

Some years ago **tuberculosis** of the digestive canal was practically unknown; tubercular lesions and ulcers of the intestine had alone been described, but the other parts of the digestive tube had not been explored. Bayle had seen tubercular ulcerations of the mouth without giving them their real significance. This question, first studied by Ricord, was at his inspiration undertaken by Buzenet, and was clearly stated by Julliard; since these early researches of French origin, numerous investigations, both in



France and abroad, have rendered the question of bucco-pharyngeal tuberculosis classical.

The ulcerations of the mouth and the throat, which are sometimes met with in tubercular subjects, are not ulcerations of a cachectic nature, as was at first believed; they are really tubercular ulcerations, resulting from tuberculosis *in situ* (Trélat). By choice their seat is on the tongue, the pharynx, and the isthmus of the gullet. They may be solitary or multiple, and show the most varied forms. I shall study them separately in the mouth and in the throat. This distinction is, furthermore, necessary, because the ulcerations of these different regions have a somewhat different course.

**Tongue.**—Tuberculosis of the tongue shows two chief forms—tumour and ulcer. The tumour, called also tubercular gumma, lingual tuberculoma, or tubercular intramuscular abscess, is a soft tumour which is not painful, projects from the surface of the tongue, and may be as large as a cherry-stone or a nut.

Tubercular ulcers of the tongue are more common; they occupy, in order of frequency, the tip, upper, and lower surfaces of that organ. They develop in the following manner: A rounded yellowish spot, about 2 lines in diameter, is seen on the mucous membrane. The epithelium falls off, and the result is an ulceration, which increases in size and depth. The ulcer sometimes commences as a fissure, and brings about hypertrophy of the papillæ. When the ulcer has formed the edges are scalloped and clean-cut; the floor is covered with a layer of mucus and saliva, which on removal leaves a yellowish uneven surface exposed. Around the ulcer a bed of yellowish points is frequently seen; they were incorrectly considered to be follicular orifices, but they are really small subepithelial abscesses, or masses of tubercular follicles, having the structure of tubercular tissue, ulcerating in their turn, and becoming part of the principal ulcer.

Histological examination reveals the following characteristics: A section through an ulcer of the tongue shows that the granulating portions of the ulcer are formed of embryonic tissue. The surface of the ulcer is likewise infiltrated with embryonic tissue, and, deeper still, bundles of muscular fibres are seen, between and around which the embryonic connective tissue shows here and there little islands of more or less developed **tubercular granulations**. The granulations, or rather the tubercular follicles (giant cells, embryonic cells, and bacilli), invisible to the naked eye, are deeply seated in the muscular tissue of the tongue; they are found as much as  $\frac{2}{5}$  inch or more beyond the ulcerated surface.

The tubercular ulcer of the tongue is almost always solitary at its onset, thus differing from ulcers of the pharynx, the isthmus and the velum, which are often multiple. As the ulcer grows older, it becomes deeper, anfractuous, and deeply excavated, in distinction to the ulcers of the pharynx,

which are generally superficial. On the tongue the ulcers make slow progress, and coincide with the chronic forms of pulmonary phthisis, whilst the tubercular ulcers of the pharynx coincide rather with the rapid and general forms of tuberculosis. The ulcers in the mouth in some cases seem to precede the lesions in the lung; they may heal and cicatrize. The ulcers of the tongue are very painful upon contact with liquids or food, and patients have much pain in the acts of mastication and deglutition.

These pains are, however, not so acute as those occasioned by ulceration of the pharynx. Contrary to what might be supposed, tubercular ulcers of the tongue are rarely followed by adenopathy.

The tubercular ulcer of the tongue must not be confounded with chancre. The chancre has not a greyish and granular surface; it is not surrounded by a bed of yellowish points; its floor is more in relief, and it is not painful on pressure. Its base is much more indurated; the adenitis which it provokes is painless. Tubercular ulceration of the tongue is distinguished from **epithelioma** by the following signs: The surface of the epithelioma shows more vegetations; it bleeds easily, and gives rise to an oozing of foetid liquid. Its edges are very much raised and, as it were, everted. It is often the seat of spontaneous lancinating pains. It produces painful adenitis of slow course.

Tubercular ulcers are very rare on the **lips** and **gums**. They are sometimes consecutive to tubercular ulcers of the mucous membrane of the cheeks. Reclus records a case in which ulcers on the gums led to falling out of the teeth. Giraudeau cites a case in which osteo-periostitis, falling out of the teeth, and necrosis of the maxilla occurred.

**Roof of the Palate.**—Tubercular ulcers of the roof of the palate are more common than was formerly supposed. Hermandier has collected eight cases. One or more ulcers may be found; they are situated indiscriminately on all parts of the roof, and, as a rule, the velum palati, the pillars of the fauces, and the lips present at the same time ulcers of a like nature. Here, as elsewhere, the outlines are well defined when the ulcer is solitary; but when several ulcers unite, as frequently happens, the ulcerated surface presents a sinuous outline, and may be some inches in extent. The edges of the ulcers are reddish, puffed, and clean-cut; they are rarely indurated. Tubercular ulceration of the roof, like that of the tongue, is frequently surrounded by projecting yellowish points, which are tubercular nodules.

The formation of the ulcers is accompanied by smarting and pain, particularly on contact with food. In some cases (Quénu, Caussade)\* the ulcers end in perforation of the roof of the palate. The ulcer is rarely cured.

Painting with a solution of lactic acid (1 in 10) gives good results.

\* This question has been treated in the section dealing with perforation of the roof of the palate, as regards differential diagnosis.



## CHAPTER II

### DISEASES OF THE THROAT AND PHARYNX

IN studying the diseases of the throat and pharynx we shall at every moment meet with the word **angina**. This word angina (from ἄρχω, I strangle) was formerly employed to designate every disease which was accompanied by troubles of deglutition and respiration and was seated above the lung and the stomach. Though the word angina does not answer to-day to the ideas which created it, it has, nevertheless, been retained, and writers have essayed to make it comprehensible by associating with it certain adjectives which give it species and varieties.

#### I. ACUTE CATARRHAL ANGINA—ACUTE ERYTHEMATOUS ANGINA.

Under the name of **erythematous angina** and **acute anginal catarrh**, we describe inflammation of the mucous membrane of the posterior portion of the mouth and the pharynx, which is superficial, sometimes limited to a diffuse redness, as the name "erythematous" indicates, and sometimes accompanied by a pultaceous covering. The localization of the angina is somewhat variable : sometimes it is diffuse, and occupies both the pharynx and the posterior portion of the mouth ; at other times it affects certain points, as the isthmus of the gullet, the tonsils, or the pharynx.

**Description.**—**Acute catarrhal angina** commences with shivering, fever, lassitude, headache, and loss of appetite, which are slight in some subjects, but in others (in children especially) become so severe that we are liable to suspect the onset of some serious disease. The general troubles, which may be accompanied or not by gastric disturbance, precede the angina by a day or so ; they may appear simultaneously. The angina is ushered in by a sensation of dryness and smarting in the throat. Deglutition is painful ; the mucous membrane is red, dry, sniny, and close set with projections due to swelling of the muciparous glands ; while the serous infiltration of the sub-mucous tissue produces swelling of parts rich in loose cellular tissue, such as the uvula and isthmus of the gullet. The angina may remain simply erythematous, but sometimes as early as the second or third day the hyper-

secretion of the mucous membrane is shown by mucus on the pharynx and by caseous concretions, or pultaceous layers on the tonsils. These products, which are not adherent, do not resemble the membranes of diphtheria, and yet they may be associated with the diphtheria bacillus, as bacteriology has proved.

Acute angina is most frequently accompanied by a gastric or bilious condition. The tongue is white and coated, the anorexia is complete, attacks of nausea are frequent, and constipation is the rule. The fever falls from the second to the fifth day. The submaxillary glands are but slightly enlarged. Acute catarrhal angina does not last more than a week; it generally ends in resolution, but relapses and passage into the chronic state may occur in patients predisposed by some diathetic condition. Paralysis of the velum, and even general muscular paralysis have been described (Gubler); these cases are obviously diphtheria.

**Ætiology, diagnosis, and treatment** will be studied in the following chapter. For the time being let me remark that the better prepared the soil, the more important the rôle of the micro-organisms.

Mouth-washes and antiseptic gargles are indicated. I generally employ very weak solutions of boric acid (6 per cent.).

## II. ACUTE TONSILLITIS—SIMPLE TONSILLITIS—SUPPURATIVE TONSILLITIS—ULCERO-MEMBRANOUS TONSILLITIS—VINCENT'S ANGINA.

Inflammation of the tonsils, or amygdalitis, is also called **tonsillar angina**. In order to facilitate description I shall describe three varieties—simple, suppurative, and infectious amygdalitis—but these three varieties have **common characteristics which are often blended clinically**.

### 1. Simple Acute Tonsillitis.

**Description.**—Acute tonsillitis constitutes an important variety, and is the most common form of catarrhal angina. When the tonsillitis is slight, its description blends in part with the symptoms enumerated in the previous section. When it is severe, it may commence with a sharp rigor; the temperature, particularly in children, rises to 104° F., and the face becomes red and feverish. Deglutition soon becomes very painful, and its every movement is accompanied by contortions and grimaces. Liquids often regurgitate through the nose, and the patient refrains from swallowing his saliva. The voice becomes **tonsillar**; the opening of the mouth and the movements of the jaw are very painful. The exterior and lateral regions of the neck are painful and brawny, and the tonsils become so swollen that respira-



tion may be obstructed. Pains in the ear and auditory troubles occur only when the inflammation reaches the Eustachian tube.

Examination of the throat is not always easy, because the pain on opening the mouth causes contraction of the masseters. The tongue is thickly coated. The tonsils, which are red and bulky, are unequally affected by the inflammation, and present whitish concretions in the follicular crypts, so that the disease has received the name of "follicular tonsillitis." These concretions are yellowish-white, soft, caseous, easily detached, and have little tendency to reproduce themselves; they sometimes appear to be encysted. In spite of the acuteness of the symptoms, the disease ends in a few days by resolution, though it ends in suppuration in the form which I shall describe next.

Acute tonsillitis attacks all ages, but principally youth. When it is primary, its cause is cold and chills; when secondary, it is associated with other affections, such as coryza or grippe. Some people are predisposed to it, and amongst them second attacks are frequent. Spring and autumn and the menstrual periods are favourable to its appearance. The disease is often contagious and epidemic. This question will be discussed under the Bacteriology of Angina.

## 2. Suppurative Tonsillitis—Phlegmonous Angina.

I shall next describe *parenchymatous tonsillitis* terminating in suppuration.

**Description.**—The name *phlegmonous tonsillitis* would lead us to suppose, incorrectly, that the tonsil itself is the seat of the suppuration. The tonsil may suppurate in certain cases, when we see on the surface little abscesses filling the follicular crypts, but this is the exception. As a rule, the abscess forms in the peritonsillar tissue, at its upper and outer part, so that the tonsillitis becomes a *peritonsillitis*.

Phlegmonous tonsillitis may have an acute onset; more often it commences as a simple catarrhal angina, and the subsequent symptoms announce the formation of pus. These symptoms are a violent rigor, rise of temperature to 104° F., intense pain in the tonsil, and extreme difficulty in moving the jaw. During the next few days the fever remains high, and the pain spreads to the neck, the jaws, and the ears. Loss of appetite is complete; swallowing is extremely painful or impossible; the breathing is laboured, the voice is nasal, and the saliva flows continuously. The patient can neither open his mouth nor move his tongue; he can hardly speak; his neck is bent, immovable, brawny, and painful. The head is retracted towards the healthy side, and movement is almost impossible. Examination of the throat is very difficult, because the patient has the greatest difficulty in opening the mouth. When the throat is seen, the tonsils are often found to be covered

with whitish exudate, and when the abscess is in process of formation, the colour is sometimes purple. The breath is foetid, and the tongue covered with a thick coat. The velum palati is lowered and pushed back; the isthmus of the gullet is narrowed by the œdema of the mucous membrane and the projection of the affected tonsil. The patient experiences a sort of suffocation, which caused the name of "esquinancia" to be given formerly to the disease. Two or three days later the pain becomes shooting, and we may perhaps feel a soft swelling indicating pus. Artificial or spontaneous opening of the abscess is followed by considerable relief. Spontaneous rupture occurs generally from the sixth to the eighth day at the intersection of the pillars of the fauces, and the patient expectorates bloody foul-smelling pus.

Phlegmonous tonsillitis is not serious as a rule, and recovery occurs in about a week; nevertheless, gangrene, œdema of the larynx, phlebitis of the jugular vein with suppuration, and opening of the abscess into the cellular tissue of the neck, thrombosis of the jugular veins, and ulceration of the arteries, followed by fatal hæmorrhage, have been noted. In spite of their great rarity some of these complications deserve notice. Vergely has collected sixteen cases of ulceration of the internal carotid artery, or of the branches which run from the inferior pharyngeal and palatine arteries to the tonsils. In some cases the hæmorrhage is lightning-like in its rapidity, and the catastrophe occurs without any warning. Sometimes the hæmorrhage recurs several times, and it is necessary to ligature the carotid artery. In one case a terrible hæmorrhage ceased spontaneously, and was not followed by death.

The **ætiology** of phlegmonous angina is in part blended with that of simple tonsillitis; it is likewise subject to recurrences, and is often grafted on a chronic catarrhal or subacute angina, which has, so to say, acted as the port of entrance.

### 3. Infectious Tonsillitis.

**Description.**—During the past few years several cases of tonsillitis have been classed amongst the **infectious** diseases, and rightly so (Bouchard). Simple or suppurative tonsillitis may be one of the numerous localizations of "tonsillar fever." Other local manifestations of the disease may be found in the testicle, ovary, or kidneys, and in this respect they bear a likeness to the localizations produced by other infectious diseases, such as mumps. Tonsillar fever is an infectious disease, like pneumonia, which chooses the lung as its seat of election, but also exerts its action on the pleura, the endocardium, the meninges, the kidneys, etc.

Attention having been drawn to this point, it is certain that tonsillitis sometimes resembles an infectious disease. In some cases the local angina is slight, and yet we find that the general symptoms, including rigors, fever.



lassitude, and want of appetite, show an **intensity** and a **duration** which are more in accordance with the hypothesis of a general infection, than of a simple inflammation of the tonsil.

**Orchitis** is one of the manifestations of tonsillar fever, and it is not without analogy to the orchitis of typhoid fever and of mumps. Orchitis occurs both in the slight and in the severe forms of angina. It appears especially during the decline of the tonsillitis; it is unilateral, painful, and characterized by inflammation of the testicle and effusion into the tunica vaginalis. It lasts from one to three weeks; it terminates by resolution, rarely by suppuration, but it may leave behind slight atrophy of the testicles. **Ovaritis** has likewise been observed.

The **nephritis** that appears in the course of tonsillar fever is characterized by albuminuria, and in some cases by lumbar pain, œdema, and uræmic symptoms. Although this nephritis is generally transitory and superficial, it may in some cases result in Bright's disease.

Articular pains, pseudo-rheumatism, cutaneous eruptions, polymorphous erythema, purpura, ulcerative endocarditis, purulent pericarditis or pleurisy, peritonitis, meningitis, broncho-pneumonia, phlebitis, suppurative otitis, nephritis, orchitis, and ovaritis may occur in the course or the decline of tonsillitis. Some of these complications are more usually associated with suppurative tonsillitis; others, again, are rather the appanage of follicular tonsillitis. Some of these complications are the index of a very grave prognosis; they may appear in the course of acute tonsillitis which began with mild symptoms.

These facts prove that many varieties of tonsillitis are ordinarily infectious diseases. We shall see what bacteriology teaches on this subject. For the moment it is important to remember that, clinically speaking, tonsillitis may vary in its severity and its course. It may behave like a local inflammation, and comprise the whole disease; it may be accompanied by multiple manifestations, like the infectious diseases.

**Bacteriology.**—Bacteriological researches have taught us that in every catarrhal or phlegmonous angina the exudate and the caseous masses in the crypts of the tonsils contain numerous microbes, which may likewise be met with in the mouths of healthy persons.

Micrococci, diplococci, streptococci, staphylococci, pneumococci, pneumobacilli, *Bacillus coli*, tetragenous pseudo-diphtheritic bacilli, and all the saprophytes of the mouth, may be found in the different varieties of angina just described. The principal share, however, belongs to the streptococcus, in the following proportions:

In twenty-two cases of angina and tonsillitis Veillon found pathogenic microbes in the following proportions: the streptococcus was present twenty-two times, the pneumococcus was associated with it sixteen times,

and the staphylococcus was associated with it twice. Not a single one of these microbes is specific. What part do they play in angina and tonsillitis? Why and how do they become virulent and pathogenic?

Sometimes there is contagion, and a healthy individual receives the pathogenic agent from the outset. In other cases we must have recourse to the old ideas of "morbid spontaneity," but we call it "auto-infection." Under the influence of conditions which concern both the seed and the soil, some of which conditions are known to us (cold, overwork, microbic associations) and some of which are unknown, the microbe or microbes increase in virulence, and disease results. The pharyngeal mucous membrane is rich in lymphoid tissue and in macrophages and microphages, which are in a continual state of defence. If the defence weakens, **phagocytosis** is in default (Metchnikoff), and the individual is "in a state of morbid receptivity."

In some cases the pathogenic agents do not invade the tonsils from the surface, but may enter on the deep side. In such a case the angina is secondary; the patient has been suffering from an infectious disease, and as the tonsils "retain and destroy the microbes, it is not surprising that they suffer in consequence from time to time."

However this may be, the pathogenic agents, and notably the streptococcus, act according to their virulence and according to the soil they meet with. The results and the complications enumerated may be due either to the streptococcal toxine or to the passage of the streptococcus into the blood and the organs. Perhaps injections of antistreptococcal serum, which are so efficacious in erysipelas, may be utilized in streptococcal angina.

**Diagnosis.**—On principle, examination of the throat in a patient attacked by even the simplest angina must never be omitted. Examination of the throat is painful, and at times difficult. Children undergo it with ill-grace, but it must be insisted on, for fear of error in the diagnosis. The throat is freed, by means of a gargle, from the mucus which encumbers it, and then explored, either by a direct or reflected light.

In one of the following chapters I shall give the diagnosis between tonsillitis and diphtheritic angina. In many cases, however, it can only be decided by bacteriological examination. Angina may precede or accompany the articular manifestations of acute rheumatism. It causes great difficulty in swallowing when the muscles of the pharynx are affected (Chomel).

The initial angina of scarlatina precedes the cutaneous eruption, and may even be present alone, without any eruption. This angina is characterized by the severity of the fever and the purple coloration of the mucous membrane, due to the scarlatinal eruption, which occupies not only the



isthmus of the fauces, but also the cavity of the mouth and the inner surface of the cheeks. Scarlatinal angina is sometimes remarkably indolent. It is often accompanied by a pultaceous exudation formed of soft, whitish patches, which are readily detached, and leave the subjacent mucous membrane quite intact. This exudate, which is also found in the angina of typhoid fever, is formed by the accumulation of degenerated epithelial cells.

The exanthem of measles is characterized by a red stippling, and by prominent spots upon the roof of the mouth, and later the back of the pharynx and the posterior pillars of the fauces (Lasègue). The bucco-pharyngeal exanthem is preceded or accompanied by ocular, nasal, and laryngo-bronchial catarrhs, which announce the invasion of measles.

**Erysipelatous angina** may be consecutive to erysipelas of the face, or may commence in the pharynx. It is ushered in by a more violent rigor than that of catarrhal angina. The difficulty of swallowing is very great, and pustules are sometimes present on the mucous membrane, which is of a claret colour (Cornil). The submaxillary glands are very congested.

The angina which sometimes accompanies urticaria is easily recognized on account of the rash on the face and the body.

Pharyngeal **syphilis** must not be confounded with non-syphilitic angina. I am not speaking of those cases in which more or less abundant mucous patches are found in the throat—in such cases the diagnosis is evident—but I allude to those cases characterized by a diffuse or circumscribed erythema of the velum palati, pillars, tonsils, and pharynx, which has often a bright red shade, and fairly often coincides with syphilitic erythema of the larynx.

I shall describe tuberculosis of the tonsil in one of the following sections. For the moment I shall confine myself to mentioning a form of acute tuberculosis of the tonsil which exactly resembles follicular tonsillitis. The diagnosis can only be decided by bacteriological examination. **Ulceromembranous tonsillitis** occurs, and is often associated with ulceromembranous stomatitis. It is due to fusiform bacilli and spirilla, described by Vincent, and runs the following course: An adult or a child is taken ill with pains in the throat. The mucous membrane is red and oedematous. Two or three days afterwards there appears on one tonsil (more rarely on both) a greyish membrane, which may be removed by scraping, and leaves bare a reddish and bleeding surface. The false membrane, which has a nauseating smell, may extend to the uvula and the pillars. "The large number of fusiform bacilli and spirilla in the false membrane, the reproduction of the false membrane as long as these microbes are present, and the clearing up of the mucous membrane as soon as they commence to decrease, are sound arguments for admitting their action in the genesis of ulceromembranous tonsillitis."

membranous tonsillitis and stomatitis." The shedding of the false membrane leaves a deep, anfractuous **ulcer** with prominent edges. The breath is foetid, and the glands are enlarged. The disease lasts from one to three weeks. Ulcero-membranous tonsillitis must not be confounded either with diphtheria or with chancre of the tonsil. The search for fusiform bacilli and spirilla can alone confirm the diagnosis.

**Treatment.**—The **treatment** of angina and of catarrhal tonsillitis consists at first in the use of lukewarm gargles of decoction of mallow, which are alternated with the following gargle :

|   |                 |    |    |    |    |    |         |
|---|-----------------|----|----|----|----|----|---------|
| R | Water           | .. | .. | .. | .. | .. | O.i.ss. |
|   | Boric acid      | .. | .. | .. | .. | .. | ℥ii.ss. |
|   | Essence of mint | .. | .. | .. | .. | .. | ℥ii.    |

The pain in the throat is relieved by the following lotion :

|   |                         |    |    |    |    |    |         |
|---|-------------------------|----|----|----|----|----|---------|
| R | Glycerine               | .. | .. | .. | .. | .. | ℥v.     |
|   | Borate of soda          | .. | .. | .. | .. | .. | ℥ss.    |
|   | Chlorhydrate of cocaine | .. | .. | .. | .. | .. | gr. iv. |

The inflamed parts are moistened every hour with this lotion.

Cold compresses, covered with oiled silk, to the throat give good results. Saline purgatives and emetics are to be reserved for cases in which the angina is associated with some gastric or bilious condition. In phlegmonous angina the best thing to do is to make use of the means previously mentioned. As to opening the abscess with a bistoury, we must not overlook the possibility of severe hæmorrhages which sometimes occur in peritonsillar phlegmon.

### III. RETROPHARYNGEAL ABSCESS.

Acute retropharyngeal abscess is frequent in very young children ; it is sometimes the result of post-pharyngeal adenitis (Verneuil). At the commencement these abscesses look like a simple pharyngeal angina ; later the intensity of the fever and of the pain indicates the formation of pus. At this period the local symptoms are very variable, according as the abscess occupies the upper or the lower region of the pharynx.

In the former case (**superior retropharyngeal abscess**), deglutition is painful, and the posterior wall of the pharynx shows a swelling which may be reached with the finger, and may yield fluctuation.

In the latter case (**inferior retropharyngeal abscess**) it is difficult to feel the tumour, because the patient does not lend himself to manipulation. Compression of the larynx by the abscess causes respiratory troubles, which have often led to a wrong diagnosis of croup, œdema of the glottis, or a foreign body in the larynx. To avoid this error, we should ever remember



the rigidity of the neck and the early appearance of severe dysphagia. The fever is very high, delirium is frequent, and the patient becomes comatose, collapses, and dies suddenly. "In view of the alarming prognosis of untreated retropharyngeal abscess, the practitioner must intervene in every case which comes under his notice. The fear of sudden death, which is always possible, demands intervention as soon as the diagnosis has been established."

Post-mortem examination reveals general purulent infiltration of the walls of the pharynx. The infiltration may reach the mediastinum, spread along the œsophagus and the vertebral column, and dissect out the muscles of the neck.

#### IV. CHRONIC ANGINAL CATARRH—GRANULAR ANGINA CHRONIC TONSILLITIS.

**Description.**—I shall include **chronic anginal catarrh** and **granular angina** in one description, because they are frequently seen in the same patient, and are not sufficiently distinct to deserve a separate description.

The symptoms of **chronic angina** show themselves slowly and without pain. The patient experiences a sensation of dryness, tickling, or pricking in the throat and nasal fossæ, which is most marked in the morning. The pharynx is covered with thick, viscid masses of mucus, which cause a kind of hawking, and are only expectorated after several attempts.

The throat is dry; deglutition is sometimes difficult, and slight deafness occurs if the inflammation has reached the Eustachian tube.

The soft palate and the wall of the pharynx are red, shiny, granular, sometimes swollen, and streaked with varicose veins. The uvula is long and œdematous. The **granulations** are formed by the hypertrophy of the glandular follicles; they may be discrete or confluent, and occupy the posterior wall of the pharynx, the anterior pillars of the fauces, and the uvula. They have a red tint, and are rarely larger than a hemp-seed. The large granulations are due to the hypertrophy of the adenoid tissue, which is so abundant in this region. The presence of small pustules characterizes **acneiform angina** (Lasèque).

The nasal fossæ and the larynx often participate in the inflammation. Chronic coryza and laryngitis result therefrom, and their symptoms are added to those of pharyngitis. Posterior rhinoscopy shows the pharyngeal tonsil to be raspberry-like, and covered with muco-purulent secretion, which flows down the pharynx. The posterior extremity of the turbinate bones is often hypertrophied.

Headache and neuralgia of the occipital nerve are often present in chronic pharyngitis, as Vergely has pointed out.

**Granular angina** is essentially **chronic**. It is subject to subacute attacks and to relapses. It is brought on by repeated irritation of the mucous membrane of the pharynx (tobacco, alcoholic beverages, and, in singers, contact with the air). It is especially frequent in people suffering from gout, rheumatism, and herpes.

The local **treatment** consists in cauterization, pigments, and gargles. Mineral waters containing sulphur, alkalis, and arsenic find their application in constitutional conditions.

**Chronic Tonsillitis.**—The disease has a marked predilection for youth. It is common up to the age of puberty, but very rare in the adult, unless it be a relic of early years. It is especially met with in lymphatic and scrofulous patients. It is sometimes the result of acute tonsillitis, but it is more often a recrudescence, for it may be said that the disease never stops completely in predisposed persons. Each crisis is marked by similar symptoms, but in different degrees, according as the attack is more or less acute.

The chronic inflammation often induces hypertrophy of the tonsils. The hypertrophy involves the lymphoid follicles, the connective tissue, and the mucous membrane which covers the tonsil, and penetrates into its crypts. The hypertrophied tonsil weighs from 40 to 100 grains (Chassaignac). It is soft if the hyperplasia affects the lymphoid tissue; indurated if the hyperplasia involves the connective tissue. It is a chronic fibrous inflammation.

The hypertrophied tonsils project more or less, and may touch the uvula. They present all possible shadings, from rose to deep red. The pultaceous secretion, rich in microbes, lodges in the sulci on the surface. The hypertrophy of the tonsils is accompanied by chronic adenitis of the submaxillary glands, which may break down. Coryza and chronic blepharitis are also seen in lymphatic and scrofulous subjects. When the hypertrophy is considerable, the breathing is embarrassed, particularly during sleep. This dyspnœa produces violent contractions of the diaphragm, which, in their turn, depress the lower ribs, and contribute to the **deformity of the thorax** (Dupuytren, Lambron).

With this question of tonsillar hypertrophy is connected the hypertrophy of the pharyngeal tonsil and of the adenoid tissue in the pharynx. Ruault has described these infantile forms. Certain infants, even though hardly weaned, show a tendency to breathe and to sleep with their mouths open, accompanied by characteristic rattling and snoring. These signs indicate hypertrophy of the pharyngeal tonsil, which may develop later into the **adenoid growth**. In other infants the adenoid hypertrophy spreads, and is, so to speak, "a hypertrophic pharyngitis, affecting all the adenoid tissue at the back of the mouth."

The **diagnosis** of enlarged tonsils in infants and youths may present many difficulties. A simple hypertrophy of the three tonsils is often in



reality latent tuberculosis. I shall discuss this point in detail under Larval Tuberculosis of the Three Tonsils.

The medical **treatment** of chronic tonsillitis consists in touching the tonsils frequently with alum, nitrate of silver, or tincture of iodine. Surgical treatment is only of use in hypertrophy. The tonsils are removed, or cauterization is employed by means of Paquelin's thermo-cautery or Krishaber's galvano-cautery. This method, which I have often found effective, gives excellent results. The tonsil is destroyed after a small number of sittings.

## V. DIPHTHERIA.

**History.**—Diphtheria has been known since the days of antiquity, but its various manifestations were regarded as so many distinct diseases. Often, indeed, the nature of the disease was misunderstood. For instance, epidemics of diphtheria, raging in the form of angina, had given birth to the names "**Egyptian ulcer**," "**Syrian ulcer**," "**ulcera pestifera**" (Areteus), observers mistaking for ulcerations the local lesions in the throat, which sometimes assume an ulcerous appearance. Epidemics of diphtheria raging as laryngitis (croup) created the names, "**garrotillo**" (epidemics of the end of the sixteenth century in Spain), "**morbus strangulatorius**" (epidemics at the commencement of the seventeenth century in Italy), "**suffocating angina**"—names which thoroughly prove the localization of the disease in the larynx. **No relation**, however, had been established by observers between the laryngeal and pharyngeal forms of the disease; they looked on them as distinct diseases.

Fothergill, during the epidemics in England (1774), and Huxham, during the Plymouth epidemic (1751), fell into a similar confusion, and spoke of **gangrene** of the throat. Home (1765), a Scotch physician, invented the name "**croup**"; he deserves the credit of having clearly separated the strangulatory affections of the larynx from the pellicular maladies of the pharynx, but he was wrong in not recognizing the identical nature of angina and of croup, and on this point confusion still continues.

Samuel Bard (1771) introduced some order into the chaos, in that he clearly established the identity of the different local forms of diphtheria. The work of Bard, however, remained in an unfinished state, and found no response. It was reserved for Bretonneau to establish by clinical and pathological proofs the **common origin** of these various morbid conditions, to prove that they are one **specific disease**, to which he gave the name of "**diphtheritis**." Bretonneau likewise showed that gangrenous angina was in most cases diphtheria, and he completed his work by creating **stridulous laryngitis** (false croup)—a disease which simulates croup, but which has nothing in common with it.

Bretonneau's teaching was spread by Trousseau, who adopted the doctrine of his master, whilst slightly modifying it. Trousseau, however, substituted for **diphtheritis**, which, according to Bretonneau, gave a kind of preponderance to the inflammatory element, the word "**diphtheria**," because he considered the inflammatory element of less account.

He described **malignant diphtheria**, which had not been previously classified; made the operation of tracheotomy popular, and foresaw the toxic origin of diphtheritic paralysis. He studied the disease in its entirety, and, with a master hand, wrote a marvellous description of it.

The ideas of Bretonneau and Trousseau found opponents in Germany. Virchow and Rokitansky tried to overturn the theory of identity advanced by Bretonneau;

they invoked the aid of pathological anatomy, but they only succeeded in re-establishing the confusion. They admitted croupous and diphtheritic inflammations—**croupous** in the case of superficial fibrinous exudates developed on a simple epithelium, like that of the respiratory passages; **diphtheritic** in the case of deep fibrinous infiltration, which developed on the thick, stratified epithelia, and ended at times in necrobiosis of the tissue. The German school was in the wrong, and the idea of unity and the specific nature of diphtheria, so well set forth by our great French masters, received a brilliant confirmation from the discovery of the pathogenic microbe of diphtheria, just as the unity and specific nature of the different forms of tuberculosis (tubercular and caseous products), the work of Laënnec, attacked by the German school, were proved by the discovery of the tubercle bacillus.

In 1883 Klebs discovered the bacillus, cultivated it, and recognized its aptitude to form membranes, but he could not give proof of its specific nature. The irrefutable demonstration of the specific nature of the diphtheria bacillus is due to Roux and Yersin, who in three successive papers have given us the results of their admirable labours. These results may be formulated in a few sentences. The bacillus of diphtheria produces the diphtheritic membranes, but it is not the sole agent capable of producing false membranes. Other micro-organisms, such as the streptococcus, pneumococcus, staphylococcus, and especially a small diplococcus, are apt to produce false membranes.

It is, then, not in the false membrane that the specific nature of the diphtheritic bacillus must be looked for. Its specific nature lies in the toxine which, amongst other complications, determines paralysis. In some cases the diphtheria bacillus may cause toxic symptoms and paralysis, without any previous production of false membranes.

Diphtheria is eminently a toxic disease; it readily becomes infectious by association with micro-organisms which increase the virulence of the diphtheria bacillus, or which add their own share of virulence. The streptococcus is the most active agent, and many complications that occur during the course of pharyngeal or laryngeal diphtheria are due to the secondary infections which these agents produce.

These views are given in detail in the sections on Diphtheritic Angina and Pseudo-Diphtheritic Membranous Angina, as well as in the section on Croup.

**The Bacillus of Diphtheria.**—A particle of false membrane is taken with a platinum loop from the throat of a child suffering with diphtheria, and spread out on slides. The films thus obtained are dried by passing them through the flame and stained with Roux's blue or with gentian violet. The preparation is washed in water and placed under the microscope. The Klebs-Löffler bacillus is seen in the midst of numerous micro-organisms (coccus, streptococcus, bacteria). It is a straight or slightly curved bacillus, as long as the tubercle bacillus, but thicker; its rounded extremities stain more deeply than its middle portion. "In diphtheria of rapid course, after staining with methylene blue, the superficial parts of the false membrane are seen to be formed by a layer of small diphtheria bacilli, almost in a pure state. They are separated from the mucous membrane, which has lost its epithelium, by a layer of granular fibrin, and by a fibrinous network adherent to the mucous tissue. The most superficial zone of the false membrane often contains different microbes, rods, and micrococci in chains, mixed with masses of diphtheria bacilli, which, on the other hand, predominate immediately below" (Roux and Yersin).

The diphtheria bacillus is readily cultivated in tubes of gelatinized serum. By means of a platinum wire a particle of mucus or of false membrane is taken from the throat, and smears are made with this wire on the surface of the coagulated serum. It is advisable to inoculate two or three tubes without taking fresh material. We thus obtain in the second or third tube



colonies which are more discrete and characteristic. The tube is closed by a plug of wool and put into the oven at a temperature of from  $35^{\circ}$  to  $37^{\circ}$  C. At the end of eighteen hours, or even earlier, colonies appear; when well developed and sufficiently isolated, they are characterized by rounded discrete or confluent spots of a greyish-white, more opaque at the centre than at the periphery. I call these colonies **papular**, because, when they are well developed, they project from the surface of the serum. We shall see in the following sections that the diagnosis of diphtheritic angina and pseudo-diphtheritic angina can only be made by cultures and bacteriological examination. For the moment, therefore, I will leave the distinctive signs of their microbes.

The diphtheritic colonies having been obtained by culture, it suffices to take a particle and stain with Roux's blue. When the preparation is finished, it is examined with an immersion lens, and the diphtheria bacillus, which is always immovable, is then seen.

The bacilli are often disposed in groups of three or four. They may be arranged in parallel lines, or may represent the letters **V**, **X**, **L**; they may resemble an acute or circumflex accent, but are never placed end on. They look like short, squat needles which have been allowed to fall in small heaps on a table (Martin). "The diphtheria bacillus remains alive in cultures for a long time. Bacilli contained in closed tubes without air, and protected from the light for thirteen months, have given active cultures" (Roux and Yersin).

In virulent diphtheria the bacilli are generally long, numerous, and sometimes curved. They form numerous colonies. When the disease is less virulent, the colonies are less numerous, and the bacilli are shorter and less curved.

A very short bacillus has been described by Escherich, who calls it the pseudo-diphtheria bacillus. Opinions are divided as to whether it is a diphtheria bacillus which has been attenuated and robbed of its virulence, or whether it represents a different species, having nothing in common with the bacillus of diphtheria. This bacillus is found in the mouth of healthy subjects, and is present in the angina of scarlatina and of measles. I have found it in a contagious angina, accompanied by herpes and membranes. Cultures inoculated into guinea-pigs never cause the death of the animal; thus this characteristic is said to differentiate the pseudo-diphtheria bacillus. The experiments of Martin, however, show that these bacilli, which are non-virulent in the case of the guinea-pig, kill small birds that are protected by a preventive injection of antidiphtheritic serum. Martin has further proved that these non-virulent bacilli can secrete the diphtheritic toxine in suitable media. Finally, several experimenters have been able to increase the virulence of these originally inoffensive microbes.

These facts show that, before creating a group of pseudo-diphtheria bacilli, very defined characteristics have to be found, which will enable us to differentiate them from the diphtheria bacillus.

The diphtheria bacillus has no tendency to penetrate the organs; Barbier and Tollemer maintain, however, that they have discovered the bacillus in the

cervical and bronchial glands, the spleen, and in the medulla (post-mortem researches).

**Experiments.**—The diphtheria bacillus is readily cultivated in alkaline veal broth. Half a cubic centimetre of culture injected into the cellular tissue of a pigeon kills it in less than sixty hours. A rabbit dies in a few days from an injection of 2 to 4 c.c. of culture. The guinea-pig is usually selected, and dies in less than thirty-six hours from a very small injection.

In a few hours local oedema, with a hæmorrhagic patch, develops at the seat of inoculation. The bacilli remain confined to the cedematous region; they do not enter the bloodvessels, the lymphatics, or the organs, and, in spite of their rapid diminution in the inoculated region, the disease continues its course, by reason of the toxine which has been formed *in situ*.

After death the inoculated animals present identical lesions: general dilatation of the small vessels, congestion of the suprarenal capsules and the kidneys, swelling of the glands, pleurisy in the guinea-pig, and degeneration of the liver in the rabbit.

By means of pure cultures, it is possible to reproduce the diphtheritic membrane on the trachea, the conjunctiva, or the pharynx of pigeons and hens, and on the vulva of the female guinea-pig; all that is required is to paint the mucous membrane, which has been previously excoriated. It is possible to cause lesions and symptoms resembling croup in the trachea of the rabbit. Diphtheria of the skin may likewise be produced in animals, provided the epidermis has been previously removed.

**Diphtheritic Membranes.**—In the pathological condition the membranes invade the mucous membrane and the skin, provided always that the skin has been denuded of its epidermis and the mucous membrane of its epithelium. The membranes appear spread out, and reform with extreme facility. The membranes are much less common on the skin than on the mucosa; they may develop on the surface of blisters, leech-bites, herpetic vesicles, cracks in the nipple—in short, wherever the integument is denuded of its epidermis.

**Cutaneous diphtheria** (for example, diphtheria which develops after a blister) presents the following characteristics: The invaded part becomes painful, red, and covered with a firm, greyish membrane, which is difficult to detach. The edges of the wound swell and take on an erysipelatous tint. Pustules form; the epidermis falls off, and the bared skin is, in its turn, invaded. The false membranes fall off and reform, but finally disappear; the cutaneous surface, however, has a feeble tendency to cicatrization, and may remain red, bleeding, and painful for a long time. Cutaneous diphtheria is usually grave, because it is often associated with secondary infections (streptococci), and is sometimes followed by gangrene, but especially because it is prone to be the starting-point of general intoxication and malignant diphtheria (Trousseau). The membranes invade the mucous membranes of the pharynx, nasal fossæ, larynx, bronchi, conjunctiva, eyelids, cornea, vulva, prepuce, anus, gums, mouth, Eustachian tube, and œsophagus. These various localizations of diphtheria, however, are far from being frequent; the usual localizations are in the nasal fossæ (diphtheritic coryza), the back of the mouth, the pharynx, which we shall study under the name of **Diphtheritic Angina**, and in the larynx, which we have described elsewhere under the name of **Croup**. The false membranes are only, as I have just said, local manifestations of diphtheria; they are the most frequent and formidable manifestation, for the membranes in the larynx and respiratory passages may choke young patients who are not treated in time. Diphtheria, however, reveals itself also by toxic manifestations caused by a poison which we shall study later.

**Diphtheritic Toxine.**—The pathogenic microbe, so plentiful in diphtheritic membranes, does not enter the blood or the organs of patients who have fallen victims to diphtheria. How are we to explain the fact that colonies of bacilli, localized to some part of the mucous membrane or the skin, can cause the organic lesions (kidney, liver,



nerves), the poisoning and the paralysis which so often occur? "In diphtheria the infection is not produced by microbic invasion of the tissues, but by diffusion within the body of a toxic substance formed on the surface of the mucous membrane outside the body, as it were."

This poison, discovered by Roux and Yersin, "has been the subject of a series of researches, considered even to-day as the best monograph which has ever appeared in bacteriology, and rightly so." Funk, in speaking thus, echoes the general opinion. Roux, by discovering the diphtheritic toxine, prepared the way for the discovery of the antitoxin.

The toxine is obtained by cultivating the virulent diphtheria bacillus on broth, in contact with air. In order to prepare an active toxine, it is necessary to make use of a very virulent culture. It is also necessary to secure the exact alkalinity of the broths; for this purpose the peptonized broth must be made alkaline, until it no longer reddens blue litmus paper. The broth is placed in Fernbach flasks with a flat bottom, so that the layer of liquid may be shallow. After sterilization in the autoclave, fresh virulent bacilli are sown in the broth, and heated in the stove to 37° C. Roux, having proved that the diphtheritic poison was produced more rapidly and in greater quantity when the cultures were made in contact with air, invented an ingenious method of passing a current of moist air into the culture.

For this purpose, he connected the side aperture of Fernbach's flasks with a tube, so as to make a vacuum. In three or four weeks a culture rich in virulent toxine is obtained. A layer of whitish sediment, like a crust of ground-glass, is deposited at the bottom of the flask. This is a deposit of bacteria, and on the surface of the broth a whitish-looking scaly veil, composed of younger bacilli, is formed. The liquid between these two layers is turbid at the commencement of the culture, but gradually becomes more and more clear.

The finished cultures are filtered through a Chamberland candle, and the clear liquid is stored in well-filled corked flasks, and kept in the dark at the ordinary temperature. The toxine thus prepared generally kills a guinea-pig of 500 grammes in forty-eight hours after a dose of  $\frac{1}{10}$  c.c.

The inoculation of the toxine into certain animals, such as the guinea-pig, rabbit, etc., produces symptoms and lesions similar to those seen in patients who have succumbed to toxic diphtheria. Subcutaneous injection in these animals produces a fibrinous sanguinolent cedema at the point of inoculation, followed by swelling of the glands, diarrhoea, and hurried, shallow breathing. The post-mortem lesions are: congestion of the intestine and bloody fluid in the bowel, hæmorrhagic congestion of the suprarenal capsules and the kidneys, yellow staining and fatty degeneration of the liver, pleuritic effusion, very marked dilatation of the vessels, myocarditis, and imperfect coagulation of the blood. Injections of toxine not only produce the acute intoxication just described, but also paralysis (and this is one of the most interesting sides of the work of Roux and Yersin), that closely resembles diphtheritic paralysis, which we shall discuss in one of the following sections. The diphtheritic toxine is closely related to the diastases and the venoms.

**Microbic Associations.**—We have just studied the products of the diphtheria bacillus—i.e., the membranes and the toxine—but the secondary infections play a great part in the symptoms of diphtheria. The streptococcus, the staphylococcus, other cocci, the pneumococcus, and the bacteria of putrefaction assist in the morbid process. These microbes, and especially the streptococcus, give rise to suppurative adenitis, bronchopulmonary complications, otitis, and gangrene.

Clinical observation has taught us that the membranes play quite a secondary part in some cases. From the very onset the malady has all the symptoms of the most septic and infectious diseases; it is accompanied by albuminuria and hæmorrhage, and invades the entire economy, causing adynamia and often killing by syncope.

Trousseau, on account of its extreme gravity, gave to this disease the name of **toxic** or **malignant** diphtheria. Bacteriology has confirmed clinical research in every point; it has given us the reasons and taught us the causes of the different forms of diphtheria. The production of the membranes is due to the presence of the bacillus. The symptoms of intoxication are due to the poison elaborated by the bacillus. The infectious symptoms are due principally to the associated microbes. We shall study later the association of the diphtheria bacillus with the streptococcus—an association which reciprocally increases the virulence of the pathogenic agents, and causes malignant and fatal diphtheria.

## VI. DIPHTHERITIC ANGINA.

Diphtheritic angina does not always show the same course and symptoms. In its most common and favourable form, which has been called since Trousseau's time normal diphtheritic angina, the intoxication and the infection are, fortunately, of secondary importance. The disease invades the throat to a greater or less extent; it often reaches the larynx, especially in children, and sometimes engenders severe paralysis, especially in adults. This proves that the disease is not without danger; but, after all, the course and complications of the so-called normal angina can be nearly always cured with serum, and the rapid and fatal complications that are so frequent in the malignant form need not usually be feared.

In the latter form, which is rightly known as toxic infective angina, and which I shall continue to call **malignant**, the general symptoms are far more important than the local ones. From the outset the malady has the appearance of a grave infectious disease. The entire economy is invaded in a few hours or in a few days. It is not the false membrane nor death by croup that is to be feared, but the rapid poisoning and collapse, to which patients often succumb.

Some authors have attempted to classify diphtheritic angina according to bacteriological examination. Normal diphtheritic angina would thus be a monomicrobial angina, in which the bacillus exists in a pure state. On the other hand, malignant angina would be a polymicrobial angina, in which the diphtheritic bacillus is associated with other microbes—notably the streptococcus. There is some truth in these assertions, and these monomicrobial and polymicrobial forms will be described in this section, but I would hasten to say that **such a radical bacteriological classification would be erroneous**. I admit that the normal angina is often monomicrobial, but in many other cases it may be associated with cocci, diplococci, staphylococci, and even streptococci (as I have just proved), and yet the angina does not lose its characteristics as a frank or normal angina. *Per contra*, it is not only a polymicrobial angina which can be malignant. I shall quote fatal cases of diphtheritic angina which were not polymicrobial, the diphtheria bacillus being present in a pure state (Roux, Martin).



It is therefore best to retain the clinical classification, and describe normal angina, which is but little toxic, and a malignant angina, which may be subdivided bacteriologically into angina with toxic symptoms and diphtheritic angina with infective symptoms. And yet these two clinical forms—normal and malignant angina—are not always clearly defined, and we find mixed forms. The disease may commence with mild symptoms, and yet after a few days it becomes malignant. Here, as in all toxic and infectious diseases, the poison and infectious agents show degrees and reserve surprises for us. Without affecting the truth of the description, it is possible to conform to the custom established by Trousseau, and to describe separately the two varieties.

### 1. Normal Diphtheritic Angina.

**Description.**—While normal diphtheritic angina spares no period of life, it is much more frequent between the ages of three and seven years. Its onset is usually insidious and less acute than that of acute tonsillitis. It **installs itself by stealth**. The fever is moderate, and may fall in three or four days; the sore throat is not severe, and the patient may feel hardly any pain on swallowing. While the above statement is practically true, we find many exceptions, since bacteriology has taught us not to be deceived by the multiple disguises of diphtheritic angina.

The disease (in my description I borrow from Trousseau) commences with more or less vivid redness of the pharynx and swelling of one tonsil; in some cases both are involved. A well-defined white spot is soon visible on the affected organ. This spot at first consists of a layer resembling coagulated mucus, which rapidly becomes thickened and of a membranous consistency. At the commencement this exudate is readily detached from the surface, to which it is adherent by filaments penetrating the follicles of the tonsil.

The underlying mucous membrane is healthy, except for the destruction of the epithelium. The mucosa may appear to be hollowed out because it is swollen, and forms a kind of pad around the exudate. Ulceration is exceptional. After some hours the pseudo-membrane is more prominent, and covers the greater part of the tonsil. It becomes more and more adherent at the points first invaded, and takes on a yellowish or greyish-white tint. As a rule, the roof of the palate commences to become inflamed. The uvula swells, and is often covered with membrane within twenty-four hours. The membrane now appears on the other tonsil and the pharynx.

We find, however, many exceptions, since bacteriology has taught us not to be misled by the polymorphism of diphtheritic angina. We often see, not membranes, but erythematous, pultaceous, lacunar, or herpetic angina.

From the first, or in a short while, we notice enlargement of the glands

at the angle of the jaw on the same side as the affected tonsil. This **adenitis** is of great importance, because it is rarely absent. The glands are hard and movable, the periglandular tissue being unaffected. The adenopathy is due to the diphtheritic toxine, and we shall see that it changes in character in the streptococcal forms.

The symptoms of angina are more pronounced by the second or third day; the dysphagia is more severe, but the fever is slight, and may even disappear at this period. The child often has an anæmic look, due to diminution in the hæmoglobin, as Quinquaud has shown.

On examining the throat, the uvula, the pillars of the fauces, both tonsils, and the pharynx are in some cases covered with false membranes, which may have a lardaceous aspect. The membranes are produced so readily that they reappear in a few hours upon an area which has been entirely cleaned. This rapid development is often seen in young subjects. The throat may be completely covered with false membranes in thirty-six hours in a child of three years, whilst several days are necessary in an adult. The patches grow thicker from the addition of new layers, which are formed below the old ones. The most superficial layers are readily detached, but the deeper ones adhere to the mucous membrane, and cannot always be removed without causing slight bleeding. Some patches are, as it were, let into the surrounding mucous membrane, which projects, and gives the false impression of an ulceration.

The membranes do not long preserve their whitish or yellowish aspect, because their colour is altered by liquids, vomited matter, drugs, or blood, which has come from the nasal fossæ. They then take on a greyish or blackish tint, which, joined to the fœtor present, leads to the wrong idea of gangrene. This gangrenous appearance is common in the adult, but rare in children, and explains the name **gangrenous sore throat** given to diphtheritic angina by several authors. Gangrene is exceptional in diphtheria. Bretonneau was loath to admit it, but it may be seen in grave cases and in certain epidemics.

Normal diphtheritic angina is often accompanied by **albuminuria**. This symptom is not of serious import.

In favourable cases the disease is not of long duration. After eight to ten days the glandular swelling diminishes, the membranes no longer reform, the mucous membrane becomes clean, and the difficulty in swallowing disappears. If the patient has not been treated in time with serum, convalescence is long, and may be complicated by serious troubles.

In the adult extension of the angina to the **larynx** is rare, but croup consecutive to the angina is very common in children when serum has not been used. This terrible complication occurs when the angina has scarcely terminated—in fact, **croup** often arises while membranes are still



present in the throat. It is announced by changes in the voice and by fits of dry cough at short intervals (see **Croup**).

The frequency of croup in children makes diphtheritic angina much more serious in them than in adults. When, however, croup occurs in the adult, it is more dangerous than in the child. The termination of the disease with syncope, which is so common in malignant forms, is unusual in normal diphtheritic angina.

Angina is not always the initial manifestation of diphtheria. It often follows diphtheria of the **nasal fossæ**, which has been latent for some days. It may also follow diphtheria of the mouth, larynx, or skin, but this sequence is very rare.

**Prognosis.**—Normal diphtheritic angina is generally benign. It may, indeed, be said that, apart from the complications of croup and bronchopneumonia, which are much more common in children than in adults, normal diphtheritic angina is almost exempt from danger. It is, therefore, important to distinguish it from the grave toxic variety. When the angina is normal, the fever is slight, and abates in a few days. The submaxillary adenitis is moderate, and rarely appears early. The colour is not from the first pale and leaden. Albuminuria is transient or absent. The pulse is of good quality. The bacteriological examination does not, as a rule, reveal the long, curved bacilli, and when other microbes are associated with the diphtheria bacillus, they are unimportant (Brisou's coccus), or are present in very small numbers (staphylococci, streptococci).

Nevertheless, although normal diphtheritic angina excludes up to a certain point the idea of toxicity, it is none the less true that in the most normal case of diphtheritic angina some toxic symptoms exist. The glandular swelling, the albuminuria, and the decoloration of the tissues are evidence of poisoning by the diphtheria toxine.

These toxic symptoms, which are slight, I admit, I shall call **primary**, because they are contemporary with the angina. Diphtheritic angina, however, may be normal and benign in appearance, and yet excite **secondary** toxic symptoms. The poison accumulates insidiously in the system, and causes diphtheritic paralysis.

Lastly, in very rare cases an angina which seemed to be normal may become grave, or even fatal, if not treated in time with serum, as the cases cited by Roux and Yersin and by Chaillou and Martin prove.

We see, then, that, for different reasons, diphtheritic angina, even when normal and but slightly toxic, is not exempt from danger. There is, therefore, all the more reason to diagnose and treat the disease **without delay**.

When we discuss the treatment, there is one point on which I shall lay stress. It is not sufficient to treat diphtheria with injections of serum. The earlier the treatment, the better it succeeds.

## 2. Toxic or Infective Diphtheritic Angina—Trousseau's Malignant Diphtheria.

In the preceding section I described the action of the bacillus in producing false membranes which are harmless in the throat, but dangerous in the respiratory passages, where they may produce croup and asphyxia. Normal diphtheritic angina is the most common form, and is generally seen in sporadic cases. It may be seen, however, in certain epidemics, even when malignant diphtheria is raging. "In fact," says Trousseau, "in a family of whom four, five, or six individuals are stricken with the disease, normal diphtheritic angina, with or without croup, will be the general rule. The malignant form, which poisons patients in the same way as septic diseases do, will be the exception."

In principle, as I stated above, the most normal case of diphtheritic angina is always accompanied by some toxic symptoms. The pallor of the face, the rise of temperature, the swelling of the cervical glands, and the albuminuria, are symptoms due to absorption of the diphtheritic poison. These symptoms do not entail danger when they are moderate; they are more or less a part of the common description of many cases of normal diphtheritic angina, and do not affect the prognosis. In some cases, however, the toxic symptoms are so acute that the patient literally dies of infective poisoning, or, as Trousseau said, of **malignant** diphtheria. The following examples will show how rapid the evolution of malignant diphtheria may be.

**Description.**—"One of my colleagues, Walleix," says Trousseau, "was in charge of a child suffering from diphtheritic angina, which was improving. While he was examining the child's throat, she coughed up some saliva, which entered his mouth. He took the disease. Next day he noticed a small pellicular concretion on one of his tonsils. Fever developed, and in a few hours the tonsils and the uvula were covered with false membranes. Serous liquid began to flow from the nose. The glands and the cellular tissue of the neck swelled considerably. He became delirious, and died forty-eight hours later, without any laryngeal complication."

A country practitioner was attending a child suffering from croup, for which tracheotomy became necessary. During the operation the entrance of blood into the trachea caused fears of suffocation. The anxious physician applied his mouth to the wound in the throat in order to suck out the fluid which was entering the trachea. He inoculated himself with the disease, and died forty-eight hours later of malignant diphtheria.

Henri Blache was in charge of a child who had undergone tracheotomy. At the end of the third night he complained of slight sore throat. Acute fever was present, and false membranes on the tonsils. In a few hours enormous swelling of the neck and continuous discharge from the nose. In twenty-four hours delirium, and death seventy-two hours later from malignant diphtheria, without having shown the slightest symptom in the respiratory passages. Some years ago Potain asked me to see a family suffering from diphtheria. On my arrival, I was told that the two children had died from diphtheritic angina. The mother had been taken the previous day with sore throat and prostration. My examination gave me a very bad impression. She was deadly pale; her lips were blue; her pulse wretched. The cervical glands and the



cellular tissue formed a brawny mass. Marked albuminuria. Throat covered with diffuent foetid membranes ; sanious nasal discharge. She was quite lucid, and had no illusions as to her condition. "Whatever you do," said she, "I shall die of the disease which has just killed my two children." She passed away next day. The respiratory passages were quite free from any infection.

The fulminant form is, fortunately, very rare. In other cases the disease runs its course in eight or ten days, as in the following example from Trousseau's writings : A child, twelve years of age, had been attacked by slight angina three days previously. She was taken to the hospital. On examination, the foetor of the breath was very striking. Putrilaginous membranes at the back of the throat and on the roof of the palate. On the right side much swelling of the cervical and maxillary glands. This glandular swelling, which from the first meant a grave prognosis, increased during the next few days, and involved the cellular tissue of the cervical and submaxillary regions. A most alarming phenomenon supervened—viz., **erysipelatous redness** of the skin, pointing to deep inflammation of the parts. On the third day diphtheritic coryza and profuse epistaxis made the outlook very gloomy.

With such formidable symptoms, although the respiration remained normal, the prognosis was most grave. She grew cold, just like a cholera patient. She had a tendency to syncope. Her pulse was extremely feeble and slow. The respiration remained free, and the respiratory passages were not affected by the diphtheria. She refused to take any nourishment. The glands became smaller, the nasal lesions improved, and the erysipelatous redness disappeared ; but yet, in spite of this deceptive improvement in the local signs, the child died on the tenth day, "poisoned by the diphtheritic venom which had infected her." She died of syncope whilst turning round and refusing to take a drink. The picture of malignant diphtheria is not always as complete as it was in the above case. The horrible foetor of the breath, the brawny induration of the cellular tissue of the neck, and the erysipelatous tint, on which Trousseau and Borsieri have laid such stress, may be absent ; in spite of the absence of these symptoms, however, the excessive pallor, the weak pulse, the obstinate refusal to take food or drink, the diarrhoea, the severe and early albuminuria, the rapid loss of strength, point to malignant diphtheria of most grave prognosis, although the respiratory passages are absolutely free.

We may now ask how bacteriological researches can explain these malignant cases.

**1. Toxic Malignant Diphtheria.**—In some cases the grave or fatal complications are due to the diphtheria toxine alone, and other microbes are absent. Roux and Yersin report half a dozen cases in their monograph of 1888. Cases 1, 3, 5, 6, 7, 10, concern young patients who died of toxic angina. Bacteriological examination yielded pure cultures of the diphtheria bacillus.

Martin has collected twenty-eight cases of fatal toxic diphtheria, in which bacteriological examination showed only the existence of long and curved diphtheria bacilli, without other microbes. Chaillou and Martin (in their memoir, July, 1894) have collected ten fatal cases of toxic diphtheria, in which pure cultures of diphtheritic bacilli were found, without other microbes. The long curved bacillus was most frequently present.

In the above cases clinical observation was always confirmed by bacteriological examination. It is therefore possible for us to give an exact clinical

description of this **pure** toxic angina, which constitutes one of the varieties of malignant angina.

The temperature is higher than in normal diphtheritic angina. It oscillates between 102° and 104° F., and remains at this point for some days. It is a bad sign if the temperature does not fall after the fourth or fifth day (Martin). The changes in the pulse follow the oscillations of the temperature. The false membranes are thick, adherent, and of a greyish-white colour. They generally cover the tonsils, the uvula, the pillars, and the back of the throat, leaving no intervals of healthy mucosa. In some cases, however, the false membranes are discrete.

The swelling of the cervical glands is more rapid and more marked in toxic than in normal diphtheria. Nevertheless, it is rare to see such marked swelling as in diphthero-streptococcal angina.

Albuminuria is more constant in toxic than in normal diphtheria, though it does not usually appear before the third day, and it does not disappear when the disease is about to end fatally.

In toxic diphtheria the colour is more pale and leaden. The lips are at times cyanosed, even though there is no danger of asphyxia. Diarrhœa is frequent. Distaste for food and drink is usually present. Rapidly increasing weakness, compressible pulse, and tendency to syncope, which do not exist, or are only slight in normal diphtheria, are a marked feature in these cases.

Bacteriological examination, without being quite conclusive, shows that numerous colonies of long curved bacilli belong to toxic diphtheria. Experimental cultures on broth furnish valuable information as to the degree of toxicity. Brieger and Wasserman were able to extract from the blood of a child who died of toxic diphtheria, with collapse and cardiac failure, a quantity of diphtheria toxine, and to prove its specific action by inoculation of guinea-pigs. Martin has obtained similar results.

Let us now compare the experimental lesions due to the injections of diphtheria toxine with those found in patients who have died from toxic diphtheria. They are quite similar: heart flabby and yellow, parenchymatous and interstitial myocarditis, ecchymotic pericarditis, congested liver, dilatation of the capillaries, and fatty infiltration of the hepatic cells; infiltration of the tubuli contorti of the kidney and dilatation of the glomerular vessels; enlarged spleen and hypertrophy of the Malpighian corpuscles; swelling of Peyer's patches and of the follicles; infiltration of the intestinal mucous membrane.

The symptoms and the lesions explain the action of the toxine in the case of toxic diphtheria. Let us now see how malignant diphtheria behaves when other organisms are also present.

**2. Malignant Diphtheria with Microbic Associations.**—In most cases the malignant forms of diphtheria are both toxic and infective. They are



due to the reciprocal exaltation of the diphtheria bacillus and of the microbes which are associated with it. The streptococcus plays the principal part, as the following experiments of Roux and Yersin show :

Prepare a weak culture of diphtheria bacilli, so that inoculation produces slight local œdema in a guinea-pig. Prepare another culture of virulent streptococci, so that 1 c.c. will kill a rabbit in twenty-six hours. Prepare likewise a broth culture containing a combination of attenuated diphtheria bacilli and virulent streptococci. Take two guinea-pigs and inoculate them with  $\frac{1}{2}$  c.c. of the attenuated diphtheria culture ; take two others and inoculate them with  $\frac{1}{2}$  centimetre of the streptococcus culture, and two more with 1 c.c. of a mixture containing equal parts of the two cultures. In a few days the guinea-pigs inoculated with the weak culture show slight œdema and a little scab at the point of inoculation, but no other complications. The guinea-pigs inoculated with the streptococcus show an abscess at the point of inoculation, but the guinea-pigs which have been inoculated with the mixture die two days later.

At the point of inoculation false membranes and œdema containing diphtheria bacilli and streptococci appear. The lesions found post mortem comprise dilatation of the vessels, congestion of the suprarenal capsules, and effusion into the pleuræ. Tubes of serum are inoculated with the œdema fluid. Next day the diphtheria colonies are well developed, while the streptococcus has only grown to a very small extent. It is, therefore, easy to separate the two microbes. We can now see how much the harmless diphtheria bacillus has been reinforced by contact with the streptococcus. For this purpose two cultures are made—one with the inoffensive diphtheria bacillus, and the other with the reinforced bacillus. The cultures are heated in the oven for a fortnight at 35° C. in a current of air, filtered through porcelain, and injected into the veins of four rabbits. Two rabbits are given 10 c.c. of the cultures of the reinforced bacillus. They die in thirty hours, so virulent is the culture. The other two rabbits are each given twice as large a dose of the culture of the weakened bacillus, and live two months. It is only after a long time that they grow thin and succumb to paralysis.

The streptococcus and the diphtheria bacillus, when **separated**, are incapable of killing guinea-pigs ; when **associated**, they kill rapidly, owing to the lesions of diphtheria.

When we compare the clinical and experimental facts (Roux, Yersin, Barbier, Martin, Chaillou), we find in both instances identical results. In angina, which is both diphtheritic and streptococcal, either of the two microbes, isolated and cultivated separately, is rarely virulent, as animal inoculation shows. The two microbes together reciprocally increase their virulence, and hence the exceptional gravity of diphthero-streptococcal angina.

The following figures indicate the gravity of these cases : According to Martin's statistics, 8 deaths in 10 cases ; Chaillou and Martin give 13 deaths in 14 cases ; Tézenas had 3 deaths in 3 cases. To this variety of malignant angina belong the fulminating cases which I referred to at the commencement of this article. Their symptoms have been admirably described by our predecessors, and bacteriological studies have in part explained their pathogenesis. At the onset of malignant angina the face is pale, leaden, and bloated ; the lips are cyanosed, though there is no danger of suffocation. The neck is enormous and " proconsular " (Saint-Germain) from the swelling of the glands and the œdematous infiltration of the cellular tissue. The skin of the face and neck is often shiny and erysipelatous in appearance (Borsieri, Trousseau). The breath is excessively foetid, especially when the exudates are invaded by the bacilli of putrefaction. Deglutition is very painful. The false membranes of the throat have often a gangrenous appearance. The temperature is usually high, and the pulse is rapid, thready, and irregular. Albuminuria comes on early and is persistent ; diarrhœa is frequent ; hæmorrhage is common (epistaxis, bleeding gums, purpura). In such cases, the nasal fossæ being often invaded by both microbes, there is a copious purulent or bloody discharge, with rejection of false membranes.

Be the angina rapid or slow, its gravity is considerable. Extreme restlessness with or without delirium, and prostration bordering on coma, are present, while collapse or syncope ends the scene. Death sometimes supervenes from certain complications, such as infective croup, purulent bronchitis, or streptococcal broncho-pneumonia. If recovery takes place, convalescence is long, and at times accompanied by ulceration of the throat, mouth, or nose, and by suppurative adenitis.

This description differs, as will be seen, from that of purely toxic angina. In the latter the diphtheritic poison is the chief factor ; in diphthero-streptococcal angina, infection by the streptococcus claims a large share. This fact is made quite clear in Barbier's paper, in which he shows that the streptococcus enters the blood. It reaches the blood of the pulmonary veins, and thence affects the lungs, the bronchi, the mitral valve, the articulations, the pleuræ (purulent pleurisy), the middle ear (purulent otitis), the tissues of the neck (adeno-phlegmon), the walls of the pharynx (retropharyngeal abscess), the spleen, etc.

What I have just said of the streptococcus is in part applicable to the staphylococcus. Although **diphthero-staphylococcal** angina is not so grave as diphthero-streptococcal angina, it still claims a place in the group of malignant anginæ. The truth thereof is evident on consulting the statistics published on this subject.

**Conclusions.**—We can now formulate the following conclusions as to the pathogenesis of diphtheritic angina :



1. Diphtheritic angina is called normal or benign when it is neither toxic nor infective. Its benign nature is due to the feeble virulence of the diphtheria bacilli, or to the feeble receptivity of the soil in which they develop. It is also due to the absence of other microbes, or to the feeble power possessed by the associated microbes to increase in virulence. Nevertheless, normal angina, in spite of its benign appearance, is not always free from danger. It may be accompanied by asphyxia, especially in children (croup), or be followed by paralysis.

2. Diphtheritic angina is called **malignant** when it is toxic and infective. It is rather toxic than infective when the predominating features can be ascribed to the virulence of the diphtheria toxine; it is rather infective than toxic when the predominating features are due to other microbes, especially the streptococcus. We must never forget the part played by the soil, which favours more or less the development of the poison.

3. The normal, toxic, and infective forms of diphtheritic angina occur with their distinctive characteristics, according as the bacillus limits its action to the production of membranes, or elaborates the poison in every degree of toxicity, with or without secondary infections. We find, however, mixed cases in which these varieties are associated, and it cannot be otherwise, because it is always the same disease, which may present different aspects, but forms, after all, only one species. Diphtheritic angina, therefore, may start with benign symptoms, and then assume the characteristics of the toxic or infective form, with a fatal result.

### **Polymorphism of Diphtheritic Angina—Herpetic, Lacunar, and Pultaceous Varieties.**

Diphtheritic angina is not always characterized by the formation of false membranes. For a long time, until bacteriological examinations were made, writers were loath to admit that membranes might be absent in diphtheritic angina. Non-diphtheritic membranous angina was well known, but few believed that membranes were ever absent in diphtheritic angina.

For some years past bacteriological researches have shed light on the chaos of angina. Thick and adherent membranes are certainly the most common clinical feature in diphtheritic angina, but in a large number of cases—more numerous than we think—membranes are absent. It resembles catarrhal, erythematous, pultaceous, lacunar, or follicular angina. In other cases diphtheritic angina assumes the guise of herpetic angina. These herpetic, erythematous, pultaceous, or lacunar varieties are more frequent in adults than in children. Of 137 cases of diphtheritic angina in adults, Roche found but 42 instances of the membranous variety. I shall review, therefore, these various kinds of diphtheritic angina, which, as will

be seen, is essentially **polymorphous**, and I shall first describe the herpetic variety, as given in my lectures at the Faculté de Médecine in December, 1894.

### 1. Herpetic Variety of Diphtheritic Angina.

I will begin with the classical description of simple herpetic angina. Whether we speak of herpetic angina (Gubler) or of membranous angina (Bretonneau and Trousseau), or whether we follow other authors, and designate it "herpes of the pharynx," it is not less admitted that the characteristic feature of herpetic angina is the presence of herpetic vesicles on the mucous membrane of the throat.

A child or adult is taken ill with symptoms of fever. The rigors and the fever are accompanied by lassitude and headache. Dysphagia appears. We find diffuse redness of the throat; the tonsils are enlarged, and small swellings, which have been likened to sudamina, appear on different parts of the isthmus of the gullet or the pharynx. This condition is known as herpetic angina.

In some cases the disease has the appearance of an erythematous angina, and the exudate is slight. In other cases we find vesicles on the mucous membrane of the throat and also a whitish pultaceous exudate. At times, indeed, we find not simply a pultaceous layer, but thick and extensive fibrinous membranes, simulating diphtheria so closely that Trousseau called this variety of herpetic angina by the name of "common membranous angina," the epithet "common" here eliminating all idea of diphtheria.

Every medical man has seen more or less frequently these erythematous, pultaceous, or membranous forms of herpetic angina. When we are able to witness the actual, but sometimes fugitive, evolution of the herpetic vesicles, no room for doubt exists, and the diagnosis of herpetic angina is clear. The diagnosis often receives considerable support when the herpes, instead of remaining confined to the throat, invades other regions, such as the labial commissure, the lips, the nostrils, the chin, the cheeks, the conjunctiva, etc. If a patient has acute painful angina, with false membranes, which may even simulate diphtheria, and if at the same time herpes is present on the tonsils, the roof of the palate, the pharynx, and the face, we may diagnose herpetic angina, but we cannot exclude diphtheria. This view is at least generally admitted, and is borne out by experience. The description of angina with herpes, therefore, must be revised. Bacteriological research in the last few years has shown, and is showing every day that angina, whether erythematous, pultaceous, or membranous, **cannot be diagnosed for certain, except by bacteriological examination.**

Herpetic angina, like every other variety, cannot escape bacteriological control.



My researches on this subject, communicated to the Académie de Médecine,\* have enabled me to place the matter on a firm basis. I have been able to prove that so-called herpetic angina is fairly often of a diphtheritic nature. I found it easy to verify the inconstancy and the insufficiency of the symptoms furnished by clinical observation, and I cannot state too forcibly the fact that the eruption of herpes, which was formerly said to eliminate the idea of diphtheria, only serves to deceive us. It inspires us with false security, as the following cases show :

CASE I.—In 1895 a man, fifty-two years of age, admitted for severe herpetic angina, which had commenced suddenly four days before with rigors, fever, dysphagia, headache, lassitude, coryza, and slight epistaxis. Axillary temperature, 103° F. Examination of the throat revealed diffuse redness and marked swelling of the tonsils, and on the roof of the palate herpetic vesicles irregularly disposed. A crop of herpetic vesicles was also present on the lips and on the right labial commissure. The maxillary glands were moderately enlarged. The natural diagnosis was **herpetic angina**.

Next day a pultaceous deposit appeared on the right tonsil. This deposit on serum and agar showed staphylococci and **diphtheria bacilli**. The bacteriological finding reversed the former diagnosis. Nasal diphtheria, clinically suspected a few days previously from slight epistaxis and coryza, now appeared. The patient had nasal discharge and epistaxis, and voided false membranes from the nose. The pillars of the fauces and the uvula were also covered with patches of membrane. In spite of injections of serum, which were given too late, acute paralysis of the soft palate appeared a few days later. The paralysis invaded in turn the lower and upper limbs and the bladder.

This case of angina showed all the clinical features of herpetic angina, and it was only by bacteriological examination that we discovered the diphtheria, which was confirmed a few days later by generalized paralysis.

CASE II.—A girl, eighteen years of age, was admitted under Gouguenheim for sore throat, accompanied by headache, lassitude, and temperature of 104° F. Generalized redness of the pharynx and the tonsils. Herpetic vesicles, whitish and slightly adherent membranes on both tonsils ; herpetic vesicles also present on the lips and the commissures. No glandular enlargement. The clinical diagnosis pointed to **herpetic angina**. Cultures made from the membranes on the tonsils yielded diphtheria bacilli, but no other microbes. An injection of 10 c.c. of serum was immediately given.

CASE III.—In 1892, at the Necker Hospital, the matron's son was taken ill with angina. The throat was so red that my house-physicians, Charrier and Renon, took it to be an early symptom of scarlatina. Next day herpetic vesicles appeared on the tonsils, and the diagnosis was **herpetic angina**. At the end of two or three days he was admitted to the hospital, and the existence of the herpetic variety of diphtheria was recognized. I also discovered diphtheritic coryza, which had commenced with epistaxis. Very severe paralysis followed. The velum palati was first affected, and then the muscles of the head and the neck, the lower limbs, and finally the upper limbs, were attacked in turn. He recovered two months later.

CASE IV.—Kelsch was asked to make a bacteriological examination in a case of angina which presented all the clinical characteristics of **herpetic angina**. The culture revealed the diphtheria bacillus, and also the pneumococcus.

CASE V.—Huchard reported the following case to me : He was called upon to give his opinion as to an angina in a child. Relying on the severity of the fever, the sudden-

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\* Dieulafoy, " Angine Diphthérique à Forme Herpétique " (*Académie de Médecine*, séance des 11 Juin, 2 et 3 Juillet, 1895).

ness of the onset, and the simultaneous appearance of herpes, he diagnosed **herpetic angina**, which was confirmed by Brocq. The case, which had every appearance of herpetic angina, was really diphtheritic, and carried off the child in a few days.

**Description.**—We are in possession of several cases which prove that the herpetic forms of diphtheritic angina are fairly common. What, then, becomes of the classical description and clinical diagnosis of the old herpetic angina? The suddenness of its onset, the severity of the fever, the acuteness of the general symptoms, the sharp pain in the throat, and the appearance of the herpetic vesicles on the tonsils, the velum palati, and the lips, formed a symptom-complex, on which it appeared possible to base the diagnosis of herpetic angina. And in the differential diagnosis from diphtheritic angina great care was taken to accentuate the difference between the acute inflammation of herpetic angina and the more insidious and less painful onset of diphtheritic angina.

This erroneous idea must be abandoned. The cases already quoted show that the herpetic variety of diphtheria may be as sudden and acute as the most classical so-called herpetic angina. In Huchard's case the angina began with very acute fever. The tonsils were red and swollen; herpes was present; and a most toxic diphtheria was masked by these acute symptoms.

My little patient in the Necker Hospital was taken ill with acute fever and severe sore throat. Herpes appeared, but here again the acute symptoms masked diphtheria, which was followed by severe paralysis. In a case reported by Roux and Martin, a child was suddenly taken ill, with a temperature of 104° F., pulse of 160, and delirium. The sore throat was severe; the tonsils were much enlarged, red, and pultaceous; herpes appeared on the lips. This picture is typical of herpetic angina, and yet it is quite deceptive, since it applies equally to diphtheritic angina with herpes.

**I therefore propose to abolish the so-called herpetic angina**, as it has been bequeathed to us by our predecessors. It can no longer retain its quasi-intangible place in our nosology. If we retain herpetic angina in the old sense of the word, we must remember that it is less a case of herpetic angina than of angina with herpes.

We see streptococcal, staphylococcal, or pneumococcal angina with herpes, and, of most interest, a group of diphtheritic anginae with herpes,\* as I hope I have clearly proved.

Nevertheless—and this is the question which redoubles the interest—it will be easy for us to connect the actual state of science with the traditions handed down to us by one of our greatest clinical physicians. For

\* Jès (of Krakow) has found the diphtheria bacillus in the liquid of the vesicles of labial herpes in a patient stricken with diphtheritic angina (1896).



this purpose let me quote from Trousseau's report, which was presented by him to the Académie, on behalf of the Commission on Epidemics, on November 22, 1859.

The opinions given in this report are stated some years later in his "Clinical Lectures at the Hôtel-Dieu": "The characteristic of the epidemics of the year 1858," says Trousseau, "was the concomitance of herpes of the pharynx and diphtheritic angina. The former, though reduced to simple herpes of the pharynx, did not always run a regular course. Some cases were very prolonged; at other times the membranous affection degenerated *in situ*. The physician ought anxiously to put the question to himself whether he is justified in maintaining a favourable prognosis. The two affections not only occurred one after the other (herpetic angina and diphtheritic angina), but in each partial epidemic the two pathological forms were found to be more or less closely associated."

I might multiply these quotations, which prove that Trousseau had seen and described in detail the relations which may exist between diphtheria and the so-called herpetic angina. The bacteriological researches which I have already mentioned are a striking confirmation of the ideas of the greatest clinical physician of our French school. Trousseau not only discovered the herpetic variety of diphtheria, but also formed a clear idea of the gravity of the prognosis. We should be wrong to imagine that herpetic diphtheria is always benign. The cases above mentioned are convincing on this point. The child at the Necker Hospital was seized with severe diphtheritic paralysis, which placed its life in danger. Another of my patients similarly suffered from severe paralysis, lasting four months. Huchard's patient died within a few days. Trousseau, then, was accurate when he wrote: "Fatal diphtheria often commences with an eruption of herpes."

**Conclusions.**—Diphtheritic angina may assume the appearance of herpetic angina. It is impossible clinically to affirm that a so-called herpetic angina is or is not diphtheritic. Bacteriological examination enables us to decide the nature of the case.

## 2. Diphtheritic Angina of a Follicular Appearance.

Let us now consider diphtheritic angina that simulates follicular tonsillitis. Here again the clinical picture of the disease is deceptive. Clinically, a case appears to be tonsillitis, and bacteriology corrects the mistake. To quote the proofs:

In 1891 Jacobi showed bacteriologically that follicular tonsillitis, especially in adults, is often diphtheritic. During an epidemic of diphtheria in a boarding-school, Mouillot found amongst eighteen patients eight cases of membranous angina and ten cases of follicular tonsillitis. All were diph-

theritic. One of the latter cases was followed by diphtheritic paralysis. Escherich has found the bacillus of diphtheria in several cases of so-called follicular tonsillitis. Koplik, in 1892, reported numerous cases of follicular diphtheritic angina.

Chaillou and Martin have quoted eight cases of diphtheritic angina, presenting white points on the tonsils, and simulating follicular tonsillitis. Gouguenheim says that in eighty-three cases of adults suffering from diphtheritic angina at the Lariboisière Hospital, he found forty cases of follicular diphtheria.

Diphtheritic angina, therefore, assumes, especially in the adult, the guise of follicular tonsillitis, and bacteriological examination alone can decide the diagnosis. This follicular diphtheritic angina, although generally **benign**, may in exceptional cases be toxic and dangerous, as the cases of Chaillou and Martin show, as well as Case CXII. in Martin's monograph.

### 3. Diphtheritic Angina of the Pultaceous Variety.

This form is the rarest and most benign of all. The patient has angina presenting all the appearances of catarrhal, erythematous, pultaceous angina, and bacteriology reveals the bacillus of diphtheria.

Feer reports three cases. One child showed at first moderate redness of the throat, with a temperature of 103° F. The next day the thermometer registered 105° F. The tonsils were swollen and red, with some pultaceous deposit. Glandular enlargement was present on one side. The child was cured in a few days. The culture revealed the presence of diphtheria bacilli. Inoculation with a pure culture of these diphtheria bacilli killed a guinea-pig in ten hours. Concetti, in 1894, reported two cases of pultaceous diphtheritic angina, arising by contagion from children suffering with fatal pharyngeal and nasal diphtheria. The tonsils were enlarged, and covered with a layer which in nowise resembled diphtheria, but had rather the coarse appearance of a pultaceous exudate. The condition was diphtheritic.

**Clinical Diagnosis.**—In a case of acute angina two mistakes may be made. The first error consists in diagnosing diphtheritic angina when it is not present. The converse mistake consists in mistaking a case of diphtheria for a non-diphtheritic angina. In the latter case we fail to recognize an existing diphtheria. The result is that the patient is not isolated and spreads the disease. Moreover, a patient who has not been treated early with serum will be more exposed to the immediate or remote consequences of diphtheria, including croup, early or late poisoning, and paralysis.

These cases, which are too numerous, prove that it is imperative to recognize diphtheritic angina from the first, under the penalty of the gravest mishaps. Clinical resources, however, are very often insufficient. What signs and symptoms are to be looked for? Can we rely upon the manner in which the angina commences? It has been truly said that diphtheritic angina at its commencement is more insidious and accompanied by less



fever than other forms of angina, which are more clearly inflammatory. If the reader will refer to the cases quoted in the preceding section, he will see that the herpetic form has often a most sudden onset, with marked symptoms of fever.

Can the severity of the dysphagia be relied on? It has been said and repeated—not without some truth—that deglutition is relatively easy in diphtheria, whilst it is, as a rule, very painful in non-diphtheritic angina. This statement is often true, but yet we find many exceptions. The cases of herpetic diphtheria enumerated above showed early and severe dysphagia.

Can the character and the growth of the false membrane be relied on? Here again the clinical characteristics of the diphtheritic membranes which were classical before bacteriological examination—viz., colour, thickness, adherence, elasticity, and reaction to chemical agents—are common both to diphtheritic and non-diphtheritic membranes. Is not the ready reproduction of false membranes at least a characteristic inherent in diphtheria? It was formerly believed to be so, but since bacteriological examinations have been made, we know that membranes caused by *Brisou's* cocci, streptococci, staphylococci, or pneumococci, may be reproduced with the same readiness as the diphtheritic membrane.

Is not the tendency which the membranes possess of invading the nasal cavities and the larynx at least in favour of diphtheria? In the section on Pseudo-diphtheritic Membranes we shall see that many of them, though due to streptococci or to *Brisou's* coccus, may invade the nose and the larynx. Cannot the glandular enlargement, which has been looked upon as a valuable sign, be of some help to us? On the one hand, enlargement of the submaxillary glands may be almost wanting in pure diphtheria, and very marked in pseudo-diphtheritic angina. *Baginski* has reported submaxillary adenitis in five out of six cases of streptococcal angina. *Tezénas* speaks of similar swelling four times in four cases of streptococcal and staphylococcal angina. *Jaccoud* found adenitis in a case of pneumococcal angina. *Martin* has repeatedly noted marked adenitis in membranous angina due to *Brisou's* coccus. I have often made the same observation: Submaxillary adenitis is, therefore, of little assistance in diagnosis.

Is albuminuria a sign of diphtheritic angina? Pseudo-diphtheritic angina, caused by various microbes—notably streptococci—is frequently accompanied by albuminuria.

This critical study of the signs and symptoms of diphtheritic angina shows that clinical signs alone are insufficient for diagnosis. Several cases regarded by eminent physicians as diphtheria have been proved by bacteriology to be not so. I need only refer to pseudo-diphtheritic angina due to the streptococci, *Brisou's* coccus, the pneumococcus, or the staphylococci, which we

shall discuss in the next section. *Per contra*, several cases considered as being non-diphtheritic have been proved by bacteriology to be diphtheria. I need only refer to the numerous cases of **polymorphous** diphtheria (herpetic, lacunar, and pultaceous forms).

I am well aware that even at the present day many practitioners make their diagnosis in the case of angina solely by the clinical signs, and reserve bacteriological examination for doubtful cases. Bacteriological examination, carried out **systematically**, is, in their opinion, unnecessary. After my communication to the Académie de Médecine, I was taken to task in the *Premier-Paris* of one of our medical journals, where I read the following sentence: "According to Dieulafoy and all bacteriologists, it is no longer permissible to make a diagnosis of angina without bacteriological examination. Is there not some exaggeration in this proposition?" That is the very word: **we are taxed with exaggeration**. We are given to understand that bacteriological examination is only useful in a case where clinical examination may be at fault. I cannot too strongly contradict such assertions.

We are told that great clinical physicians in the past did not await the aid of bacteriology in order to diagnose cases of angina. I apologize to those who speak thus, but we must speak the whole truth. No one respects tradition more than I do, and I place great reliance on clinical investigation, but evidence must be accepted. I would refer my critics to the diagnosis made in his own case by Gillette, physician to the Children's Hospital, and one well versed in the diagnosis of diphtheria. Gillette thought he was suffering from herpetic angina. He congratulated himself on the severity of the inflammatory symptoms, the whiteness of the membranes, the bright redness of his throat, and the pain which he felt; and yet the angina was diphtheritic, and proved fatal in a few days. I would ask my critics what they think of the diagnosis of Gubler, who was a man well versed in the study of angina. He taught that herpetic angina causes general paralysis, as diphtheria does, thus committing an error which he would never have made if bacteriology had enlightened his diagnosis.

I would refer my critics to the classification of Lasègue, who did not know exactly where the group of diphtheritic anginae began or ended, and invented the name **diphtheroid angina**. This name created the greatest confusion, which only bacteriology has been able to dissipate.

In the subject under discussion clinical observation must give way to bacteriology. I know that it is hard to give up deeply-rooted convictions, but, at the risk of repetition, evidence must be accepted. A knowledge of the bacteriological work of the last few years shows the innumerable errors committed when the diagnosis of angina rested on clinical observation alone. In order to convince my readers, I think it useful to point out the errors corrected by bacteriology.



In the third report of the Pasteur Institute, Roux and Yersin state that out of 52 cases of membranous angina of diphtheritic appearance 19 were not diphtheritic. Morel, in his thesis on diphtheria, states that in 86 cases of membranous angina simulating diphtheria 20 were not diphtheritic.

The most important paper published in France on this subject is that of Martin, who says: "Of 112 patients admitted to the Hospital for Sick Children for diphtheritic angina 36 had not diphtheria at all." He takes care to add: "Clinical study gave no information as to the nature of these 36 cases. Physicians had taken them for diphtheria, and yet bacteriological examination proved the absence of the diphtheria bacillus. They gave rise, therefore, to 36 diagnostic errors which it was clinically impossible to avoid, and which, consequently, exposed to contagion 36 children who were in a state of receptivity."

Baginsky, in 1891, published a series of 93 cases of membranous angina of diphtheritic appearance; bacteriological examination proved that 25 of these cases were not diphtheria. In 1892 Baginsky published a second series of 154 cases of angina of diphtheritic appearance, in which bacteriology revealed 36 errors in diagnosis. William Hallock Park says: "In 159 cases of membranous angina of diphtheritic appearance, 89 cases—that is to say, more than half—were not diphtheritic."

In 1892 Koplik reported 33 cases of membranous angina of diphtheritic appearance; Löffler's bacillus could not be found in 16 of them.

Errors are more numerous in the case of secondary anginae supervening in the course of the infectious fevers, and notably in scarlatina.

We have, therefore, a series of cases in which hundreds of errors have been made, because certain microbes—namely, Brisou's little coccus, streptococcus, staphylococcus, etc.—gave to these cases of angina the clinical appearance of diphtheria.

Another series of errors consists, as we have said, in mistaking diphtheria for simple, lacunar, pultaceous, or herpetic angina. As this question has been fully discussed in the previous section, I shall not reopen the discussion. This **polymorphism** was a very frequent cause of error before bacteriology established the facts. Practitioners were too much accustomed to the idea of membranous diphtheria. We know to-day that herpetic, lacunar, and pultaceous forms of diphtheria exist. We must therefore eliminate a partially correct diagnosis. In the case of angina the only way to arrive at an absolute diagnosis is by bacteriological examination. Jaccoud expressed this opinion in 1891, and Landouzy made an important communication to the Académie de Médecine on the same subject in 1895.

The above discussion applies equally to syphilitic angina. **Chancre of**

**the tonsil**, which is covered with greyish false membranes and accompanied by dysphagia and glandular enlargement, somewhat resembles diphtheria; but in the case of the chancre the lesion is unilateral, the tonsil is **indurated**, the disease runs a slow course, the ulcerated surface is readily cleaned, and the detritus is pultaceous rather than membranous. **Mucous patches** of the throat and of the tonsils are sometimes covered with false membranes, which simulate diphtheria. These membranes are whitish or greyish, of a gangrenous appearance, adherent to the mucous membrane, and accompanied by submaxillary adenitis. These forms of **syphilitic** angina closely resemble diphtheria. These two lesions may, indeed, appear simultaneously, and are so much alike that the diagnosis can only be made by bacteriological examination.

**Ultero-membranous** angina, described in Section 2, is accompanied by diphtheroid membranes, and may simulate diphtheria. The absence of diphtheria bacilli and the presence of fusiform bacilli and spirilla (Vincent) prove the diagnosis.

**Phlegmonous tonsillitis** itself may present difficulties in diagnosis from certain forms of diphtheritic angina. In both cases the pain is very severe, deglutition is difficult, the swelling of the neck may be considerable, and the pulpy, sanious, diffuent coating sometimes present in malignant angina closely resembles the diphthero-streptococcal coating which covers the throat in phlegmonous tonsillitis. Albuminuria may exist in either case. On what symptoms can the clinical diagnosis be based? In phlegmonous amygdalitis the pain is more acute and more general, the dysphagia is excessive, and the patient can scarcely turn his head, which is rendered immovable by the contraction of the muscles of the neck; he cannot open his mouth without acute pain, and can scarcely move his tongue. These symptoms are not so severe in diphtheria. In both cases the neck is puffy and swollen, but the swelling appears **earlier** and is more marked in the **glands** than in diphtheria. In both cases respiration may be interfered with on account of the contraction of the isthmus of the gullet, but this symptom is much more prominent in phlegmonous tonsillitis, and gave to it the name of quincy. In both cases examination of the throat may be difficult, but the patient suffering from diphtheria is the better subject, because **he feels less pain**. One essential feature is absent in diphtheria—viz., the tonsil and the velum palati are not pushed back and depressed by the peritonsillar phlegmon. Certain cases of suppurative tonsillitis, however, may be associated with the diphtheria bacillus and other microbes. I saw a case of diphthero-streptococcal angina which simulated suppurative tonsillitis. Bacteriological examination is therefore indispensable.

**Bacteriological Examination.**—In a case of angina, what is the correct technique in a bacteriological examination? A piece of membrane may be



stained and examined under the microscope ; this method, however, is not trustworthy, and I therefore prefer to make cultures. A small piece of membrane is placed in a tube of gelatinized serum, as described in the preceding section.

**Diphtheritic Angina.**—In the case of diphtheria, either pure or associated with other microbes, it is possible, after an incubation of eighteen hours, to make out diphtheria colonies, which are the more characteristic the more spaced they are. The rapidity with which the first colonies appear is almost pathognomonic of diphtheria ; it is only in membranous angina due to Brisou's coccus that we find equally rapid growth. Typical colonies of the diphtheria bacilli are rounded, whitish, and more opaque at the centre than at the periphery. They project slightly above the surface of the serum, and I have therefore called them **papular**. A fragment stained with Roux's blue shows the Klebs-Löffler bacillus (Section V.).

**Membranous Angina due to Brisou's Coccus.**—A patient is suffering from membranous angina which might easily be considered diphtheritic ; it has all the characteristics of a case of normal diphtheritic angina. I have seen such a case at the Necker Hospital, and the resemblance to diphtheria was such that bacteriology **alone** could decide the diagnosis. On making a culture, after eighteen hours colonies appear in the gelatinized serum ; they closely simulate diphtheria colonies. They appear early ; they are rounded and whitish, but their centre is not opaque. They are transparent throughout their whole extent, and have a humid appearance ; moreover, they are **flat**, and I have therefore called them **macular**, to distinguish them from the diphtheria colonies which are **papular**. On examining a stained fragment under the microscope we find no diphtheria bacilli, but only a small coccus, which is often paired like a diplococcus. We know, therefore, the nature of the angina which at first sight so closely **simulates** diphtheria. We know that it is neither toxic nor infective, that it will not be followed by paralysis, and that even if croup is present, tracheotomy will not be required.

**Membranous Angina due to Streptococci.**—These cases simulate diphtheria so exactly that in Martin's report we find an account of eight patients who were sent to the **diphtheria ward** when they had streptococcal angina. This angina will be studied in the next section. I will here content myself with giving the distinctive bacteriological signs. When membrane from the pharynx is placed on the culture medium, the colonies of streptococci make their appearance somewhat later than those of diphtheria. We find numerous small **punctiform** colonies, which I call **powdery** ; they show but little tendency to grow larger, and the microscope reveals the streptococcus in straight or bent chains of three, four, five, or six elements. Primary or secondary streptococcal angina is frequent in the early stages of scarlatina,

whilst the angina which appears during the decline of scarlatina may be diphtheritic.

**Membranous Angina due to Staphylococci.**—Membranous angina due to staphylococci has been taken for diphtheria. The colonies (*Staphylococcus albus* and *aureus*) usually develop in twenty-four hours, and the bacteriological examination is so characteristic that a mistake is impossible.

**Membranous Angina due to Pneumococcus.**—This variety of angina (see Section VII.) has been described by Jaccoud. The diagnosis from diphtheritic angina is absolutely impossible without bacteriological examination. This variety of angina, which is due to the pneumococcus, is not toxic, and does not spread to the larynx.

**Prognosis.**—The prognosis of diphtheritic angina depends upon the clinical and bacteriological examination. Clinically, we must beware of cases of angina preceded or accompanied by **nasal diphtheria**. The nasal cavities are an excellent soil for the growth of the diphtheria bacillus; it elaborates its toxine under the most favourable conditions, for it finds an even temperature and an incessant renewal of air by nasal respiration, just as in the method devised by Roux for the manufacture of the toxine. In mild diphtheria, early and grave complications need not be feared, but yet the poison is made on such a surface that sufficient toxine to cause diphtheritic paralysis may penetrate it. In malignant diphtheritic angina the presence of nasal diphtheria is of evil omen, as Trousseau rightly insisted. Early pallor, leaden and puffy face, abundant albuminuria, and tendency to prostration are bad symptoms. When the submaxillary glands are much enlarged from the first, the prognosis is usually grave. In some cases the inflamed glands behave like buboes—"they reek of the pest"—and form abscesses. The glandular suppuration may not be discovered, because the patient succumbs before the formation of an abscess. **Early suppuration is a fatal sign**: this statement does not hold in late suppuration. If the adenitis commences to suppurate when the angina is at an end, recovery may follow. These cases of adenitis are due to the association of the streptococcus with the diphtheria bacillus.

**Bacteriological examination** furnishes valuable information as to the prognosis. A culture which shows discrete colonies of medium bacilli indicates less grave diphtheria than if the culture produce confluent colonies with long curved bacilli. As regards the prognosis, it is very important to know whether the bacillus of diphtheria is associated with other microbes. **Bacteriological examination is, as we shall see, an indispensable element in the prognosis.** Angina in which the bacillus of diphtheria is present alone is generally normal, with firm and elastic fibrinous membranes. It may be followed by croup, especially in children, and by paralysis, but it rarely assumes the malignant form. Diphtheritic angina which is associated with



Brisou's coccus, is generally benign. Diphtheritic angina with which the staphylococcus is associated, is far more serious than the preceding forms.

In the most serious or malignant form the **streptococcus** is also present. It is in such cases that the membranes may have a putrilaginous aspect and foetid odour, while the enlarged glands lead to the condition known as "the proconsular neck." Malignant angina is fairly frequently met with as a secondary affection in the decline of scarlatina or during the course of measles and whooping-cough.

**Ætiology.**—Diphtheritic angina is especially common in young subjects. It is **endemic** in certain countries, and when it rages in an **epidemic** form, the epidemic is often relatively benign or malign in a family, a town, or a district. In countries visited for the first time by diphtheria the angina and the other manifestations of the disease are generally severe. For example, in Bessarabia, where the scourge made its appearance for the first time in 1872, it carried off more than 12,000 victims in eight years. "In 1875 it appeared in the Province of Kerson, where the mortality varied from 27 to 62 per cent."

Diphtheria is contagious, and the contagion may be direct or indirect. Inoculation of diphtheria, tried by Trousseau and Peter upon themselves, did not succeed; it was a lucky failure, which only proves that the subjects were not in a state of receptivity. Direct contagion is only too well proved by the numerous examples of parents and physicians who have contracted the disease from a patient. I need only cite the case of Walleix, already mentioned, and of Herpin, who from a piece of membrane which entered his nostril, contracted coryza, angina, and paralysis. The cases of Blanche, Clozel de Boyer, Armango, and many others, whose memory we revere, will suffice.

The contamination may be contracted from patients who have recovered from the attack because virulent bacilli are present for weeks in their oral and nasal cavities. After the disappearance of the membranes, virulent bacilli may exist for a fairly long time, although the mucous membrane appears quite healthy (Roux). Tezéas, who studied the duration of the contagious period in convalescent patients, furnishes us with the following information: He made cultivations daily in sixty cases after the disappearance of the membrane from the throat. In five cases bacilli were present for a variable period. In eleven cases the bacillus was present in the nose, while it was absent from the mouth and pharynx. It was found in the nose for fifty-five days, and while it was present, it gave rise to a clear nasal discharge, that was generally unilateral. Centres of contagion may thus result.

In other cases the contagion is indirect, and spreads through the intermediary of the membranes, or of the dried sputum which has fallen on bedding, clothing, toys, or toilet articles. Cases of diphtheria occurring six months or

a year later in previously infected surroundings are thus explained. These clinical cases agree with experimental researches, Roux having found that a particle of diphtheritic membrane, wrapped in a piece of linen and placed in a wardrobe, kept its virulence for more than five months.

In other cases the diphtheria bacillus exists in the naso-pharyngeal cavity without causing the slightest complication (Löffler). If, however, the virulence of the bacillus is increased, diphtheria occurs, and appears to be spontaneous.

A first attack does not confer immunity; diphtheritic angina may occur a second time.

In some cases the angina is **secondary**—that is to say, it supervenes in the course of some other disease (scarlatina, measles, typhoid fever). These cases will be described under the primary disease.

Do fowls suffer from diphtheria, and can they transmit it to human beings? Saint-Yves Ménard denies, with good reason, the identity of human and avian diphtheria. The latter is a pseudo-diphtheria which is not transmissible to man.

**Pathological Anatomy.**—I described under Diphtheritic Angina the appearance of the false membranes. These fibrinous membranes adhere more to the chorion when the mucous membrane is covered with stratified epithelium than when it is covered with simple epithelium, as in the air-passages. The membranes may acquire great thickness ( $\frac{1}{2}$  millimetre to 2 millimetres) from the **stratified** layers which are formed on the deep surface in contact with the mucous membrane. The younger the layers the more resistant they are, whilst the older ones are pushed towards the surface and become friable. Each layer of the false membrane is developed at the expense of the corresponding layer of the epithelium, and becomes more superficial as a new layer is produced beneath. “A discussion has been raised as to whether the false membrane is situated above or below the epithelium. From what has gone before, we see that it is formed in the epithelial lining, and partially at the expense thereof.” The false membrane replaces the epithelium. On post-mortem examination the diphtheritic membranes have in part disappeared, but examination during life shows that they are composed of a more or less dense network of fibrin, that encloses in its meshes altered epithelial cells, lymphocytes, red corpuscles, and micro-organisms. Many of the cells are dead, and their nuclei do not stain with picro-carmin.

The epithelial cells are infiltrated with colloid substance, lose their nucleus, and are converted into homogeneous refracting blocks with prolongations which branch like a stag's horns (Wagner)—Weigert's coagulation necrosis. A hæmorrhagic exudate is sometimes found under the false membranes, and gives rise to ecchymoses. The structure of the false membrane changes somewhat at different periods of its growth. At first



the epithelial changes are most marked and the fibrinous network is less important. Later the membrane becomes epithelial, fibrinous, and purulent, and in the last stage the fibrin is in excess (Leloir). The behaviour of the diphtheria bacilli was described in the previous section.

The mucosa on which the membranes are about to develop is inflamed and swollen. After the membranes have disappeared, the mucosa has a dull look from the absence of epithelium. Ecchymoses may be seen, but ulcers are rare. Ulceration, hæmorrhage, and gangrene are chiefly seen in malignant diphtheria.

When attacked by diphtheria, the **tonsil** undergoes changes, the description of which I borrow from Cornil. In a section we see the following features from the surface to the deep tissue: The false membrane which has replaced the epithelium burrows into the tonsillar crypts. In its deepest part it seems to blend with the mucous chorion. The connective tissue of the mucous membrane is infiltrated with red and white corpuscles, its capillary vessels are filled with white corpuscles, and the inflammation which affects the reticular tissue and the follicles of the tonsils explains the marked swelling of these organs. The **pharynx** is the seat of similar lesions; inflammatory hypertrophy of the lymphatic follicles is seen. The **lymphatic glands** of the neck are swollen and infiltrated with a turbid, serous, or purulent fluid.

The membranes on the **skin** closely resemble those on the mucous membrane. They are in part formed at the expense of the modified epidermal layers, and are adherent to the papillæ. Gangrene of the derma is sometimes found. Ecchymoses have been found in the sulci of the **cerebral convolutions**. **Pulmonary** lesions (bronchitis, broncho-pneumonia) are very common, especially when croup complicates angina. They are generally the result of secondary infections, the staphylococcus and streptococcus being responsible in many instances. The **kidneys** are almost always affected in severe diphtheria. We find hyperæmia and hæmorrhage in the cortical layer, with cloudy swelling of the epithelium of the tubules. The changes in the kidneys, like those in the liver (fatty degeneration of the cells), are due to the diphtheritic toxine.

The heart muscle is affected, and the papillary muscles in particular show granular degeneration. Interstitial myocarditis is at times fairly well marked. The valvular endocardium shows changes described by Labadie-Lagrave as endocarditis. They are rare, and result from secondary infections. In malignant angina the blood is fluid and sepia-coloured (Millard), and the number of red corpuscles is diminished. This condition of dissolution is met with in certain infectious diseases.

**Treatment.**—Since Roux's communication at the Buda-Pesth Congress, the treatment of diphtheria by serotherapy has replaced other methods.

Behring first thought of applying serotherapy to the treatment of diphtheria, but it was Roux who, by discovering the toxine, rendered the discovery of the antitoxine possible. Roux selected the horse as the animal to produce the serum, and, while the labours of Behring gained few adherents in Germany, Roux's reports and results of his labours and those of his collaborators were sufficient to insure universal employment of the new method.

The method consists in making horses immune against diphtheria, and in making use of the serum from an immune horse as a preventive and curative agent in diphtheria.

The animal is inoculated under the skin of the neck and shoulders with progressively increasing doses of diphtheritic toxine. We start with very weak doses—less than 1 c.c., with or without the addition of iodine—and in a few weeks it is possible to inject at one sitting doses which are 200 and 300 times as large. In less than three months the horse is immune. As suggested by M. Nocard, some 10 pints of blood are taken from the jugular vein, yielding about 5 pints of serum. The same horse, if he continues immune, can supply the above quantity of antidiphtheritic serum every three weeks. Antidiphtheritic serum does not quite deserve the name antitoxine. The serum is not antitoxic in the true sense of the word, as it does not destroy the toxine. It does not affect the toxine, but acts on the cells of the organism, making them, for the time being, insensible to the poison. The toxine destroys the activity of the cell, while the antitoxine revives and stimulates it (Roux). The serum restores to the cells of the organism a part of the phagocytic activity which they have lost through the action of the poison (Metchnikoff).

**Experiments.**—The following method is used in studying the action of the serum on infected animals :

Vulvar diphtheria is induced in a female guinea-pig. A few hours after inoculation we find, first, redness of the vulva, with swelling and œdema of the mucous membrane, and later the appearance of diphtheritic membranes, vaginal discharge, fever, and loss of appetite. Some animals die in a few days from diphtheritic poisoning, some recover, and others suffer from paralysis.

If, however, these animals receive a prophylactic dose of serum equal to one ten-thousandth part of their weight, the diphtheritic membranes disappear by the second day; fever is less severe, and recovery always follows.

If, instead of giving the antitoxine as a prophylactic, we inject it twelve hours after inoculation with diphtheria, curative results are obtained. The diphtheritic membranes disappear on the second day, and do not reform; the bacilli disappear, and the animals recover. In any case, the curative dose of antitoxine used (after inoculation of diphtheria) must be very much larger than the prophylactic dose.



Curative experiments give similar results in croup. If croup is produced in rabbits, these animals die in a few days, the respiratory troubles and laryngo-tracheal lesions resembling those found in croup in children.

Rabbits which have been inoculated in the trachea after injection of serum, "do not take diphtheria, or, at least, it does not show itself by any apparent malaise." If the serum is injected into animals after tracheal inoculation, it may arrest well-marked diphtheria.

These experiments, then, are very conclusive, and prove the efficacy of antitoxic serum, provided the injection is not given too late.

The serum, however, does not produce the same benefit in diphtheria associated with the streptococcus. "The association of the two microbes (diphtheria bacillus and streptococcus) produces in the rabbit rapid diphtheria, such as we see in young children. The pathological picture is the same." These two microbes reciprocally increase their virulence, and injections of antidiphtheritic serum have not the same efficacy.

Let us now consider the treatment in a case of diphtheria. A sterilized syringe of a capacity of 20 c.c. is used, and the serum is injected under the subcutaneous tissue of the flank, the skin being first rendered aseptic. To a child under fifteen years of age 10 to 15 c.c. of serum are given; above fifteen years of age 20 to 25 c.c. are injected at one sitting. In some cases it is necessary to repeat the injection on one or more occasions. At present, there is a tendency to multiply the injections from the first, so as to avoid having recourse to the serum during the anaphylactic period.

Let us analyze the results—(1) in pure diphtheria; (2) in diphtheria with other micro-organisms.

**Pure Diphtheritic Angina.**—Recovery is the rule after the serum has been injected. Fresh membranes do not appear twenty-four hours after the injection, and those already present become detached in two days. The temperature falls abruptly (Martin) and the general condition speedily improves. Furthermore (this point is very important), complications and croup are very rare, or, at least, if croup appears, it is very mild. The following quotation deserves consideration: "In 169 children admitted into hospital with diphtheritic angina, 36 showed laryngeal troubles, 31 had croupy cough, and 25 had lost their voice and showed marked sucking-in, so that tracheotomy appeared advisable. Under the influence of the serum (and an injection may be given every twelve hours) the sucking-in diminished, and only returned at intervals. The child coughed up the false membranes, and at the end of two or three days the respiration was normal, to the great surprise of the house-physicians and the nurses, who, from their experience of croup, were convinced that operation could not be avoided.

Let us now see the action of serum in a case of diphtheritic angina when other microbes are present.

The association of the diphtheria bacillus with Brisou's coccus is benign, and recovery is the rule after injections of serum.

The association of the bacillus with the staphylococcus causes more severe angina; nevertheless, recovery almost always follows after injections of serum, and this association is not as serious in the case of angina as it is in the case of croup after tracheotomy.

The presence of the streptococcus gives rise to grave angina, in which injections of serum are not so effectual. The mortality has been 25 per cent.

In diphthero-streptococcal angina, antidiphtheritic serum and Marmorek's antistreptococcic serum have been used together, but the results so far obtained with the latter serum have not been encouraging: "There is no reason to expect a positive action. We can only look for a modification of the condition of the throat and of the glands; we cannot rely on any anti-toxic action" (Sevestre).

I need not refer here to the treatment of croup by injections of serum (*vide* Croup), but, taking all the cases of diphtheria (angina or croup) as a whole treated with antitoxic serum, we obtain the following figures:

The statistics of Roux, Martin, and Chaillou refer to 446 cases. Death-rate, 24·5 per cent.

The statistics of Moizard refer to 231 cases. Death-rate, 14·7 per cent.

The statistics of Le Gendre refer to 16 cases. Death-rate, 12·5 per cent.

The statistics of Lebreton refer to 242 children. Death-rate, 12 per cent.

The statistics of Variot for the year 1895 give a total death-rate of 14·5 per cent.

"The year 1894," says Bayeux, "divides the therapeutics of diphtheria into two distinct epochs: the first, in which 55 per cent. of the cases died, and the second, when the mortality fell to 16 per cent., thanks to the use of **antitoxine**. This figure (16 per cent.) is supported by my own returns of more than 200,000 cases."

The rate of mortality is diminishing continually, and will diminish still further, in proportion as cases of broncho-pneumonia are isolated, and tracheotomy is replaced, as far as possible, by intubation, and serum is injected without a moment's loss of time. We have lived to see a thing hitherto unknown—that is, a week passing in Paris without a single case of diphtheria being notified!

A condition of success is to make the injection as soon as possible after the outbreak of the disease. Diphtheritic paralysis only supervenes, as a rule, when injection of serum has been delayed. I hold, therefore, that in a suspicious case of membranous angina, before bacteriological examination has decided the nature of the disease, we should begin by giving an injection.



No one will ever repent having done so, even though the angina is not diphtheritic, whilst delay till the next day may cause regret.

As for **local** treatment in diphtheria, we must be satisfied with irrigations containing chlorinated soda (Roux). Painting with poisonous or caustic substances, such as carbolic acid and sublimate, must not be prescribed.

I asked myself whether local painting of the throat with serum might not be beneficial. I therefore conducted some experiments with my house-physician, Marion. Female guinea-pigs were inoculated with vulvar diphtheria, and, when the disease appeared, the parts were painted several times a day with serum. No result was obtained. This conclusion, however, refers only to ordinary serum obtained by the injection of toxine into animals. Martin, by injecting the bodies of the bacilli themselves into animals, obtained a serum which was no longer antitoxic, but anti-infective. Rist has shown that the bodies contain a poison differing from the soluble toxine. Martin's anti-infective serum can be made up in pastilles, which dissolve in the mouth and seem to have an action *in situ* on the diphtheria bacillus.

Injections of antidiphtheritic serum sometimes cause cutaneous eruptions, urticaria, and articular pains, which are less frequent in proportion as the technique has been perfected. Other complications for which the serum has been blamed (albuminuria, tachycardia, arrhythmia, muscular pains) must be set down to diphtheria or to streptococcal infections. It is important however, to make use of properly prepared serum; bad serum might lead to trouble.

**Prophylactic Treatment.**—As serum injections have a **prophylactic** action, it may be beneficial to inoculate the contacts, especially children and nurses. This prophylactic application of serum has given most satisfactory results (Mewim, Schüler), and is a means of stamping out epidemics or of preventing their spread.

Netter and Guinon have made systematic use of this prophylactic power. Every child admitted into the Trousseau Hospital receives 5 c.c. of antidiphtheritic serum, subject to reinoculation every three weeks. Internal cases of diphtheria in the wards have become unknown.

Patients must be closely watched, even after complete disappearance of the membranes, because the bacilli may remain for weeks in the throat or nose. Tezéas has published a very interesting work on this subject. In sixty cases of diphtheria he found diphtheria bacilli in the nasal cavity, although the angina was completely cured and the bucco-pharyngeal cavity was free from them. This persistence of the bacillus in the nasal cavity is always associated with a limpid nasal discharge, which generally comes from one nostril. "As long as this discharge lasts, Löffler's bacilli are found in the nasal cavity. The bacillus disappears with the discharge."

The most minute precautions must be taken with regard to things that have been in contact with a diphtheritic patient. Linen or bedding must be baked in a steam-oven under pressure, for the diphtheria bacillus is very resistant, and persons have contracted diphtheria in a bed which a patient suffering from diphtheria had used several months, or even a year, previously, no antiseptic precautions having been taken.

## VII. PSEUDO-DIPHTHERITIC MEMBRANOUS ANGINA.

Before the discovery of bacteriology it was well known that certain cases of membranous angina **simulated** diphtheria, though they were not diphtheritic. Bretonneau had expressed this view, and Trousseau wrote a most remarkable chapter on this subject. Returning to the familiar question of specificity, Trousseau reviews the forms of membranous angina which are not diphtheritic: membranous angina following cauterization of the pharynx with nitrate of silver and ammonia, and membranous angina due to the abuse of mercury. He teaches us that in scarlatina membranous angina is rarely diphtheritic (and bacteriology has proved that he is correct), and quotes cases of angina in the course of enteric fever which have been wrongly taken for diphtheria. Finally, following Bretonneau, he separates diphtheria from herpetic membranous angina, which he calls **common** membranous angina.

The distinction made by these great masters, as a result of clinical observation, between diphtheritic and pseudo-diphtheritic membranous angina, has been clearly established by bacteriological researches. Bacteriology is responsible for this section, which is only the continuation and the complement of the two preceding sections. Bacteriology has enabled us to classify and to enumerate the nature and the characteristics of pseudo-diphtheritic membranous angina.

The different microbes which will be referred to in the description of pseudo-diphtheritic angina, include Brisou's coccus, the streptococcus, the pneumococcus, the staphylococcus, and the *Bacillus coli*; they may be associated in all the varieties of angina, whether catarrhal, pultaceous, herpetic, or suppurative. In some cases, however, they are associated with the formation of membranes, and the angina then simulates diphtheria, and merits the name "pseudo-diphtheritic."

**Pseudo-Diphtheritic Angina due to Coccus.**—A child four or five years of age has, two days before, been taken ill with moderate fever, headache, loss of appetite, and sore throat. The temperature has been about 103° F. On examining the throat the mucous membrane is found to be red and covered with patches of membranous exudate. If the angina be of two or three days' duration, the tonsils, the uvula, and the pharynx may be covered with false membranes which simulate diphtheria. No distinction is possible between these membranes and those seen in certain cases of diphtheritic angina, as they are similar in appearance and structure. In each case they show the same power of adhesion, mode of invasion, and reproduction after removal. In addition, glandular enlargement is often found, though in a moderate degree.

These signs and symptoms, therefore, are those of normal diphtheria. They were complete in a case at the Necker Hospital. My diagnosis of normal



diphtheria, which seemed obvious, was proved to be incorrect by bacteriological examination.

This variety of angina which simulates diphtheria so closely is due to Brisou's coccus, and was thus named from the child in whom Roux and Martin first observed this kind of angina.

As it is absolutely impossible to make a diagnosis between this false and true diphtheritic angina by clinical observation alone, bacteriological examination is necessary. For this purpose we make a culture. A particle of the membrane is removed on a platinum loop and sown on a tube of gelatinized serum, which is placed in the oven at a temperature of  $36^{\circ}$  to  $37^{\circ}$  C. Colonies appear on the surface of the serum after about eighteen hours; they have, when well developed, the greatest analogy to those of diphtheria. In the first place they appear early—in fact, almost as early as those of diphtheria; they are also rounded and whitish. They differ, however, in some points. Their centre is not opaque; they are transparent throughout their whole extent, and present a humid appearance. In addition, they are **flat**, for which reason I have named them **macular**, in order to distinguish them from the diphtheritic colonies which are **papular**.

A stained fragment of the culture under the microscope shows no diphtheria bacilli, but only Brisou's coccus, the elements of which are often paired. We know, therefore, the nature of this angina, which at first sight closely simulates diphtheria, and we may be satisfied as to the prognosis, because angina due to Brisou's coccus does not give rise to toxæmia and is not followed by paralysis. In some cases it has been followed by croup, but in a very mild form.

This pseudo-diphtheritic angina most often simulates diphtheria. It is liable to recur. It has been met with three times by Roux and Yersin. Martin's returns give twenty-five times in 200 cases of membranous angina; Chaillou and Martin's figures give eleven times in ninety cases.

**Streptococcal Pseudo-Diphtheritic Angina.**—The streptococcus may be found in every variety of angina. We are here concerned with membranous angina simulating diphtheria, for in the throat, as elsewhere, the streptococcus readily produces membranes (Widal).

This angina, though generally benign, may be in some cases of exceptional gravity. The disease sets in with rigors, fever, headache, and lassitude. The dysphagia is acute, and, on inspection of the throat, the mucous membrane is found to be red and inflamed, and the tonsils are sometimes enlarged. In some cases the exudate is pultaceous, but in others we find thick adherent membranes on the tonsils, the posterior wall of the pharynx and the velum palati; they may even extend to the tongue and the lips. It has been rightly said that the streptococcal membranes are less elastic and

more friable, yellowish, and oedematous than the diphtheritic membranes, which are whiter and more nacreous, as well as more firm and dry. These differences are more easy of description than recognition. As a matter of fact, some streptococcal membranes so closely resemble diphtheritic membranes that we cannot tell the difference. Inflammation of the submaxillary glands is said to appear earlier in streptococcal than in diphtheritic angina. Here again there are constant exceptions.

As a clinical resemblance between streptococcal pseudo-diphtheria and true diphtheria, let me add that we often find rhinitis and laryngitis in both cases. The nasal discharge in each disease may be mucous, blood-stained, or puriform, with rejection of membranous shreds. In both cases the cough and the hoarseness indicate the invasion of the larynx by the streptococcus or by the diphtheria bacillus.

In some cases streptococcal angina brings about such grave infection that erythema, polymorphous eruptions, albuminuria, and rheumatic pains may be noticed, just as in diphtheria, and such a grave general condition that death may result. Streptococcal angina is often secondary (grippe, measles, typhoid fever); it is most common at the commencement of scarlet fever. These secondary forms may be grave. We see, however, primary cases which prove fatal.

The above description shows that the clinical diagnosis between this pseudo-diphtheria and true diphtheria is quite impossible. Streptococcal angina simulates diphtheria so closely, that in Martin's monograph we read that eight patients who were sent to the diphtheria ward were really suffering from streptococcal angina. Chaillou and Martin mention eight cases of streptococcal angina simulating diphtheria.

I had quite recently at the Necker Hospital a case of streptococcal membranous angina which simulated diphtheria so closely that a diagnosis would have been impossible without bacteriological examination. This examination is carried out in the manner already described. The streptococcus colonies appear rather later than those of diphtheria and of *Brisou's* coccus. After some twenty-four hours they appear in the form of a whitish stippling, which never shows much increase in size. I have, therefore, thought it advisable to call these colonies dusty, and thus distinguish them at first sight from the macular colonies of *Brisou's* coccus and the papular colonies of the diphtheria bacillus. Even after fourteen to fifteen hours, stained fragments of the colonies on the surface of the serum show under the microscope little chains of mature streptococci. The cocci are arranged in chaplets, and in straight or wavy chains. Four or more cocci may be seen in each fragment of the chain.

The streptococcus may be found in the mouth in the normal or pathological state under different conditions. Widal and Bezançon have found



it in pseudo-membranous, diphtheritic, pultaceous, phlegmonous, and tubercular angina.

**Staphylococcal Pseudo-Diphtheritic Angina.**—Staphylococci also may be present in every variety of angina. I shall here discuss only the pseudo-diphtheritic variety. This form is very much rarer than those previously described. I have, however, found four cases in Martin's paper, and four in that of Chaillou and Martin. I have seen three cases in adults.

The patient shows the symptoms common to acute angina: febrile onset, dysphagia, inflammatory redness of the palato-pharyngeal mucous membrane, and swelling of the submaxillary glands. The membranes then appear, but they are not as thick, adherent, and extensive as in diphtheria. It is, however, quite impossible to diagnose the condition from true diphtheria by clinical methods alone.

Recourse must therefore be had to bacteriological examination. The staphylococcus forms, in less than twenty-four hours, wide, flattened, and irregular colonies on the serum, which at once indicate the diagnosis. The same culture, if allowed to remain in the oven, will soon give rise to much larger colonies, and after suitable staining the *Staphylococcus albus* and *aureus* are readily found. The slide shows a heap of grains which do not form little chains like the streptococcus, but are collected in grapelike bunches.

**Pneumococcal Pseudo-Diphtheritic Angina.**—This variety has been described by Jaccoud. It is very rare in children, and Chaillou and Martin have only seen one case. This disease has a sudden onset, with rigors, general malaise, and rapid rise of temperature to 103° or 104° F. On the first day the dysphagia is acute; the mucous membrane of the throat is red and shiny; the tonsils are swollen and purple. On the next day false membranes are seen in the throat. They commence in the form of white points, become confluent, and then spread and thicken like diphtheritic membranes. The angina is usually accompanied by marked swelling of the glands. If to these symptoms we add albuminuria, it is evident that this clinical picture closely resembles diphtheria. In the few cases which have so far been published, the disease has not the slightest tendency to invade the nasal fossæ and the larynx. In such a case it is impossible for clinical observation alone to decide whether the angina is diphtheritic or not. Bacteriological examination is necessary, and reveals the presence of the pneumococcus.

**Membranous Angina due to the Coli Bacillus.**—This variety of pseudo-diphtheritic angina is very rare. The pathological agent is the coli bacillus, which has been found as an accessory organism in several cases of angina; in some cases, however, pseudo-diphtheritic angina is due to the coli bacillus alone. Martin and Chaillou mention two cases, while Lermoyez has published a most conclusive case.

**Herpetic Angina.**—Herpetic angina (common membranous angina, Trousseau) will form the subject of a special section.

**Angina due to the *Micrococcus Tetrigenus*.**—In some cases this organism gives rise to an angina which somewhat resembles diphtheria. I give three cases which I have observed :

CASE I.—A healthy man was taken with fever, malaise, and pain in the right side. Pleural effusion was found on the right side. When I saw him some days later the effusion had almost disappeared, but the throat was covered with a peculiar white layer, which extended over the back of the pharynx, the velum palati, the pillars, and the uvula. In places there appeared a multitude of prominent isolated granules, which gave the throat the appearance of having been sprinkled with sand. I have therefore proposed the name "**sandy angina**" for this variety. Tonsils not swollen ; no hypertrophy of the submaxillary glands.

Apert sowed two tubes of coagulated ox serum and two tubes of peptonized agar with the exudate ; he also made preparations by crushing one of the granules between two slides, on which he found present, almost exclusively, encapsuled cocci, arranged in groups of four or in pairs. The serum culture showed no growth till the fourth day, but the tubes of agar from the first day showed a large number of prominent whitish colonies, which were glue-like, and formed threads when a fragment was lifted with the platinum wire. On microscopic examination, they were composed of tetrads, having all the characteristics of the *Micrococcus tetrigenus*.

CASE II.—The patient had been under my charge for a fortnight with pleurisy, and the effusion had dried up, when he was seized with angina. On each tonsil five or six lenticular points of a clear white ; it had the appearance of a follicular angina, but resembled the form which diphtheria also assumes. The serum culture gave within twenty-four hours streptococci and some colonies of staphylococci. The tubes of agar gave an almost equal number of tetrads and staphylococci. The *Micrococcus tetrigenus*, on being isolated, was cultivated in broth. A few drops of this broth, injected into a mouse, killed it in twenty-four hours ; the encapsuled tetrad was found in the blood.

CASE III.—A man was admitted to the Hôtel-Dieu with symptoms of influenza, accompanied by râles, due to pulmonary congestion and pleural friction sounds ; some whitish exudate on the throat. The serum culture gave a negative result as regards diphtheria, but on agar there were mysterious colonies, including streptococci, small cocci, alone or in pairs and heaps, and about a dozen colonies of the *Micrococcus tetrigenus*. This coccus, cultivated on broth, proved harmless on injection of a mouse with doses of  $\frac{1}{4}$  c.c.

In these three cases "the tetragenic angina was accompanied or preceded by pleurisy. In the cases of tetragenic septicæmia hitherto observed (Chauffard and Ramond, Castaigne) pleurisy was present. Netter, Faisans, and Le Danamy have found the tetrigenus in the fluid from sero-fibrinous pleurisy. The *Micrococcus tetrigenus* shows a liking for the pleura."

**Syphilitic Membranous Angina.**—In the previous section I said that the mucous patches on the throat and tonsils are at times covered with adherent greyish membranes which simulate diphtheria, the more as the glands at the angle of the jaw are generally enlarged. The diagnosis is easy if specific lesions are present in the mouth and pharynx, or if cutaneous or mucous syphilides have been found. In cases of doubt, or even in cases where the **two infections are superposed**, bacteriological examination can alone confirm the diagnosis. The absence of Löffler's bacillus excludes the





A. — CULTURE OF DIPHTHERIA ON GELATINIZED SERUM.  
DISCRETE COLONIES.

When the colonies of diphtheria are well developed, they are characterized by round whitish spots, more opaque in the centre than at the circumference. I call these colonies *papular*, because they project from the surface of the serum. Some resemble little grains of semolina.

B. — COLONIES OF DIPHTHERIA.

C. — BACILLI OF DIPHTHERIA.

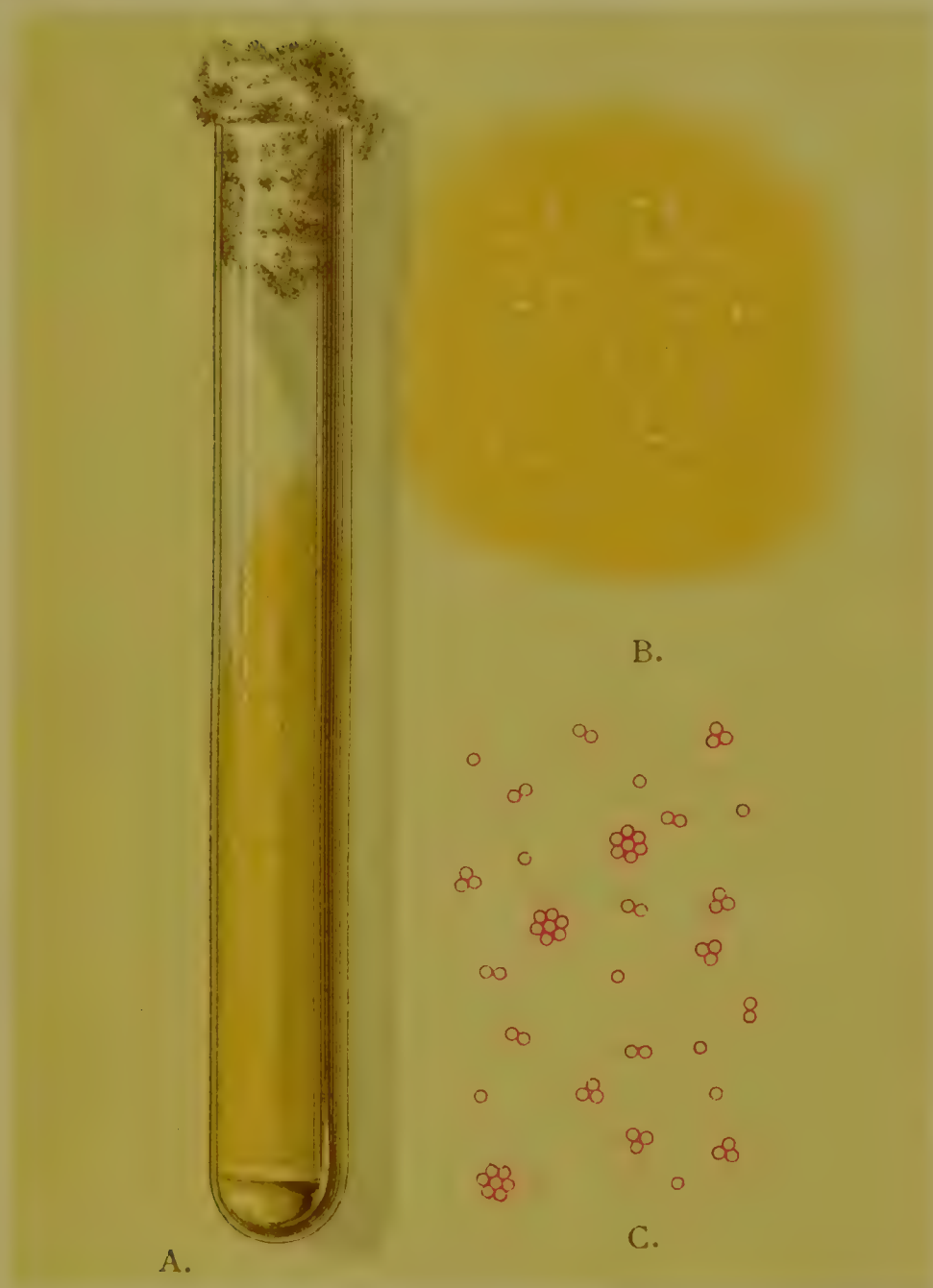
They are as long as, but thicker than, the bacilli of tuberculosis. They are swollen at their extremities, and are straight or slightly curved inwards. They are often disposed in groups of three or four, arranged in parallel lines, imitating the letters V, X, L, or simulating the acute (') or the circumflex (^) accent. They are never placed end-on. They look sometimes like short square needles which have been allowed to fall on a table in little heaps (Martin).





SMALL COCCUS, BRISOU'S COCCUS.

Plate II.



A. — CULTURES OF BRISOU'S COCCUS ON GELATINIZED SERUM. DISCRETE COLONIES.

The colonies are characterized by rounded and whitish spots, simulating at first sight colonies of diphtheria; but they are smaller, more humid, and more transparent than those of diphtheria. Their centre is not transparent, and they are *flat*, forming no prominence. I therefore propose to call them *macular*, in contradiction to the *papular* colonies of diphtheria.

B. — COLONIES OF BRISOU'S SMALL COCCUS.

C. — ELEMENTS OF BRISOU'S COCCUS.

Isolated micrococci are seen, as well as numerous diplococci and some little heaps of micrococci.





## THE STREPTOCOCCUS.

### Plate III.



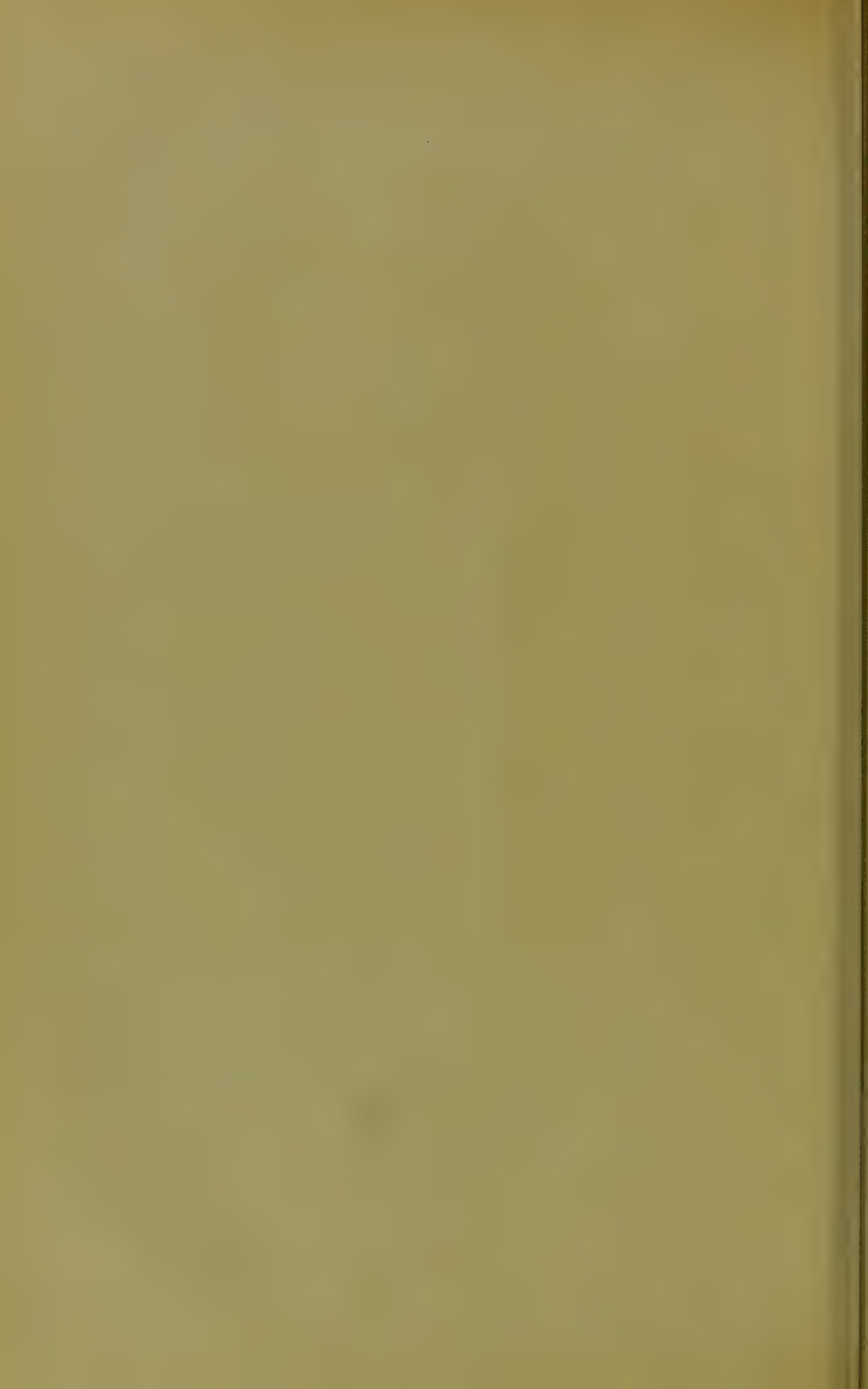
A. — CULTURES OF THE STREPTOCOCCUS  
ON GELATINIZED SERUM.

Colonies of streptococci, so small that they look like a whitish stippling. I therefore propose to call them *dusty*.

B. — COLONIES OF STREPTOCOCCI.

C. — LITTLE CHAINS OF STREPTOCOCCI.

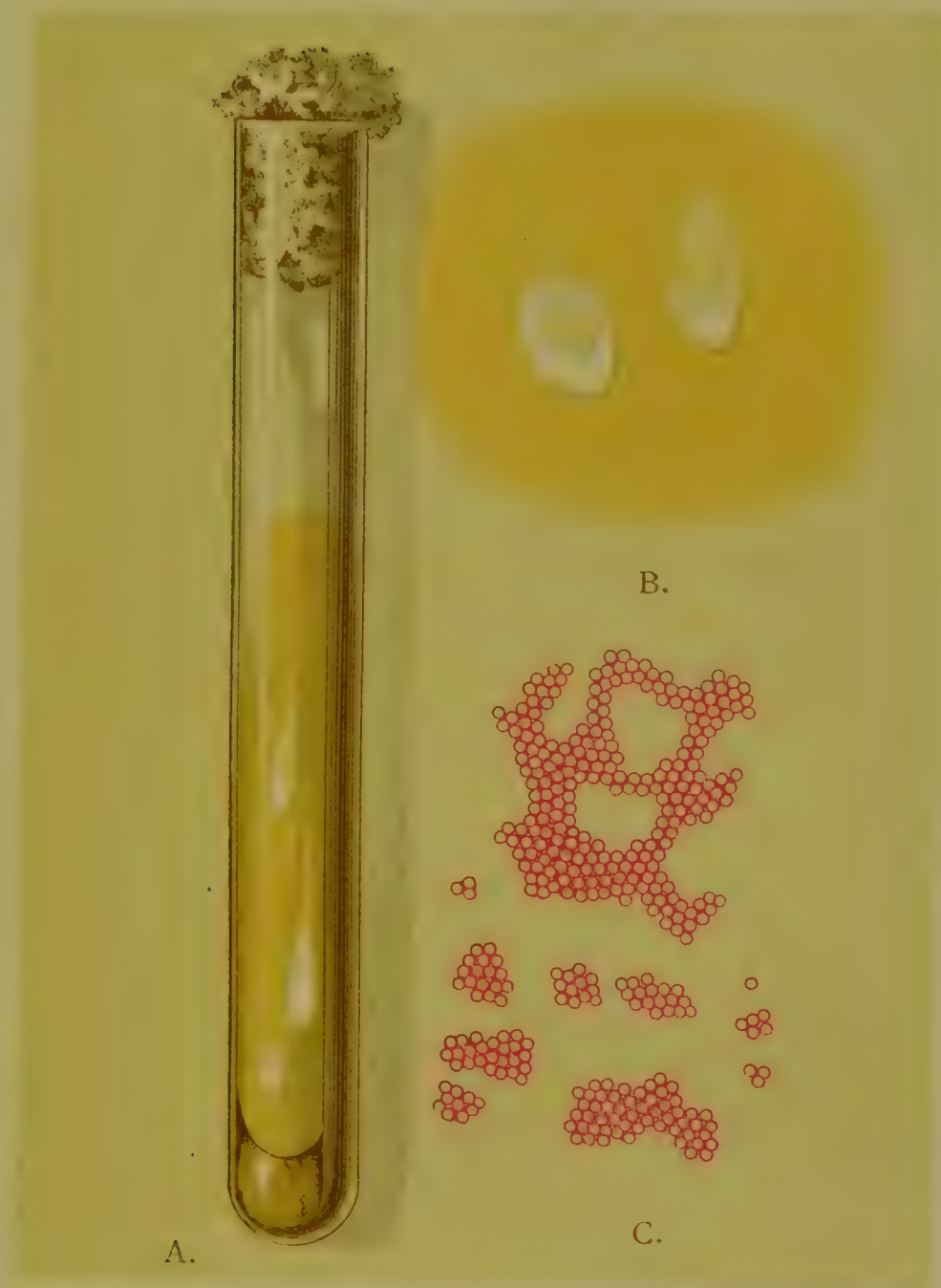
These chains vary in length, being straight, wavy, or curved inwards; they are formed of rounded granules, placed like a chaplet, containing three, four, five, ten, or fifteen elements, as the case may be.





# STAPHYLOCOCCUS ALBUS.

## Plate IV.



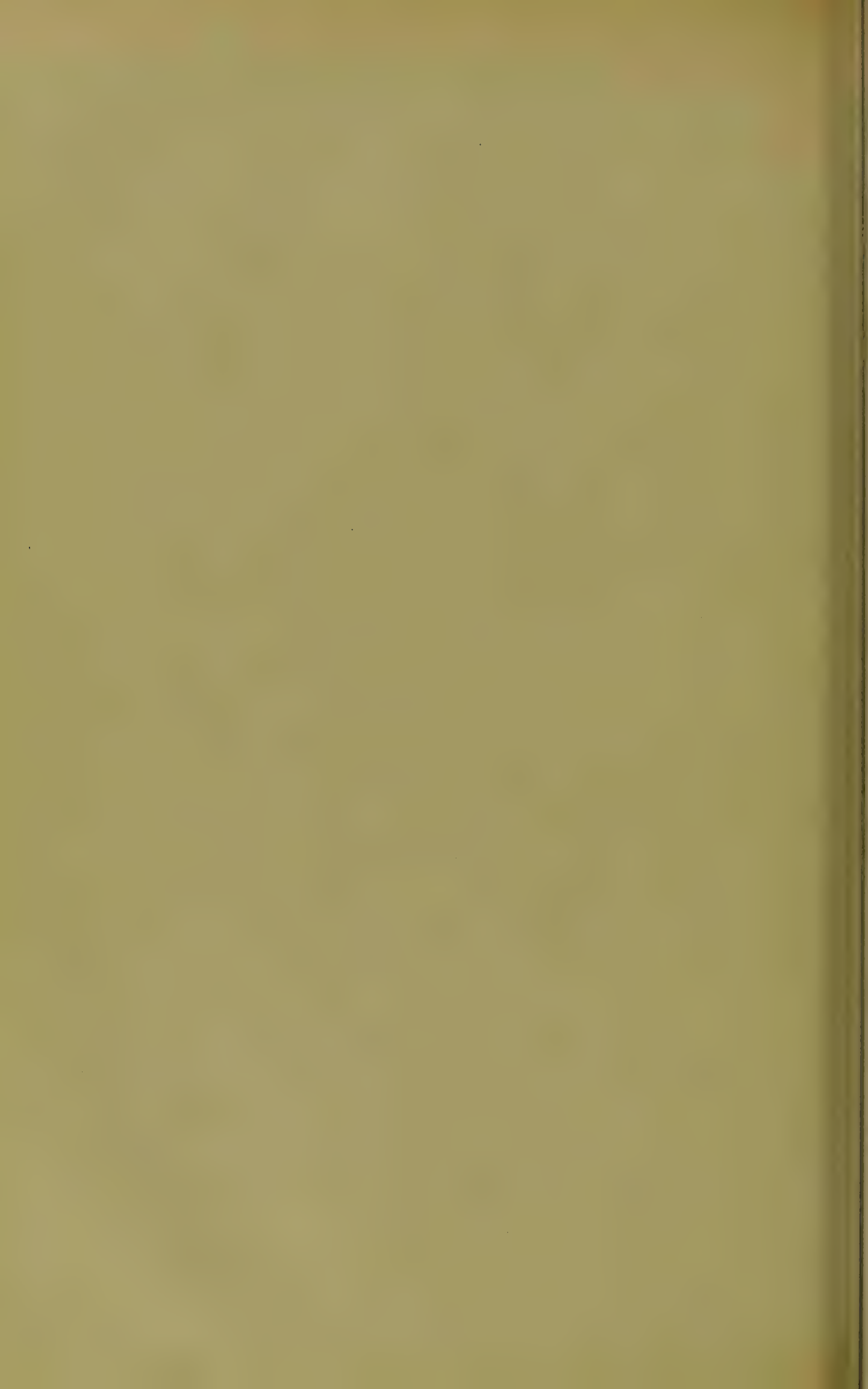
A. — CULTURES OF THE STAPHYLOCOCCUS  
ON GELATINIZED SERUM.

Colonies of white, flattened, and irregular staphylococci. These colonies are yellowish in the case of *Staphylococcus aureus*.

B. — COLONIES OF STAPHYLOCOCCUS ALBUS.

C. — CLUSTERS OF STAPHYLOCOCCI.

The elements of the staphylococcus do not form chains, like the streptococcus, but are united in clusters or masses.





hypothesis of diphtheria. Other microbes, either cocci or streptococci, may be present.

**Summary.**—I think that the reader will agree that clinical observation alone is often incapable of formulating a diagnosis. We are, however, armed with means which no longer admit of mistakes. In angina with whitish deposits, and especially with membranes, bacteriological examination must always be made, even though we feel certain of our diagnosis. The **diagnosis** and **prognosis** depend on this examination.

It is as easy and as simple to make a bacteriological examination in a case of angina as to make an examination of urine. The physician only requires some tubes of gelatinized serum. As soon as the tube has been sown, it is sent to a laboratory. The diagnosis and prognosis of the angina are verified, and we are not exposed to the uncertainty and the errors which I have already pointed out in this study.

Plates I., II., III., and IV., give an idea of the cultures and the morphology of the diphtheria bacillus, of Brisou's small coccus, of the streptococcus, and the staphylococcus, which may all be found in membranous angina.

### VIII. DIPHTHERITIC PARALYSIS.

**Description.**—Paralytic troubles may supervene in diphtheria which involves the skin or the mucous membranes, but they are chiefly seen in faucial or nasal diphtheria. Some days or weeks after recovery from the angina, or even in some cases while the angina is at its worst, paralysis affects the **velum palati**, which hangs motionless; the mucous membrane has lost all tonicity; the voice is nasal, the articulation of sounds is defective, and deglutition difficult, while food and drink regurgitate through the nose. When the pharynx is also paralyzed, the dysphagia is so great that, in spite of every effort, the bolus cannot be properly swallowed, and food may lodge in the larynx, causing fits of suffocation, which may prove fatal.

An exact idea of the hindrances to the act of deglutition requires a brief description of the physiological mechanism of this function. During the first act of deglutition the bolus is collected into a mass on the surface of the tongue, which is applied to the roof of the palate. The bolus, pushed from before backwards, passes the isthmus of the gullet, and enters into the pharynx. During this act the palato-pharyngei contract and obliterate the posterior nares. "The pharynx is raised, so that it meets and seizes the bolus of food; the larynx is also raised, because the middle and lower constrictor muscles and the stylo-pharyngeus are elevators of the pharynx and larynx. The larynx, therefore, follows the upward movement of the pharynx, and is carried forward under the base of the tongue, which is retracted. This

mechanism, which protects the orifice of the larynx, is completed by the movement of the epiglottis over the upper opening of the larynx" (Mathias-Duval). This mechanism prevents the entrance of food and drink into the nose or the larynx during deglutition. When, however, diphtheritic paralysis attacks the palato-pharyngei, the food passes into the nose; when the paralysis involves the muscles of the pharynx, the food passes into the larynx.

When the paralysis extends to the tongue and the lips, the symptoms at first sight simulate labio-glosso-laryngeal paralysis; the difficulty in pronunciation is very marked, and the saliva drools from the mouth.

The paralysis may be limited to the velum palati or be general, and affect in an irregular manner the limbs, the muscles of the neck and trunk, the sensory nerves and sense organs, as well as the sphincters, the œsophagus, the larynx, the muscles of respiration, and the heart. The paralysis almost always commences in the velum palati, but it may appear simultaneously at several points—viz., the limbs or the face. In rare cases the limbs are paralyzed before the palate, which may remain quite free. A paraplegic form that is independent of any other paralytic trouble has been seen. The paraplegia may be slight or severe; sphincter troubles and Babinski's sign are absent. The reflexes of the patella and tendo Achillis are abolished. Early paralysis of the muscles of the trunk and nape of the neck has been noticed. In the face the paralysis may affect one or both sides. When it is double, the physiognomy loses all expression and looks like a mask (Duchenne).

The patient may be almost unable to move his legs and arms. He has not the strength to sit down or turn in his bed; the head may be inclined to the right or the left, or may fall upon his chest. The muscular weakness is sometimes so pronounced that the paralysis is complete. These symptoms sometimes show variations; paralysis which was present in one limb may improve and show itself in another one. We may find neither muscular atrophy nor altered electrical reactions (Duchenne), but in other cases atrophy may be present and the reactions disturbed. In a few rare cases the paralysis is persistent. The **sensory** troubles consist of tingling, pricking, and pains, which generally accompany the muscular paralysis from the first; the pain may be very pronounced. Anæsthesia is frequent in the hands and feet; it affects all forms of sensibility and may involve one part of the body.

All the **sense organs**, but especially the eyes, may be affected by the paralysis. Ptosis, strabismus, mydriasis; myopia, amblyopia, and complete blindness have been observed. These troubles are **transient**, and the ophthalmoscope reveals no lesion of the fundus oculi. The **sense of taste** is often blunted, and the **sense of hearing** is sometimes affected.



The **bladder** does not escape, and retention or incontinence of urine may result, according as the paralysis affects the body of the bladder or the sphincter. Similar troubles are seen in the **rectum**.

Vomiting is common. Sexual debility or impotence may be present. The most serious complication of those just enumerated is the entrance of food into the respiratory passages, because death may result from asphyxia. In other cases, however, the paralysis may become dangerous, because it affects **the organs of respiration and the heart**. The **bulbar** form (Duchenne) causes troubles, which vary in severity. I shall merely sketch the principal varieties.

The muscles of the **larynx** may be paralyzed. If the paralysis attacks the muscles of phonation, dysphonia or aphonia results. If it attacks the posterior crico-arytenoidei muscles, the breathing is seriously compromised. When the diaphragm and the intercostal muscles are affected, the patient becomes breathless, because he cannot properly renew the air in his lungs. He may suffer from terrible **fits of dyspnœa**. Suffocation may also arise from paralysis of the bronchial muscles of Reissessen, which are intrinsic muscles of expiration (Duchenne). In addition to dyspnœa, cardiac troubles are sometimes seen: the pulse is irregular and slow, or more often quickened; angina, intermittence of the heart-beats, and fatal syncope may occur. The asphyxia and the syncope must be set down to central or peripheral poisoning of the pneumogastric nerve, and perhaps also to cardiac lesions (myocarditis).

In some cases the symptoms are so severe from the outset as to simulate angina pectoris; terrible precordial pain radiating into the arms and shoulders, cutis anserina, cold sweats, acute mental distress, and syncope characterize the **cardio-aortic** form. In other cases the abdominal symptoms are very marked. Gulat speaks of a patient who had paralysis of the velum palati and ocular troubles, when he was suddenly seized with abdominal pain, cardialgia, vomiting, restlessness, extreme dyspnœa, delirium cordis, and fatal syncope.

**Duration.**—The duration of the paralysis is variable. When the mischief is limited to the throat, the velum palati, and the pharynx, it usually lasts only a few weeks; when it becomes general, and affects the face, the limbs, the bladder, and the eyes, it may last several months. The paraplegic form may be of unlimited duration. In certain cases the paralytic complications are very rapid. During an epidemic, of which I shall speak shortly, some patients were carried off in a few hours, the diphtheria having from the outset attacked the organs of respiration and the heart.

**Ætiology.**—Paralysis is most common in **faucial** diphtheria. The angina may have been slight and apparently quite benign, but yet the paralysis may be so grave as to cause death. I am, however, of the opinion

that **nasal** diphtheria plays a large part in causing paralysis. The diphtheria bacillus finds a vast culture field in the nasal cavities, where it can elaborate its toxine. The proper temperature and the incessant renewal of air at each nasal inspiration are both present. Roux elaborated the diphtheritic toxine in a model of this apparatus. If we examine the cases of diphtheritic paralysis, we find that nasal diphtheria is the most frequent cause. The patients, whom I have had under my care during several years past with paralysis, have almost all had nasal diphtheria. Still, cases are known of unilateral paralysis of the soft palate following unilateral angina, and, moreover, similar results have been obtained experimentally.

Paralysis may follow diphtheria **affecting any part of the body**. Grave paralysis has been known to follow diphtheria which developed on the raw surface of a **blister**, whilst the mucous membranes were not affected. Paralysis may, indeed, appear in the aberrant forms of the disease in which the eruption is absent, though the other symptoms are severe.

Boissarie, in an interesting paper, describes an epidemic of diphtheria in which several people were carried off by acute paralysis without previous angina or other signs of diphtheria. In this epidemic some patients were attacked by membranous angina, which followed the paralytic troubles, instead of preceding it, as is usually the case. Other patients, again, had angina only, and were not troubled with paralysis. These facts are of extreme importance, since they prove that in diphtheria the membrane has only a relative importance. The poisoning may appear when the membranes are slight, or when a mucous coating without membranes is the only local manifestation of the disease.

Paralysis is far more common in adults than in children. Paralysis limited to the palato-pharyngeal region has been noticed in one out of every six persons with diphtheritic angina (Roger); general paralysis only occurs in about 5 per cent. of cases.

**Pathogeny.**—Many opinions have been expressed as to the **nature** of the paralysis. The most natural view is that the paralysis is due to **toxic poisoning**. This theory was upheld by Trousseau, and, although it was opposed for a long time, it has received abundant confirmation from the experiments of Roux and Yersin. As we have seen in a previous section, the microbe of diphtheria elaborates a toxine which is the cause of the paralysis. Roux and Yersin have reproduced paralysis in animals by the injection of filtered culture liquid which is free from micro-organisms.

This toxic poisoning having been proved, how is the paralysis produced? Is it the result of a nervous lesion, and what is this lesion? In 1862 Vulpian and Charcot published a case of diphtheritic paralysis of the soft palate, with changes in the muscular fibres and the palatine nerves. Pierret later described changes in the nerves, patches of spinal meningitis, and perineuritis



of the nerve roots. Dejerine has found atrophy of the sheaths of the nerves and parenchymatous neuritis of the anterior roots, consecutive to interstitial and parenchymatous myelitis.

The question has advanced a step farther. For several years past writers have described cases of **peripheral neuritis** which **seem** to run their course without affecting the nerve centres. This peripheral neuritis, which has been described under tuberculosis, diabetes, alcoholism, lead-poisoning, etc., has also been met with in diphtheria.

In a case of cutaneous diphtheria with subsequent paralysis, Pitres and Vaillard found peripheral neuritis of the radial, ulnar, median, external and internal saphenous nerves, etc., while the spinal cord was absolutely healthy. The lesions were very slight in the anterior and posterior roots. It is true that the patient had tuberculosis, which predisposes to peripheral neuritis. The progress and nature of the paralysis in this patient, its commencement in the soft palate, and its gradual extension to the limbs and the diaphragm, were certainly the result of diphtheria. It seems certain, therefore, that the diphtheria toxine can produce paralysis by acting on the peripheral nerves; but, in view of the cases of sudden death from affections of the lungs and heart, it is probable that the poison also acts on the cells of the nerve centres and paralyzes them.

**Diagnosis—Prognosis—Treatment.**—The diagnosis is simple and easy when the course of the angina has been followed, but it sometimes happens that a patient presents himself with general muscular weakness, incomplete paraplegia, incontinence of urine, facial paralysis, amblyopia, amaurosis, or dyspnoea, which may at first sight point to some affection of the spinal cord, the brain, and the nerves. If the paralysis has commenced in the soft palate, the nasal voice and the difficulty in swallowing at once indicate the correct diagnosis; but this guide is sometimes at fault, and we must then examine for sore throat with membranes and enlarged glands. We must remember that any form of diphtheria may be followed by paralysis, and that in some cases paralytic troubles may precede the formation of membranes, or may even be present in epidemics without any apparent membrane.

The **prognosis**, which is benign when the paralysis is limited, and very grave when the paralysis attacks the muscles of respiration and the heart, is always a matter of uncertainty, because we can never foretell what the course of the paralysis will be.

The treatment is difficult. As soon as deglutition becomes difficult, bougies must be employed. Preparations of nux vomica, liniments, hydrotherapy, and electrical treatment, should be employed, in addition to tonics, such as quinine and iron. Sulphate of strychnine gives good results. Injections of serum, given during the onset of diphtheria, diminish the risk of

paralysis, but seem to have little effect when once paralysis is present. This proves that injections of serum must be given without delay, for early injections prevent complications, while later they have not the same effect.

### IX. HERPETIC ANGINA.

Erythematous, lacunar, pultaceous, or diphtheritic angina may be accompanied by an eruption of herpes. When membranes appear, we reserve for it, with Trousseau, the name of **common membranous** angina, the epithet "**common**" removing all idea of diphtheria.

Herpetic angina does not, however, represent a morbid entity, and the disease, as our predecessors understood it, must, in my opinion, be split up. It is rather an angina with herpes than herpetic angina. In these cases we may find any of the following organisms—viz., micrococci, streptococci, staphylococci, pneumococci, and diphtheria bacilli. The vesicles, although analogous to those on the skin, run a slightly different course, because the morbid process which produces crusts on the skin may produce false membranes on the mucosa (Gubler).

**Description.**—Though herpetic angina may occur at any age, it is most common in children from three years onwards. It has a latent incubation period, which lasts from a few hours to two days, and it then bursts forth with violent rigor, high fever, and intense headache, which is sometimes as severe as in meningitis (Lasègue). This febrile condition is often accompanied by dyspepsia, and the patient has a burning feeling in the throat. The pain is generally limited to one side of the pharynx, and the submaxillary glands are but little swollen.

If the throat be examined at the outset, we may see the eruption of the vesicles. The mucous membrane is red, and the tonsils are swollen. Vesicles then develop on the tonsils, the pillars of the fauces, the uvula, and the soft palate. When they are discrete, they resemble **sudamina**; when confluent, they form more or less extensive and irregular groups.

These vesicles become opalescent, excoriated, and then covered with a whitish membrane. This process is often **so rapid** that the vesicular stage may pass unnoticed, and the false membrane only be seen. In other cases, on the contrary, the vesicles disappear without the formation of membranes. The false membranes may be found on the pharynx, the tonsils, the uvula, and the pillars of the fauces. When the membranes are clearly limited, they are generally surrounded by a reddish areola; when they extend beyond the limits of the erosion, they blend with the neighbouring exudates, and large membranous patches are formed. Another factor assists in the growth of the membranes. The inflammation is not solely confined to the vesicles, but attacks the neighbouring mucous mem-



brane also, and "though it is not ulcerative, it none the less gives rise to the exudation of plastic products like those which are formed by an ulcerated surface" (Trousseau). The membranes are frequently adherent to the subjacent mucous membrane. They may, however, become detached, and expose the mucous membrane, which may be ulcerated or completely cicatrized. These false membranes have almost the same structure as those of diphtheria. The fever and the dysphagia last for some days, and restoration to health takes place suddenly. **Second attacks** are not rare. Herpetic angina is sometimes accompanied by an eruption of vesicles on the nostrils, the lips, the mouth, the prepuce, the vulva, or the cervix uteri. These eruptions are of help in diagnosis and indicate the **nature** of the angina. Vesicles may be found in the **larynx**; **herpetic croup** is the result, but it has neither the character nor the gravity of diphtheritic croup.

We have seen in a previous section that the diagnosis is made by **bacteriological examination**.

Herpetic angina may be epidemic. It is liable to occur a second time in menstruating women. Cold is the chief determining cause. The **prognosis** is benign. The **treatment** is very simple—viz., soothing and antiseptic gargles.

## X. GANGRENOUS ANGINA.

**Ætiology.**—Gangrene of the pharynx is more common in children than in adults; it may be primary or secondary. The **secondary form** is by far the more frequent, and is caused by diphtheria, measles, scarlatina, dysentery, smallpox, or typhoid fever—in short, by any morbid condition which lowers the vitality and favours secondary infection and death of the tissues, not only of the pharynx, but also of other regions. Other causes, such as inflammation of the throat, and especially phlegmonous angina, are much less common. Diphtheritic angina may sometimes apparently become gangrenous in adults, but we must not mistake the appearance for the reality. On the other hand, the opposite error must be avoided, for diphtheria may cause gangrene of the throat, the vulva, and the vagina. In this event the condition is grave, and the diphtheria is associated with other organisms. **Primary** gangrenous angina may be seen in adults. Its chief feature is early mortification of the pharyngeal mucous membrane, comparable with gangrene of the mouth.

Many organisms are present in gangrene of the pharynx. Pyogenic micro-organisms and saprophytes are associated with the specific germs.

**Description.**—The invasion is generally insidious in the secondary form. The primary disease begins with fever and pain. On examining the throat, greyish or blackish **patches of gangrene** are seen on the tonsils

and other parts of the pharynx, varying in size from a lentil to a shilling. The edges of the patch are prominent, irregular, and surrounded by livid red mucous membrane. If the gangrene is circumscribed, the scab becomes detached, and leaves in its stead an ulcer, which attacks the subjacent muscular layer, and may terminate by cicatrization. If the gangrene is diffuse, the gangrenous patches unite, invade the soft palate and the uvula, and in some cases extend forward as far as the lips, or backwards as far as the aryteno-epiglottic folds.

Gangrene of the pharynx is generally accompanied by marked glandular enlargement. The breath is **fœtid**, salivation is abundant, and the voice is nasal. Difficulty in swallowing varies. There is a foul discharge from the nose. Prostration and adynamia are always present. The pulse is feeble and slow; the skin is pale; delirium and restlessness are sometimes present. The **prognosis** is often fatal. The patient may survive two to six days in the secondary form, and eight days to a fortnight in the primary form, when death occurs from coma or sudden syncope.

Two patients of Richardière recovered. "The first patient, who had diffuse gangrene, recovered, with much loss of substance, due to sloughing of the soft palate, the tonsils, and the pillars of the fauces. This loss of substance interfered considerably with deglutition and phonation; food and drink regurgitated through the nose; the voice was nasal. The child was fed for several weeks with the œsophageal tube. After four to five weeks the loss of substance was limited, as it were, by regeneration of the velum; deglutition became easier, and the child could take nourishment by the mouth without regurgitation through the nose. The second child, who suffered from gangrene limited to the tonsils, recovered without other deformity than the loss of the tonsils."

The **treatment** consists in cauterization of the sloughing surfaces (hydrochloric acid, thermo-cautery), and frequent cleansing by means of disinfectants (hyposulphite of soda, eucalyptus). The patient's strength must be supported by tonics (quinine, wine, etc.).

## XI. CHANCRE OF THE TONSIL.

**Description.**—Chancre of the tonsil is very common. Every year I see four or five cases at the Hôtel-Dieu. If mistakes are frequent, it is because we are not familiar with its various symptoms and forms. This point is well worthy of recognition. Due attention has not been directed to the fact that this chancre often commences with **pain, fever, and sore throat**. We are so accustomed to consider chancres in general as indolent and apyretic lesions, that symptoms of acute angina, with fever, lead us wrongly to reject the idea of chancre. I have consulted the recorded cases as well as my personal observations, and I maintain that in half, or even more, of the cases this chancre commences, like tonsillitis, with fever, headache, and malaise. In a clinical lecture on **chancre of the tonsil**, I have recorded



several cases which began like acute tonsillitis, but were really chancres of the tonsil.

I do not say, of course, that every chancre of the tonsils has such an acute onset, with fever and pain, but this feature is so common that it deserves mention. We find at this period diffuse redness and swelling of the tonsil, which may be covered by a pultaceous coating. The chancrous erosion may be apparent or hidden. Later, when the chancre is developed, it may present the following varieties :

**First Variety.**—The chancre resembles an acute tonsillitis; pain, fever, dysphagia, headache, and lassitude are present. The patient thinks of simple tonsillitis, and employs soothing gargles, and hot compresses to the neck. As he begins to feel worse he seeks advice, telling the physician : “There is hardly anything wrong with me, but a sore throat has been causing me some trouble for the last three or four days.” The physician examines the throat, finds it red or covered with a pultaceous coating, while the tonsils are swollen. It looks like an ordinary tonsillitis, “without special characteristics, at any rate, for the time being.” The diagnosis is clearly difficult at this period. The chancre develops in the sulci of the enlarged tonsil, which may be covered by a pultaceous coating, so that even an experienced eye may not always distinguish it. We can only make a diagnosis by means of the signs, which we shall study later.

**Second Variety.**—In this case the chancre is visible on the tonsil, as an erosion which may be red and glossy, or grey and opaline, varying in size from a lentil to a shilling. The following case is typical of this erosive chancre :

A patient came to the hospital to consult me for painful angina, which had begun a fortnight before with high fever. He had been obliged to take to his bed. Multiple painless glands had made their appearance in the submaxillary and retro-maxillary regions on the right side. The symptoms were quite in accordance with the diagnosis of infectious angina. The fever and general malaise did not cause the least suspicion of chancre of the tonsil. On examination of the pharynx, however, I saw that the right tonsil was alone affected. It was prominent, and on its surface there was seen a **glossy red patch**, with a curly border. The left tonsil was healthy; no false membranes were present. As the erosion and the induration of the right tonsil, together with the presence of painless swollen glands, were unilateral, I diagnosed chancre; my diagnosis was soon confirmed by the appearance of the roseola.

**Third Variety.**—The chancre is ulcerated and the tonsil, which is more or less enlarged, is hollowed by an ulcer, which may be as large as a florin. The ulcer may be situated on any part of the tonsil. The colour is brownish or greyish, and the raised edges, formed by the projection of the tonsils, cause it to look deeper than it really is. The following case (Le Gendre) gives an idea of it :

A man was admitted for sore throat, pain, fever, malaise, and headache. An ulcer of the size of a sixpence was visible on the right tonsil. The edges were clean-cut;

the floor was greyish, sanious, and anfractuons. The ulcerated tonsil was a third larger than the other one. Palpation showed that the ulcer rested on an indurated base, while the corresponding maxillary glands formed a mass as big as a pigeon's egg. The pain radiated from the throat to the ear. The patient complained of insomnia, caused by headache, which was worse at night. Fournier diagnosed chancre of the tonsil, and the subsequent roseola confirmed his diagnosis.

**Fourth Variety.**—The chancre is diphtheroid. The ulcerated surface of the chancre is covered with a greyish or yellowish adherent membranous exudate, which at first sight looks like diphtheria. Whether the membrane is due to the tendency which every chancre of the mucous membrane has to become covered with a more or less thick pseudo-membrane, or whether it is due to secondary infection by the streptococcus, *Brisou's coccus*, etc., it is none the less true that in certain cases the chancre is hidden under a more or less adherent membrane of a greyish colour, which may give rise to errors in diagnosis. This mistake consists in not recognizing an existing chancre, or in diagnosing diphtheria which does not exist. The next case is taken from my clinical lectures :

In February, 1898, I saw a patient who had been taken ill in January with membranous sore throat and enlarged glands on the left side. The glands were hard, painless, and quite separate from one another. On examining the throat, an ulcer, covered by a thick, greyish, adherent membrane, was seen on the left tonsil. I was in doubt between chancre and diphtheria. Bacteriological examination of the exudate revealed short diphtheria bacilli. Next day numerous colonies of the same bacillus were found in the serum. Was this a case of only pseudo-diphtheria—for the short bacilli are not considered by most authors as true virulent diphtheria—or was it one of diphtheria ?

An injection of serum was given, but led to no improvement in the local conditions.

A guinea-pig was inoculated with 2 c.c. of a broth culture of the same bacillus ; neither fever nor œdema at the point of inoculation. Further injections of serum were therefore considered useless, as the diagnosis of chancre was clear, and specific treatment was promptly commenced. On February 8, thirty-five days after the appearance of the chancre, the roseola appeared. When I saw the patient, the tonsil was still indurated and of large size, the adenitis was still present, and the roseola was in course of evolution.

**Fifth Variety.**—The chancre is gangrenous. The appearance of the lesion, its blackish colour, and the foetor of the breath, give the impression of gangrene of the tonsil. This gangrenous chancre was well marked in the following case :

A young man admitted into the Charité had suffered for a month from difficulty of swallowing, headache, and a painful swelling in the neck. In the right cervical region there was a mass formed of large tender glands. The isthmus of the gullet was red ; the right tonsil was swollen, and on its upper portion was a gangrenous patch  $\frac{1}{2}$  inch in diameter. The eschar was separated from the rest of the tonsil by a demarcation sulcus filled with pus. The breath was foetid and the dysphagia marked. No diagnosis was made, and tonsillectomy was performed. The cause of the lesions in the tonsil was soon explained by the appearance of the secondary rash.

**Sixth Variety.**—By slightly straining the analogy, a chancre of the epitheliomatous kind has been described.



These different cases prove that chancre of the tonsil is **polymorphous**. Whatever its morphology may be—whether it resembles hypertrophic tonsillitis, or whether it is erosive, ulcerative, diphtheroid, or gangrenous—its onset is often painful, and accompanied by fever and general symptoms. It is well known how painless chancres of the genital organs are; chancre of the tonsil, on the other hand, often causes pain and fever. In one of my cases the chancre began with sore throat, rigors, weakness, severe dysphagia, and pains which radiated to the ear, neck, and face. In another patient an erosive chancre also began with intense dysphagia, rigors, fever, and lassitude. Le Gendre's case of a diphtheroidal chancre started with rigors, dysphagia, malaise, and attacks of vertigo. In other cases quoted by Le Gendre the disease began with fever and acute pain on swallowing.

**Diagnosis.**—Chancre of the tonsil begins in many cases like an acute tonsillitis, so that the diagnosis is obscured when the characters of chancre of the tonsil are not familiar. This fact so impressed me, that some years ago, in my lectures at the Faculté,\* I pointed it out at some length. Fournier states the same opinion, and describes this form under the name of **chancre of the anginoid type**.

At first the diagnosis is naturally doubtful. The patient is examined, and tonsillitis or peritonsillar abscess is discussed, and even if the idea of a chancre has occurred to the mind, the means of confirming the diagnosis are not yet at hand. The variety of chancre soon becomes clear: the tonsil, in which the chancre is more or less hidden, is red, swollen, and furrowed. In other cases an erosive, ulcerative, diphtheroidal or gangrenous chancre appears. The aspect of the chancre may vary; pain and fever may or may not be present; but yet in a few days we find the triad of symptoms, which makes diagnosis possible—unilateral lesion, induration of the tonsil, and development of a glandular mass.

The chancre is unilateral, which means that only one tonsil is affected, and this unilateral character is of great value. In every other variety of tonsillitis both tonsils are attacked, though possibly to an unequal degree; they are more or less swollen, red, and painful. In chancre, on the other hand, only one tonsil is affected. There are, however, exceptions to this rule. Julien has recorded the case of a young girl suffering from general roseola, with an ulcerative chancre on each tonsil, and enlarged glands on both sides of the neck.

Cases of this kind are, however, exceedingly rare, and Fournier, who quotes those of Duncan Bulkley, has never seen an instance. I have seen several chancres of the tonsil, but I have never seen a case in which the lesion was bilateral. The chancre may not be absolutely confined to the tonsil.

\* "Le Chancre de l'Amygdale" (*La Semaine Médicale*, 3 Avril, 1895).

It may encroach on the anterior pillar or on the base of the tongue, but it still preserves its unilateral character.

**Induration** of the tonsil is a sign of the utmost importance. If both tonsils are palpated, the healthy organ feels supple, while the other is indurated, especially around the base of the suspected lesion. This induration of the chancre, though not so readily made out as on the genitals, is none the less a valuable sign, which is present in at least eight cases out of ten.

“Some chancres of the tonsil are as indurated as those on the prepuce or the vulva, and it is not impossible that a chancre of the tonsil may form a cartilage-like meniscus, which is as hard as wood.” The induration, just as in genital chancres, persists for a long time after the disappearance of the chancre, and allows a retrospective diagnosis to be made. In palpating the tonsil we may place one hand on the side of the neck and push the tonsil against the examining finger inside the mouth.

The third sign—viz., **glandular bubo in the neck**—must be carefully sought for, as its absence practically excludes chancre, while its presence confirms the diagnosis. The satellite bubo appears a few days after the chancre, and is situated in the neck on the same side, and not far from the thyroid cartilage. We often find, in addition to the bubo, a chain of smaller glands, which are mobile, hard, and painless. It is by “*hunting after glands*” that we may sometimes assert the previous existence of a chancre after it has disappeared. The chain may extend from the angle of the jaw to the supraclavicular region. In exceptional cases some of the glands on the opposite side may be enlarged, as in a case which I have reported. Although this sign has not previously been described in unilateral chancre of the tonsil, I point it out, because the presence of a bubo on both sides of the neck must not lead to mistakes. With chancre of the genital organs the inguinal bubo is usually bilateral; with chancre of the tonsil it is quite the exception. In order to explain the presence of enlarged glands on the side opposite to the chancre, we must admit that the infection has been spread by the lymphatics of the tonsillar and palatine mucous membrane, which anastomose with those of the opposite side at the base of the tongue. To sum up: the unilateral position of the lesion, the induration of the tonsil, and the presence of the satellite bubo, with its shotty glands, form a triad of symptoms which proves chancre of the tonsil. In making this diagnosis, however, we must remember that chancre of the tonsil often resembles an acute angina.

The diphtheroid chancre may simulate diphtheria, but the limitation of the membranes to the tonsil and the chancrous ulceration, exposed after the removal of the membranous layer, are signs which, with the triad of symptoms, will give the correct diagnosis. However, in cases of doubt bacteriological examination and cultures must be made.



A chancre must not be mistaken for ulcerative tonsillitis, due to fusiform bacilli and spirilla (Vincent). See section on Tonsillitis.

There remains, then, the differential diagnosis between chancre and epithelioma of the tonsil. Some analogy exists between these lesions as regards appearance. Enlarged glands and pain are present in each case. There are, however, certain differential characteristics. In chancre the adenitis appears much earlier than in cancer. In cancer it is a question of weeks, in chancre of days. In doubtful cases the progress of the affection decides the question, for chancre shows a tendency to recovery after three weeks, whilst cancer is of slow growth, and does not recede, but extends and bleeds. Finally, cancer is not accompanied by roseola and mucous patches, which closely follow the appearance of the chancre. Doubt, therefore, cannot last long.

I have still to mention the diagnosis of chancre from gumma and tertiary ulceration of the tonsil. Tertiary syphilides of the tonsil are not accompanied by the satellite bubo, unless secondary infection is present. Furthermore, the chancre occurs as the primary lesion in an individual so far free from signs of syphilis, whilst gummata occur in persons who have previously shown such signs.

The diagnosis of chancre of the tonsil is confirmed by the appearance of the roseola and mucous syphilides, which must be carefully looked for five to eight weeks after the appearance of the chancre.

Before closing this section I should like to say a few words on **herpes**, which sometimes accompanies chancre, and may lead to the erroneous diagnosis of herpetic angina. One of my patients had a chancre of the tonsil, and also an eruption of herpes on the left pillar of the fauces, the right tonsil, the lower lip, and the tongue. Had the herpes supervened fortuitously, or was it connected with the attack of syphilis? In 1866 Diday and Doyon published a work on genital herpes, and later, in 1868, Doyon described recurrent herpes of the genitalia in syphilitic patients. In 1879 Julien discussed the question fully. "A fact," says he, "that deserves notice in connection with uterine chancres is the frequent coincidence of genital herpes: the perineum, the vulva, and the cervix uteri are often the seat of herpetic eruptions. A syphilitic lesion of the cervix must also be thought of when a woman presents herself without other apparent lesions than a crop of herpes."

Fournier has also discussed the question of recurrent herpes of the tongue in syphilitic patients. "You see people," says Fournier, "who, after an attack of syphilis one or more years previously, consult you, because they fancy that they have bucco-lingual mucous patches, which recur in spite of treatment. If you examine carefully, you will find that these so-called mucous patches are polycyclical or microcyclical erosions—that is

to say, herpes." Syphilis, therefore, favours the eruption of "parasyphilitic herpes." Sometimes the herpes supervenes at a period more or less distant from the chancre, and reappears more or less often in the recurrent form. The eruption of herpes is sometimes contemporary with the chancre. In my patient, herpes supervened during the active stage of a tonsillar chancre, and in Le Gendre's monograph I find that one of his patients had a chancre on one tonsil and a crop of herpes on the other.

The reader is referred to the next chapter for the **Treatment** of tonsillar syphilis.

## XII. SYPHILIS OF THE SOFT PALATE, THROAT, AND PHARYNX.

While chancre of the isthmus of the gullet and of the pharynx is rare, secondary and tertiary lesions are common in these regions.

**Secondary Lesions.**—I must first mention the **erythematous** angina, characterized by erythema, which is diffuse or limited to the soft palate, pillars, and tonsils. The erythema often has a red colour. Sometimes the anterior pillars of the soft palate and the uvula appear to have been painted with a brush. The erythema, which coexists with mucous patches, causes some dysphagia, and is often accompanied by cold and hoarseness, because erythema of the larynx is also present in many cases. The condition deserves mention, because it has a certain value in diagnosis, as I have often proved.

**Mucous patches** in the throat may present various appearances. They include erosions, papules, hypertrophied papules, and ulcerations (Fournier). The patches are usually situated on the tonsils, the pillars, and the soft palate. They are extremely rare in the pharynx, no doubt because the mucous membrane is almost wanting in papillæ. The tonsillar region is a "very nest for syphilides." The patches are flat, opaline, and sometimes so confluent that they cover the tonsils and the pillars of the fauces. In some patients the mucous patches in the throat and on the tonsils are covered with greyish adherent false membranes, which simulate diphtheritic angina, the more in that the submaxillary glands are swollen.

The forms of syphilis just described cause discomfort and even pain. They interfere with mastication and render deglutition painful. They make their appearance about two to three months after infection, and are prone to recur for two or three years. They are moist and essentially **contagious**. Syphilis of the throat affects the corresponding glands and determines adenitis at the angle of the jaw.

As regards secondary syphilides, a point in diagnosis, which I have not seen mentioned elsewhere, was suggested to me by the history of a case in the Necker Hospital.



A woman had a red and painful swelling of the vault of the palate. I found on the swelling and in the neighbourhood superficial polycyclical ulcers and some rounded opalescent granules, which looked like yellow tubercles; the granules were also visible on the left tonsil. The diagnosis had been either syphilis or tuberculosis, but I accepted neither view. A few days before, she had been taken ill with pain, which interfered with mastication and deglutition. The polycyclical aspect of the ulcers did not in the least resemble those of syphilis. The so-called tubercles proved on close examination to be vesicles of herpes. The groups of vesicles on the roof of the palate and the tonsil, the surrounding erythema, and the excessive pain, led me to diagnose **zona palato-pharyngea**. As a matter of fact, new groups of vesicles developed in a few days on the roof of the palate, soft palate, and tonsil. The patient stated that she had often had similar troubles at the menstrual period. She recovered in a fortnight. This case is not an isolated one, and others have been published.

**Tertiary Lesions.—Gummata** of the throat and of the pharynx deserve careful attention, because they are common, not only in acquired, but also in **hereditary syphilis**. In acquired syphilis the gummata do not appear, as a rule, until some years after infection. In the hereditary disease lesions of the throat are met with at all ages between five and twenty-five, and the proof that these lesions are common is that they were found forty-six times in 212 cases of hereditary syphilis, which make up the returns of Fournier. The description refers, therefore, both to the gummatous lesions of **acquired** and of **hereditary syphilis**.

The lesions are of different kinds; the infiltration is sometimes circumscribed and forms a tumour, but in other cases the infiltration is spread out, and we see but little tumour formation. The **gumma** commences with infiltration. At this stage, though the soft palate is deformed, thickened, violet in colour, and indurated, the patient may experience no functional trouble, no pain, and only slight dysphagia. The gumma projects as a little tumour. We find next a period of softening, characterized by pain, which may be so slight that patients do not trouble about it, and are quite surprised when symptoms of perforation appear.

The ulcerated gumma varies in appearance, according as it is followed by perforation or not. In the latter event the ulcer forms a deeply excavated cavity, large enough to contain a lentil or a hazel-nut. The floor is covered with a greyish sloughy layer; the edges are clean-cut, adherent, and often perpendicular, while the surrounding tissues are smooth, or red, hard, and infiltrated. At this period the pain is very great, and if the ulcers run together at the isthmus of the fauces, the dysphagia is extreme.

Gummata of the soft palate often end in perforation, which establishes communication between the throat and the posterior cavity of the nasal fossæ. The progress may be so rapid that the opening may become as large as a lentil in twenty-four hours, and in three weeks as large as a penny. The perforation may be median or lateral; and rounded or elongated in a transverse direction. The ulcerative process continues its ravages, so that

the soft palate is sometimes split in two, or almost entirely destroyed. In some cases several perforations have been found. The perforation at once causes alteration in the voice, which becomes nasal, and regurgitation of food and drink into the nasal cavity. In some cases the sudden appearance of these symptoms greatly surprises the patient, because he has had no idea of the nature or the gravity of "his sore throat."

Tertiary ulceration often occurs in the triangular space formed by the junction of the two pillars of the fauces and the tonsil. The ulcerations extend from this space and "destroy the upper half of the tonsil and the pillars, and in some cases the free portion of the soft palate, the lateral wall of the pharynx, and the Eustachian tube. This fact explains the presence of deafness and acute pains in the ear.

When the syphiloma is diffuse, it does not stand out in relief, but gives rise to spreading ulcers, which are extremely painful. They are phagedænic, serpiginous in outline, and are frequently found on the soft palate. The ulcerative process may be so rapid that the tissue seems to melt away. The soft palate may be partly or entirely destroyed, though it may be impossible to see a single slough separate. The condition is one of rapid necrobiosis, presenting here, as on the skin, the characteristics of **phagedæna**. The ulcer is bathed in ichorous pus, the floor is pale, and the edges are loose and jagged. Hæmorrhage is frequent. The pain is so acute that deglutition and alimentation become most difficult.

The tertiary lesions which we have just described in the soft palate and throat are likewise common in the pharynx. The posterior wall of the pharynx is a favourite seat for gummata. They are generally painless, causing only slight dysphagia, as long as ulceration is absent. Patients, therefore, do not trouble about them, and "gummatous syphilides are only seen in practice in the stage of ulceration. At this period they may be very painful and have definite characteristics: circular shape, yellowish, sloughy floor" (Fournier). When the gummata are seated in the **naso-pharynx**, they can only be discovered by special examination. The tertiary ulcers of the pharynx present special peculiarities. The mucous membrane of the posterior wall of the pharynx is swollen and reddish. In some places it is covered with crusts, and in others a greenish pus bathes the furrows between the projections of the mucous membrane. The ulcers may leave deep whitish scars, which cause deformity, and favour adhesion to the neighbouring parts. Out of 150 cases of syphilitic stenosis of the pharynx, Lubinski found sixteen cases of complete atresia of the isthmus of the gullet. When the soft palate is fixed to the posterior wall of the pharynx, nasal respiration is impossible, and the senses of taste and smell are lost.

Diffuse syphiloma of the **sclerous form** is also seen in this region. The pillars of the soft palate may be so infiltrated that they become three or



four times as thick as normal. The parts lose their suppleness and become indurated and deformed. The pillars assume a furrowed and mammillated aspect; the isthmus of the gullet is constricted; the uvula is thick and indurated; and the tonsils are hypertrophied, lardaceous, and cracked. These lesions interfere with phonation and deglutition to a greater or lesser degree. The affection begins in an insidious manner, so that the only symptoms may be those of angina, and the patient does not worry about the disease, which slowly pursues its course.

**Diagnosis.**—The tertiary ulcers of the throat must be distinguished from **tubercular** ulcers. Many errors were made until late hereditary syphilis became known, and many troubles due to hereditary syphilis in children, youths, and even in adults, are still set down to scrofula.

This question has been ably discussed by Fournier, whose writings will serve me as a guide. In a case of acquired syphilis, when the subject, who is attacked by gummatous ulcers, has already had a series of syphilitic lesions, the diagnosis is often simple; when, however, we find ulceration of the throat in a young subject from twelve to fifteen years of age who never had syphilis, but who has had since childhood chronic discharge from the ear, persistent eye troubles, or chronic coryza, with or without ozæna, and thus presents “the old picture of scrofula, such as our predecessors bequeathed to us,” the idea of syphilis is rejected. An error then occurs, because this **old picture of malignant scrofula** is precisely the description of *heredo-syphilis*.

Let us see, then, what are the local differences between syphilitic and tubercular ulcers of the throat. The former have often been confounded with the ulcerations of **lupus** and of **tuberculosis**. Lupus and tubercle are varieties of the same morbid species, and, “in any case, they present, in their chronic form, characters so different that it is incorrect to unite them in one description” (Fournier).

Let us commence with **lupus**. In the first place, lupus of the throat is extremely rare—so rare, indeed, that out of 100 cases of lupus Fournier found only two cases of lupus of the throat. Furthermore, lupus of the throat exists almost always concurrently with lupus of the face, and the ulceration of the throat only occurs as a late phenomenon of the infiltration period, which is very long. This infiltration of the tissues is shown by a granular hypertrophy, which gives to the soft palate and the affected parts the appearance of “a carpet of mulberry-like granulations,” and has not the slightest resemblance to the syphilitic gumma. The phase of ulceration is likewise dissimilar. In the gumma the ulceration is sudden, rapid, and invades from the first nearly the whole of the infiltrated zone; but in lupus the ulceration is slow, and spreads by successive outbursts. In lupus the ulceration is only a part of the lesion; in the gumma it is the

entire lesion. The gummatous ulcer has a circumscribed outline, clean-cut adherent edges, and an excavated floor, which is greyish and sloughy. In lupus the ulcer has no defined outline, the flat edges are not clean-cut, the floor is but slightly depressed, and is covered with fleshy granulations. Syphilis perforates the velum palati and "makes a hole," but does not involve the surrounding parts. Lupus eats away the tissues, and the surrounding parts are never healthy. Syphilis takes only a few weeks, or at most a few months, to produce its effects, while lupus takes years. These local characteristics suffice to differentiate the lesions in acquired or hereditary syphilis from those of lupus. In a suspicious case of hereditary syphilis the inquiry must include the parents.

Let us now consider the diagnosis between gummatous and tubercular ulcerations of the throat. In the first place, a patient with tubercular ulceration of the throat is almost always suffering from pulmonary phthisis or from intestinal tuberculosis. Ulceration of the throat is hardly ever the first manifestation of tuberculosis. These features, however, are not sufficient to prove the diagnosis, because the patient may have both tuberculosis and syphilis. The tubercular ulcerations are less extensive and not so deep as those of syphilis; they have neither the clearly-notched edges nor the excavated sloughy floor. The tubercular ulcerations are frequently surrounded by projecting yellowish granules; they are sometimes accompanied by swelling of the cervical glands, whilst the "gummatous lesions only react in exceptional cases on the neighbouring glands." Finally, the secretion or the scrapings from a tubercular ulcer reveal the presence of the *bacillus*.

**Treatment.**—In tertiary lesions mercury is the best drug. Daily injections of a watery solution of biniodide of mercury are given. The injections are given for twelve to fourteen days; they are then stopped and repeated as required. Prompt action is imperative. We must remember that gummata in the throat or the soft palate terminate rapidly in perforation and destruction of the tissues. Correct treatment prevents these complications. The future will teach us the value of Ehrlich's 606.

Tobacco, alcoholic drinks, and highly-spiced foods should be forbidden, because they favour the return of mucous patches. The mouth must be kept scrupulously clean. If the mercurial treatment produces inflammation of the gums, chlorate of potash should be given daily in solution. The following remedies may be employed locally:

1. Soothing gargles of decoction of mallow and poppy.
2. We must clean the ulcerations with the following solution:

|                     |    |    |    |    |    |    |    |            |
|---------------------|----|----|----|----|----|----|----|------------|
| Water               | .. | .. | .. | .. | .. | .. | .. | 100 parts. |
| Tincture of iodine  | .. | .. | .. | .. | .. | .. | .. | 5 "        |
| Iodide of potassium | .. | .. | .. | .. | .. | .. | .. | 5 "        |

3. We must hasten healing by mild applications of nitrate of silver.



## XIII. TUBERCULOSIS OF THE BACK OF THE MOUTH AND OF THE PHARYNX.

**Description.**—The disease may be either acute or chronic. In the **acute** form the patient complains of smarting pain in the throat and a crop of yellowish granulations, which may be disseminated or confluent, promptly appear on the soft palate, the pillars of the fauces, and the walls of the pharynx. A few days later the granulations ulcerate, and give place to irregular extensive ulcers. As the lesion spreads, the mucous membrane is destroyed in places, and replaced by a pulpy surface; the uvula is swollen; the pillars of the fauces are deformed, and the tonsils are destroyed by the ulceration.

**Acute** tubercular angina causes such intense dysphagia and pain that the patient finally refuses all food. The angina is accompanied by salivation, earache, and submaxillary adenitis, which may end in suppuration. The disease lasts from six weeks to four months; death is hastened by the lesions in the lung.

In its **chronic** form the disease differs slightly, according as it follows **laryngeal phthisis** or not. When it is primary, the ulcerations are generally more discrete, like those on the tongue; they develop on the tonsils, the pillars of the fauces, and the pharynx.

When it is secondary to lesions in the larynx, the morbid process appears to spread from the larynx to the throat; the tubercular lesions invade the epiglottis, the base of the tongue, and finally become general. The duration of the chronic form is from seven to nine months, unless interrupted by acute attacks, which hasten its course. Its symptoms are at times masked by the concomitant laryngeal phthisis.

Histologically the lesions in the throat resemble those in the mouth, but changes are also found in the **adenoid tissue**, which is so abundant in this region. The adenoid follicles of the pharynx, the tonsils, and the tongue become inflamed and ulcerated.

The tubercle bacillus is present in the lesions just described. It is found in the small vessels, the giant cells, the granulation tissue, and at the side, or in the midst of the tubercular follicles.

During life it is often easy to obtain the bacillus by collecting the secretion from the ulcers, or by gently scraping the surface.

Tuberculosis of the throat is generally associated with ulcers in other parts of the alimentary canal, and, strange to say, tubercular ulcers of the mouth and the tongue do not usually accompany those of the throat, for in forty-six cases buccal tuberculosis has only been found seven times (Barth). The ulcers are generally found in the intestine. In nineteen

autopsies ulcers of the intestine have been met with fifteen times and ulceration of the anus once (Martineau).

I need not refer to the **diagnosis** of tubercular ulcers of the throat, as the question has been treated in the previous section.

It is often difficult to obtain results in acute tuberculosis of the pharynx, but in all other cases of bucco-pharyngeal tuberculosis, treatment may be very effective. Applications of tincture of iodine and iodoform have given good results, and the thermo-cautery has often limited the spread of the ulcer.

Local applications of cocaine will diminish the pain caused by the taking of food.

#### XIV. MASKED TUBERCULOSIS OF THE THREE TONSILS.

**Discussion.**—When we read the classical descriptions of tuberculosis of the back of the mouth and pharynx, we see that the authors had two principal forms of tuberculosis in view—the one acute and the other chronic—both characterized by granulations, infiltration, and more or less deep and extensive ulceration.

**Acute** tuberculosis of the back of the mouth and pharynx appears in the course of acute phthisis, or during the decline of chronic pulmonary tuberculosis. The patient complains of smarting pain in the throat, and a crop of discrete or confluent granulations of a yellowish-white colour is seen on the soft palate, the pillars of the fauces, and the walls of the pharynx. These granulations soon give place to small ulcers, which in turn give rise to more extensive and irregular ulcers. The lesion spreads, and the mucous membrane is in part replaced by a pulpy surface. The uvula, the pillars of the fauces, and the tonsils may be swollen and ulcerated. The disease causes such intense dysphagia and pain that the patient at last refuses all food, because he is afraid of swallowing. Acute tubercular angina is accompanied by abundant salivation, earache, and submaxillary adenitis, which in some cases suppurates. In its **chronic** form the disease is somewhat different, according as it is associated or not with laryngeal phthisis. We find solitary or multiple ulcers, which are of slow growth, and invade the pillars of the fauces, the pharynx, and the tonsils. When the ulcer forms, the edges are festooned, the floor is sanious, and around the ulcer a crop of yellowish points is observed. The chronic form is not always accompanied by submaxillary adenitis. It is less painful than the acute form, and its symptoms are sometimes masked by those of the concomitant laryngeal phthisis.

This brief sketch shows the varieties of pharyngeal tuberculosis described in the classical treatises. I would add that this form of tuberculosis is relatively rare, because only one case may be seen during a year of hospital practice.

There is another form of tuberculosis, however, which I consider very common. It has not been previously described, because it is quite unlike the classical forms. We find neither granulations nor ulcers; as pain is absent, it may not be discovered until it causes functional disorders, which are apparently quite benign; and yet it is a grave condition, because it may be the starting-point of extensive tuberculosis.

This almost latent form chiefly affects the adenoid tissue of the naso-pharynx. Its presence is sometimes revealed by overgrowth of the lymphatic tissue in this region—that is to say, by the hypertrophy of the palatine and pharyngeal tonsils. As regards the pharyngeal tonsil, the lesion is sometimes confounded with adenoid vegetations; and, as regards the palatine tonsils, the lesion may be taken for hypertrophy of these



organs. In many cases the tonsils present a normal aspect (Escomel). At first sight, there is nothing to show the tubercular nature of the lesion; we do not find granulations or ulcers, but only hypertrophy, which varies in degree, and causes the well-known symptoms of adenoid vegetations and enlarged tonsils. I have therefore named this form **masked tuberculosis of the three tonsils**. I made a communication concerning it to the Académie de Médecine in 1895,\* and described it in my lectures at the Faculté in 1894. In the same year Lermoyez showed at the Société Médicale des Hôpitaux a very interesting case of tubercular adenoids; he discussed the question further in an important article in 1895. The conclusions which I stated in my communication to the Académie raised some surprise, but subsequent researches have confirmed these conclusions, which have become classical. Moure and Brindel have proved histologically that in thirty cases of adenoid growths tubercular tissue was present in eight. Cornil at first considered this tuberculosis as doubtful, "but he has changed his opinion since he saw the slides prepared by Letulle from one of my cases" (Lermoyez).

The whole question was taken up again in 1902 by Jankelevitch; and Escomel of Lima, who carried out some important work in Letulle's laboratory, states as his chief conclusions:

"Tuberculosis of the tonsil is excessively frequent, and the macroscopical diagnosis of the affection bristles with difficulties. The tonsil is more liable to tuberculosis than any other organ in the body. Koch's bacilli may be met with in every part of the affected tonsil; they are, indeed, to be found in the tonsillar crypts of persons who show no tubercular lesion. Tubercular infection of the tonsil allows the bacillus to penetrate the lymphatics as well as the bloodvessels."

I do not say, of course, that every adenoid growth and every enlarged tonsil, though simple in appearance, is tubercular; but I do say that these lesions, which are benign in appearance, are very often masked forms of tuberculosis, and that in every case they form a field most favourable for the growth of the tubercle bacillus. I shall now give the experiments on which I base the above facts.

**Experiments.**—I requested several of my colleagues to be good enough to place at my disposal adenoid growths and tonsils affected with hypertrophy, which, while simple in appearance, had required the removal of these organs in patients of various ages. They kindly sent me 100 tonsils and adenoid growths, accompanied by their clinical notes. With the valuable help of Marion, portions of the tonsils and of the adenoid growths were inoculated, with every precaution, under the skin of the abdomen in several series of guinea-pigs. The portion was taken as near as possible to the centre, and not from the surface, of the tonsil or adenoid growth, to remove the objection that my experiments dealt, not with tubercular tissue, but with tissue serving as a vehicle for the tubercle bacillus.

**EXPERIMENT 1.**—On September 1 we inoculated a guinea-pig with a fragment from the centre of an enlarged tonsil removed from a child five years of age, who was healthy in other respects, and had no enlarged glands in the neck. Three weeks later a **tubercular chancre** containing Koch's bacilli appeared at the point of inoculation. The satellite glands in the groin became affected, and the guinea-pig succumbed on October 28. The post-mortem examination showed general tuberculosis, starting at the point of inoculation with a tubercular chancre as large as a sixpence. The following lesions were also found: tuberculosis of the inguinal glands and of both lungs, with cavities at the apices, as well as caseous tubercles in the spleen.

**EXPERIMENT 2.**—On October 20 we inoculated a guinea-pig with a fragment of adenoid growth removed from a child eleven years of age. She was in good health, but had abundant adenoids, with submaxillary and cervical adenitis on both sides of

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\* Dieulafoy, "Tuberculose Larvée des Trois Amygdales" (*Académie de Médecine*, séance du 30 Avril, 1895).

the neck. Three weeks afterwards a **tubercular chancre**, with Koch's bacilli, appeared at the point of inoculation. The guinea-pig died on November 28. Post mortem we found general tuberculosis, inguinal adenitis, pleuro-pulmonary tuberculosis, and effusion into the left pleura.

**EXPERIMENT 3.**—On January 16 we inoculated a guinea-pig with a fragment of an enlarged tonsil removed from a child six years of age, in other respects quite healthy, and suffering apparently from simple hypertrophy of the tonsils, without enlarged glands. On February 6, after an incubation of three weeks, a tubercular chancre, with Koch's bacilli, appeared at the point of inoculation. The guinea-pig died on February 26, and the post-mortem examination revealed general tuberculosis. On opening the thorax, we found general tuberculosis of the lungs; in the abdomen we found tuberculosis of the liver and of the spleen.

I append a summary of our experiments:

Ninety-six guinea-pigs were inoculated—61 with fragments of tonsils, and 35 with fragments of adenoid growths.

Out of the 61 guinea-pigs, 8 became tubercular, which gives an average of 12 per cent. In 6 of the cases the tubercular chancre was the initial lesion.

Out of the 35 guinea-pigs, 6 became tubercular, which gives an average of 20 per cent. In 3 of these cases the chancre was the initial lesion.

**Pathogenesis.**—We are face to face with the clearly-established experimental fact that many enlarged tonsils and adenoid growths formerly considered as simple are really a latent form of tuberculosis. Clinically it was thought that these lesions were brought about by an “unknown cause.” I feel that I have sufficiently proved that they are only too often cases of latent tuberculosis. The old names of “lymphatic and scrofulous temperament” are thus explained, when applied to children or young adults with enlarged tonsils or adenoids. They are, after all, perfectly justified, since we know the predilection of the tubercle bacillus for lymphatic tissues, and also that scrofular and tubercular lymphatism belong to the same pathological family.

We must now try to find the cause of the tuberculosis and the path by which the bacillus reaches the adenoid tissue of the three tonsils. It is convenient to speak here of German investigations, because tuberculosis of the tonsil is considered to be quite common in Germany.

In 1884 Cohnheim and Weigert drew attention to the frequency of tuberculosis of the tonsils in phthisical patients: the lesion is scarcely visible to the naked eye, and presents few clinical symptoms. Orth found tuberculosis of the tonsils in children who had died of diphtheria, the lungs being free from tuberculosis. Schenkler, in 1893, found tuberculosis of the tonsils in phthisical patients, and reported that of twenty-one cadavera on which he had made investigations the tonsils were tubercular in thirteen. He asked himself what were the relations between tuberculosis of the tonsils or the glands of the neck and the same disease in the lungs, and concludes by saying: “My opinion is that the glands of the neck become infected through the tonsils, which receive the infection from the lungs by means of



the sputum." Schenkler therefore thinks that tuberculosis of the tonsils is secondary to that of the lung.

In 1894 Krueckmann (of the Pathological Institute of Rostock) produced an excellent work on the relations of tuberculosis in the glands of the neck, the tonsil, and the lung. He says: "In the half-year from January 1 to July 15, 1894, I examined the tonsils microscopically in cases where tuberculosis was present in the glands of the neck. In each case I found tuberculosis of the tonsils, and, seeing the great importance of the subject, I thought it incumbent to publish the results." Krueckmann, like Schenkler, concludes that tuberculosis of the tonsils follows the same disease in the lungs, and is produced by means of the sputum. He quotes, however, two cases of primary tuberculosis of the tonsils due to infected food.

The German writers, therefore, had recognized the latent existence of tuberculosis of the tonsils, but they had only studied, save in a few cases, **the secondary form, in which phthisical patients had infected their tonsils by their own sputum.**

My experiments were undertaken with quite another object. I was anxious to prove that in certain patients the three tonsils are frequently the seat of **primary** tuberculosis, which is latent in character, capable of becoming general, and analogous to the latent tuberculosis of the lymphatic glands so clearly proved by Pizzini. We must consider how the bacillus enters the economy in these cases.

### **The First or Tonsillar Stage.**

A child or a youth is born of phthisical stock; he has, as a hereditary taint, a tendency to the so-called lymphatic temperament, and it is almost certain that he will show a tendency to overgrowth of the lymphatic tissue in the naso-pharyngeal cavities.

Is it not evident that these regions will offer a favourable retreat for the tubercle bacillus?

The bacillus can only reach them by the acts of respiration and deglutition. In nasal respiration the bacillus readily enters the nasal cavities. Strauss has shown the presence of active tubercle bacilli in the nasal cavities of healthy individuals who live in contact with phthisical patients. If the inhaled bacilli reach the lymphatic tissue in the naso-pharynx of a subject predisposed by heredity, they will find a field favourable to their development, and in their migration it is the adenoid tissue of the pharynx with which they first come in contact. The tissue undergoes defensive hypertrophy; if the bacillus finds a footing, we have the growth of tubercular adenoids.

I think that infection of the lymphatic masses in the naso-pharynx occurs rather from nasal respiration than from the swallowing of food-stuffs. The

proof rests on my experiments, which show that the pharyngeal tonsil (adenoid growth), which is directly concerned with nasal respiration, is invaded almost twice as often as the faucial tonsils, which are directly concerned with deglutition. In other words, the tubercle bacillus is more often inhaled than swallowed. This fact need cause no surprise, for people frequently live in an atmosphere loaded with bacilli, whilst they but rarely swallow active tubercular products.

A child who is of tubercular stock, and therefore predisposed to lymphatism, lives in close contact with tubercular parents or relatives. In this case nasal respiration gives the tubercle bacillus many chances of fixing itself in the lymphatic masses of the pharyngeal tonsil.

Alimentation, as we have said, may to some extent favour infection of the tonsils by the tubercle bacillus. The question is settled experimentally, since Chauveau caused cows to swallow tubercular material, and then found tuberculosis of the pharyngeal lymphatic tissue. Orth fed rabbits on tubercular food, and several times found tubercular lesions of the mouth and the tonsils. In 1884 Baumgarten, who made similar experiments, produced tubercular infection of the tonsils, and subsequently of the cervical glands. Cadéac induced tuberculosis in the tonsils, and subsequently in the sub-maxillary glands of guinea-pigs, by food rich in tubercular products.

During the experiments in question, it is no doubt true that the experimenters made use of food which was largely composed of tubercular matter. These conditions, though useful in experiments, do not obtain in the ordinary course of alimentation. It must, nevertheless, be said that certain foods and drinks, especially milk, whey, some kinds of cheese, raw and salt meat, may all carry virulent bacilli, and may consequently infect the soil with which they happen to be in contact, especially if it is predisposed. One cow with tubercular mastitis can infect a large quantity of milk, when all the milk is collected in the same vessel. Galtier of Lyons has produced general tuberculosis in guinea-pigs with salted or unsalted cheese ten, fourteen, and even seventy days old. The whey, separated from the cheese a fortnight before and inoculated into guinea-pigs, has produced typical tuberculosis. Galtier has proved that salt meat is virulent; the salting may not destroy the virulence of the bacillus, especially in the centre of thick pieces of meat.

We see, therefore, how the tubercle bacillus can, by respiration or by deglutition, affect the lymphatic tissue of the three tonsils.

In order to penetrate this tissue, an erosion or a pre-existing wound need not always be present. We know that the tubercle bacillus can traverse the epithelium without previous lesions, and that the tonsils, larynx, pharynx, bronchi, trachea, uterus, or intestine, may be penetrated by the bacillus, which makes its way through the epithelial cells.



The bacillus, once installed in the lymphoid tissue of the tonsils, induces multiplication of phagocytes, which often leads to considerable hypertrophy of the tonsils. Sometimes, however, the reaction is almost wanting, and the tonsils remain practically normal in size.

Tuberculosis of the tonsils is often painless, but it does not by any means always pass unnoticed. Many children or adolescents complain of "sore throat" when they catch cold or take the slightest chill. Acute tonsillitis, with or without enlarged glands, often supervenes. The case is looked on as simple acute tonsillitis, and there is an apparent return to the normal condition. The tonsils and the adenoids, however, remain enlarged and cause discomfort.

In many cases, fortunately, the disease does not proceed beyond the first stage. After a variable length of time the phagocytosis gains the upper hand. The tonsil becomes fibroid (Grancher), indurated, and atrophied. Here, as in all cases of local tuberculosis, recovery is effected, and the infection does not become general.

### Second or Glandular Stage.

In many cases, however, the first stage is, unfortunately, passed. The bacillus remains for a variable period in the tissue of the tonsils, and then, as the result of secondary infection, enters the lymphatic network, which drains the three tonsils, the **second stage** betraying itself by the appearance of glands in the submaxillary and cervical regions.

Durochonsky actually observed the migration of the tubercle bacillus into the lymphatic vessels which lead from the tonsils to the glands of the neck.

One fact is worthy of notice—namely, that larval tuberculosis of the tonsils reacts more surely and more frequently on the lymphatic glands than the large tubercular ulcers of the bucco-pharyngeal cavity. I have often noticed this fact, which other authors have also described (Reclus, Péan). Tubercular ulcers of the tongue or of the throat, even though large and deep, may not lead to infection of the glands, while a tubercular lesion of the tonsil, though apparently trifling, induces considerable enlargement of the cervical glands, which seems at first to have no connection with the masked cause that has given rise to them.

When once the lymphatic glands of the neck are invaded, the lesion may be limited to a small number of glands, or else the whole chain of superficial and deep glands may be affected. The trouble is always **descending**. It commences in the upper cervical glands with which the lymphatics of the three tonsils communicate, and gradually descends, until it may invade the superficial and deep glands of the whole cervical and clavicular region.

In some cases the glands are small, hard, painless, and separate, but in

others they are enlarged, painful, and matted together. The disease may sometimes improve or disappear, but in other cases the glands break down and suppurate. This condition was called **king's evil**. We may then find fistulæ and chronic suppuration of the superficial and deep glands, ending in scars, which occupy the submaxillary, submental, premastoid, retromastoid, and subclavicular regions, and are the indelible witnesses of what was formerly called scrofula.

Scarlatina, whooping-cough, influenza, syphilis, and angina of every kind, play a part in the extension of the process, and in the formation of pus.

Pizzini has shown that tubercular adenitis may persist indefinitely in a latent state, until a secondary infection brings about the dissemination of the bacillus. The second or glandular stage does not always run a progressive course. Tuberculosis of the cervical glands may end in recovery. An individual who in childhood or youth has suffered from adenitis of the glands in the neck, caused by larval tuberculosis of the tonsils, may recover from the glandular lesions, and escape general tuberculosis.

We are not yet sufficiently familiar with these latent forms of tuberculosis, which may remain in the glands or the tonsils for an indefinite length of time, and be quite harmless. They may, however, at a given moment increase in virulence, and lead to general infection.

A person suffering from this descending adenitis, then, does not of necessity become phthisical, but it can never be stated at what moment the possibility of his becoming so will cease.

### Third or Pulmonary Stage.

When the bacillus reaches the **lung**, we have the **third stage** of tuberculosis of the tonsil. The bacillus may finally reach the great lymphatic canal or thoracic duct, whence it is launched in the venous circulation, passes through the right heart, and finally reaches the lung, causing pulmonary tuberculosis.

The bacillus may enter the lung in such small numbers that the disease can be arrested. The lung defends itself, and replies to the attack of the bacillus by **defensive** hæmoptysis. In other cases the disease follows its course, and ordinary phthisis results. Finally, the bacillus may enter the lung in such large numbers as to cause acute tuberculosis.

The interval between the pulmonary and glandular stages varies. The disease may remain latent in the gland for many years, and the lung is only invaded very late. The cases which I have published in the *Bulletin de l'Académie* show that the lung infection appeared after three months (Case 1); five months (Case 2); four years (Case 3); six years (Case 4); ten years (Case 5); sixteen years (Case 6); and twenty-eight years (Case 7).



CASE 1.—(Three months' interval between the glandular and pulmonary stages.) Ch——, eighteen years of age, admitted into the Necker Hospital under my care. Six months before two glands at the angle of the jaw were removed at the Beaujon Hospital. Other glands have since appeared, forming a descending chain, best marked in the left cervical region. The glands were as large as a filbert or a walnut; several of them were adherent, soft, and suppurating. Bacilli present in the glandular fluid.

The man, who had never been ill, commenced to cough three months previously. At the same time he had attacks of hæmoptysis, which were the first symptoms of the pulmonary stage, and appeared three months after the glandular stage. Auscultation revealed incipient tuberculosis at the apex of the right lung.

CASE 2.—(Five months' interval.) In consultation with Dr. Gérard Marchant, I saw a young girl, thirteen years of age, suffering from phthisis, with numerous bacilli in the sputum. The cough and other symptoms of tuberculosis had set in about two months previously.

Five months prior to these pulmonary symptoms she had been attended by Marchant for acute inflammation of the maxillary and cervical glands on both sides (glandular stage). The inflammation had been marked, and pus had formed in the glands.

It is very interesting to note that the glandular trouble had been preceded two months previously by hypertrophy of the tonsil (tonsillar stage).

CASE 3.—(Four years' interval.) A young man, twenty-five years of age, was admitted to the Necker Hospital under my care on August 1, 1894. Until he joined his regiment four years before, he had never shown any chest symptoms. In 1890 the submaxillary glands showed such marked swelling during the winter that Le Fort removed them. Some small superficial glands were not removed. He was able to complete his military service.

Five months previously, in April, 1894, cough, expectoration, and two attacks of hæmoptysis appeared. Loss of weight and night-sweats followed, and we found advanced tuberculosis at the apex of the right lung and incipient disease at the apex of the left.

CASE 4.—(Six years' interval.) In consultation with Dr. Blocq, I saw a patient, aged twenty-four, suffering from advanced tuberculosis of the right lung. At the age of seventeen suppurative adenitis of the left submaxillary glands, which were removed; no chest symptoms.

Six months later suppuration in the left cervical glands, which were removed.

Some months later suppuration in the supraclavicular glands on the same side; the glands were removed.

Some months later suppuration in the glands on the right side, which were also removed.

It was only after these successive attacks of descending adenitis that pulmonary symptoms appeared.

CASE 5.—(Ten years' interval.) A young man, twenty years of age, admitted to the Necker Hospital. Between the age of ten and twelve years he was subject to sore throat, and later, when about seventeen, suffered from double tonsillitis.

For many years glands had appeared in the cervical region; as they grew larger and suppurated, operation became necessary.

On the right side the glands were soft and of the size of a walnut, while those on the left side were larger. Fistulæ and cicatrices present on the skin of the cervical region.

The pulmonary stage did not appear till ten years after the glandular one. Tubercular bronchitis was present, though the patient had had no previous cough, and we found on examination phthisis of the left apex. Marion discovered bacilli in the pus from the glands and in the sputum.

CASE 6.—(Sixteen years' interval.) In 1894 I saw a patient, aged thirty-six, suffering from early phthisis of the right apex. The cough and the slightly blood-streaked sputum appeared to be recent, but the glandular stage had commenced sixteen years

before. In 1877 he had an attack of progressive adenitis in the glands on the left side of the neck. The adenitis ended in suppuration, which lasted for several months. I found two large cicatrices—one below the jaw and the other on the posterior edge of the sterno-mastoid muscle.

CASE 7.—(Twenty-eight years' interval.) A patient, forty-eight years of age, whom Potain, in consultation with me, had seen several times, was taken ill with hæmoptysis and pulmonary tuberculosis in May, 1893. The latter ran a somewhat rapid course, and the patient succumbed a year later. Though he had never had any symptoms of phthisis, he had in his youth suffered from suppuration of the cervical glands. He had large cicatrices. After a latent period generalization occurred, and the glandular stage ended twenty-eight years after in the pulmonary stage.

CASE 8.—Quite recently I had at the Necker Hospital a most typical case of this tubercular infection in a young woman. She married a tubercular husband, who died in a few years of phthisis. She was attacked by hypertrophy, which, though simple in appearance, was in reality larval tuberculosis of her three tonsils. In spite of energetic treatment, the cervical glands had been invaded about a year previously, and a few months before the signs and symptoms of phthisis had supervened. The apex of the left lung was tubercular.

How many cases of this kind have I not met with since my attention was called to this mode of propagation in tuberculosis! It is surprising that this fact has not been demonstrated earlier by experiment.

The invasion of the lung is revealed by the ordinary signs of tuberculosis—namely, hæmoptysis, suspicious bronchitis or pleurisy, wasting, loss of appetite and of strength, etc. Percussion, auscultation, and examination for bacilli readily indicate the seat of the disease.

To sum up: It is truly said that the lymphoid tissue of the three tonsils offers, especially in young and predisposed subjects, an entrance and a sure asylum to the tubercle bacillus. The result is a latent form of tuberculosis, which simulates adenoids, or hypertrophy of the tonsils. Frequently the tonsil is not even enlarged. This **masked tuberculosis of the three tonsils** may not overstep the first stage, and may end in recovery.

In other cases the infection, at first limited to the tonsils, invades the lymphatics of the neck. Numerous varieties of tubercular adenitis are the result. This glandular stage may end in recovery without other complications.

Too often, however, after a period which varies from some months to several years, the second stage is passed, and the tuberculosis affects the lung, or becomes general. The three tonsils must therefore be reckoned as one of the **most formidable portals of entry** in human tuberculosis.

**Treatment.**—The first point is immediate recourse to prophylactic measures. In practice they consist in preventing the entrance of the bacillus during respiration and alimentation.

Children must therefore be removed from all sources of contamination, and the most important source is the dust of the dried sputum. It is necessary, as far as possible, to change the infected atmosphere, in which



the child lives for an inoffensive medium. I know well that the matter is easier to discuss than to accomplish, but in matters prophylactic we must always return to the question of the seed and the soil. Heredity in particular provides the soil, and it is our duty to keep the seed away from it. Alimentation may be, to a certain extent, a source of infection. From the experimental point of view the question is settled, and I urged this when considering the modes of entrance of the bacillus. Hygiene, in the case of a predisposed child, must therefore consist of many precautionary measures.

And even though the seed have germinated, and the bacillus have passed the tonsillar stage, certain prophylactic measures must be employed. It is often only through secondary infection that tuberculosis, hitherto latent, reveals itself in a subject who seemed secure from harm. Tuberculosis often seems to break out after measles, whooping-cough, scarlatina, influenza, typhoid fever, or syphilis, when these infectious diseases are only the occasional cause of a hitherto concealed tuberculosis. Children and young lymphatic subjects born of tubercular stock, whose adenoid tissue is already affected, must be therefore carefully preserved from the infectious diseases which I have just enumerated.

Let us now consider curative measures. What treatment are we to adopt in a case of masked tuberculosis of the tonsils? Before approaching this question, which is both medical and surgical, I must confess that the means at our disposal are not always efficient. The time will come, no doubt, when, with the aid of serotherapy, we shall be able to fight tubercular infection, as we fight diphtheritic infection, and as we are beginning to fight streptococcal infection. Until then let us employ the means at our disposal, and see which appear to be the best.

Let us commence with the general treatment, which aims at placing the system in a state of defence. Our means prepare the body for the fight, and sometimes help it to gain the victory. Personally, I am sure that the best treatment is to give food rich in fatty substances, such as cod-liver oil, caviare, sardines in oil, tunny-fish, pâté de foie gras, slices of bread and butter, etc.

**Hygienic** measures occupy an important place in cases of tonsillar and glandular tuberculosis. Sea air has a powerful action. Out of 1,293 cases of enlarged cervical glands admitted into the hospital at Berck, the glandular inflammation disappeared in 900 cases. The Arachon cure and the waters of Salies-de-Béarn, Salin, or Kreuznach, are all means to the same end.

**Surgical** treatment remains to be discussed. What course should be adopted in a case of enlarged tonsils or of adenoid growths? Ought they to be removed, or destroyed by the galvano-cautery? Opinions are divided on this point. Though I have several times proved the necessity for surgical intervention, great judgment is required, and each case must be treated on its merits.

## CHAPTER III

### DISEASES OF THE ŒSOPHAGUS

#### I. ŒSOPHAGITIS.

**Pathological Anatomy—Ætiology.**—Œsophagitis, or inflammation of the œsophageal mucous membrane, may be primary or secondary. The **primary** form is always caused by the ingestion of hot or corrosive liquids (sulphuric acid, solution of potash), of irritating substances (tartrate of antimony), or by the presence of a foreign body (fragment of a bone, fish-bone, pin). **Secondary** œsophagitis develops by the spreading of thrush or of diphtheritic angina; it supervenes in the course of some other diseases (variola, typhoid fever).

The lesions are most common in the upper third of the œsophagus, and differ greatly according to their cause. The mucous membrane is red, thick, eroded, or ulcerated; sometimes, indeed, the walls of the œsophagus are perforated. When suppuration occurs (which is very rare), the **abscess** may be either submucous or peri-œsophageal. In the latter case the suppuration may invade the cellular tissue and cause a cervical abscess. Scarring of the mucous membrane frequently results from the ingestion of caustic substances, and is often followed by **stenosis** of the œsophagus. In addition to the causes which I have just enumerated, the simple ulcer and syphilis also produce cicatricial stenosis.

The simple ulcer may occur in the œsophagus, as in the stomach or duodenum, and cause a fibrous stenosis (Debove). A patient who had cicatricial stenosis of the œsophagus, which was rightly attributed to a simple ulcer, died two years afterwards from perforation of a gastric ulcer. Post mortem the gastric ulcer, which was the cause of death, was found. An ulcer was also present in the cicatrized œsophagus 2 inches above the cardiac orifice; the cicatrix was circular,  $\frac{1}{4}$  inch across, and of the same aspect and nature as the simple ulcer of the stomach. The œsophagus, which had been dilated by sounds for two years, was somewhat contracted at the cicatrix and slightly dilated above it. **Syphilis** may cause cicatricial contractions of the œsophagus, which result from ulcerated gummata.

**Description.**—The initial symptoms are only marked when the in-



flammation is very acute. Pain may be felt along the œsophagus, in the epigastric region, or between the shoulders, and is caused by the passage of food and of hot or cold liquids. It is generally accompanied by **spasms** of the œsophagus and regurgitation of ingested substances.

**Foreign bodies** in the œsophagus may produce, not only inflammation and ulceration, but also perforation of the œsophageal vessels, followed by profuse hæmorrhage. **Caustic substances** cause ulceration, sloughing, and perforation, which may be followed by death; and, if the patient recovers, the cicatrices usually induce **stenosis** of the œsophagus. The formation of an **abscess** is announced by febrile symptoms, with increase of pain and dysphagia. If the abscess is submucous, the pus may be rejected by the mouth, or may pass into the stomach. If it is peri-œsophageal, the pus burrows into the neighbouring tissues, and produces very grave mischief.

The symptoms of **cicatricial contraction** of the œsophagus at first closely resemble those due to malignant stricture. I shall therefore discuss this question under Cancer of the Œsophagus.

Soothing drinks, applications of leeches to the painful region, and injections of morphia, are employed to relieve the pain and the inflammation in acute œsophagitis. Catheterization of the œsophagus in order to dilate the stricture is the usual treatment for cicatricial contractions. Œsophagotomy should be reserved for special cases. In the case of syphilitic stricture the patient must be put on a course of iodide of potassium and mercury.

## II. SPASM OF THE ŒSOPHAGUS.

**Description.**—**Spasms** of the œsophagus are often described under the name of **spasmodic stenosis** of the œsophagus. These spasms are of multiple origin, being **idiopathic** in hysteria, in hypochondriacs, and nervous people; **symptomatic** in the case of œsophageal lesions (foreign bodies, cancer); and **sympathetic** in some ill-determined cases (tænia, uterine affections). It is the **idiopathic spasm** which I have particularly in view in this description.

Spasm of the œsophagus comes on suddenly at the sight of food, with the first mouthful, or during the course of a meal. Emotion, annoyance, or some particular kind of food may provoke it. If the dysphagia is complete the food does not pass and is regurgitated, or if it should pass, it is only when it is crumb-like, well moistened, and slowly swallowed. The spasm is generally accompanied by choking and a painful feeling of constriction in the upper third of the œsophagus. By the use of the catheter the presence and the seat of the spasmodic contraction are proved: the instrument is sometimes arrested at the level of the spasm; at other times it goes through after some difficulty. Transient spasm is not dangerous; serious anæmia and weakness may follow persistent spasm.

The **diagnosis** of spasmodic contraction from cancer of the œsophagus will be given in the next section. The **treatment** consists in the use of the catheter. Bromide of potassium, valerian, and belladonna, are also administered. Hydrotherapy may be employed.

### III. CANCER AND STRICTURES OF THE ŒSOPHAGUS.

**Pathological Anatomy.**—The varieties of cancer of the œsophagus are, in order of frequency: epithelioma, encephaloid, and scirrhus. The middle third of the organ is the most common seat of cancer, and next the lower third. The growth occurs in the form of a patch or of a projecting tumour, which surrounds the œsophagus like a ring over an area of half an inch or more, and gradually narrows the lumen. The œsophagus is almost always **dilated** above the tumour and **contracted** below it. The stricture is rarely cylindrical; it is usually oblique and anfractuous. The **dilatation** above the tumour may form a pocket or a lateral diverticulum; this is peculiar to malignant strictures, and is hardly ever seen in those of inflammatory origin. The internal surface of the cancerous œsophagus is sometimes normal, and the mucous membrane preserves its integrity (scirrhus). At other times it is ulcerated or covered with fungating growths, the mucous and submucous coats have disappeared, and the tunica muscularis is thickened.

The trachea, bronchi, lungs, pleura, aorta, glands, and vertebral column may be invaded by the growth, and we find adhesions, ulcerations, or perforation, with or without fistulæ, which establish communication between these different organs and the œsophagus. The Dupuytren Museum has some excellent specimens, showing communications between the œsophagus and the neighbouring organs.

**Symptoms.**—Pain and dysphagia are the first symptoms of cancer of the œsophagus. The pain, however, is not constant; it consists in a feeling of constriction behind the sternum or between the shoulders, becomes worse at meal-times, and may be very sharp, but the radiation is not always sufficient to localize the seat of the disease. Progressive dysphagia is the chief symptom of œsophageal cancer. The troubles in deglutition are due to the thickening and induration of the gullet, and often to the gradual narrowing of its calibre. The patient instinctively takes smaller mouthfuls as soon as he feels discomfort in swallowing, and takes frequent sips to facilitate the passage of the food bolus. For some time the food passes fairly well if it is well masticated. This condition lasts weeks and months, with alternate improvement and aggravation, but later the dysphagia becomes more marked, until at last even liquids are arrested at the stricture. Both solid and liquid foods are frequently regurgitated.



The foregoing description holds good in most cases. In many instances the dysphagia associated with cancer of the œsophagus is progressive and continuous, and, while slight at first, it gradually reaches its maximum as the lesion progresses. Events, however, do not always take this course, and several cases have been collected in which the dysphagia was sudden and acute from the outset, just as in simple spasm. In some patients the dysphagia comes on suddenly during the swallowing of a large mouthful, or in consequence of vomiting caused by a hearty repast. The dysphagia is almost absolute for two or three days, and then the normal condition returns, deglutition being easy for several weeks, until a fresh attack comes on, the dysphagia taking on this intermittent form until it becomes continuous. It would be quite wrong to rely on the sudden or acute onset of the dysphagia, or on its temporary disappearance, in order to eliminate the idea of cancer and show that a case of stricture is simply due to spasm. Spasm of the œsophagus plays a large part in the dysphagia, even when organic lesions are present.

No matter what are the symptoms at the commencement, if the cancer terminates in stricture of the œsophagus, the food passes with difficulty and is regurgitated. The **regurgitation** follows immediately on the ingestion of food when the constriction is in the upper part of the œsophagus; it occurs later when the growth is seated in the lower portion. This delay is favoured by the dilatation of the tube, which forms a kind of pouch, where the food rests for a time. In the latter case food is rejected some time after ingestion, as a pulp-like foul-smelling mass. It has undergone but little modification by the saliva. The accumulation of food in the pouch causes a feeling of pain, which may give rise to choking, and only ceases after vomiting. The vomit is at times mixed with blood, blackish clots, and detritus from the fungating growth.

Some patients have **salivary regurgitation**, or “fits of mucous vomiting.” Glue-like masses of mucus, which the patient removes from his mouth with his finger, accumulate at the level of the stricture, and are brought up in the morning on awakening or prior to the regurgitation of food. Amongst the symptoms of cancer of the œsophagus there have been noted **hiccough** (Mondière) and the “gloo-gloo” sound (Béhier), which occurs when the air swallowed with the food passes the stricture. Alteration in the voice and paralysis of a vocal cord from destruction of one recurrent nerve have also been noted. Enlargement of the glands above the clavicle is a valuable but inconstant sign.

In some cases, particularly in old men, cancer of the œsophagus is latent, pain, dysphagia, and regurgitation being absent; so that we should think of cancer of the œsophagus in a patient suffering from malignant cachexia, when the site of the growth cannot be discovered.

**Complications.**—The disease lasts from one to two years, and the prognosis is hopeless. When there is no constriction, and the patient continues to take food, cachexia may appear late; but when the cancer ends in stenosis, the loss of weight progresses, and the cachexia is earlier, as it depends both upon the presence of the cancer and the insufficiency of food. Death is, however, not always due to cachexia, but is often caused or hastened by various complications.

In purely cicatricial stenosis the danger arises only from the inanition which may result, but in stenosis due to cancer the complications, which depend on the seat of the cancer and its propagation to neighbouring organs, are added to the dangers of stenosis and inanition. Amongst the most frequent complications I shall quote compression and perforation of the trachea and the bronchi. The perforation is sometimes direct, at other times fistulous, involving the trachea alone, or the trachea and the bronchi at the same time. This complication is announced by fits of coughing and suffocation, which come on as soon as food or drink enter the respiratory passages.

Other complications comprise: communication of the cancer with the pleura and lung, especially on the right side (Vigla); pleuro-pulmonary inflammation; perforation of the pericardium or of the aorta; and the invasion of the vertebræ. According to some authors, constrictions of the œsophagus, whether they are cicatricial or cancerous, are apt to favour tuberculosis by inanition.

**Diagnosis.**—As soon as a patient shows symptoms of constriction of the œsophagus, we must make certain that it is a case of constriction, and not of compression by a tumour in the neighbourhood (cancer of the mediastinum or bronchial glands, aneurysm of the aorta or of the subclavian artery). The signs obtained by auscultation and percussion and the various symptoms inherent in each of these diseases generally clear up the point. When we have proved constriction of the œsophagus, the next question is to decide whether it is spasmodic, cicatricial, or malignant.

1. Let us commence with the diagnosis from spasmodic constriction. I have described in the second section the characteristics of **spasmodic constriction**. This spasmodic constriction, which is usually found in hysterical, nervous, or hypochondriacal patients, does not come on gradually; it appears suddenly, and at once reaches its maximum. In spasmodic constriction the symptoms sometimes disappear suddenly, and return some days, weeks, or months later. They often recur from mental causes, or from the use of certain foods and beverages. In cicatricial or cancerous constriction these symptoms are not often seen, but we must not forget that organic lesions of the œsophagus, as of all muscular canals, before causing persistent organic constriction may give rise to spasms of greater or less duration,



which simulate spasmodic constriction. Such is the case in cancerous constriction. On the other hand, spasmodic constriction may, from its duration and its cachexia, simulate malignant stricture, and writers have recorded cases of purely spasmodic constriction which, like the permanent contractures of hysteria, last for weeks at a time, and bring about a state of inanition and emaciation which might give rise to the idea of cancer. It will therefore be seen that dysphagia alone is often insufficient to prove that a stricture is malignant, and that hysterical œsophagismus, in order to have an absolute diagnostic value, must be associated with other symptoms common to hysteria.

In certain rare cases the spasm comes on in fits, which are due to stimulation of the recurrent nerve by a neighbouring tumour. This symptom belongs to the category of spasmodic constrictions described under Aneurysms of the Aorta of the Recurrent Type.

1. Let us now consider the diagnosis of cancerous and cicatricial constrictions. Certain symptoms aid us in distinguishing between these two forms. I shall divide cicatricial constrictions into three varieties. In the first variety the constriction is due to the ingestion of corrosive substances. Béhier has collected a large number of cases in which the patients had swallowed liquor potassæ, nitric acid, or sulphuric acid, and had, in consequence, suffered from cicatricial constrictions, which were sometimes multiple, and more or less extensive. In these cases, however, the symptoms of constriction are preceded by **acute œsophagitis**. This acute stage, with the information given by the patient, proves the case. Several constrictions may be present in such cases at various points of the œsophagus, and as they may be some inches in extent, catheterization is very difficult.

2. In the second variety the constriction is due to a **simple ulcer** of the œsophagus, which is identical with the simple ulcer of the stomach and duodenum. Its cicatrization may lead to constriction of the œsophagus, as was evident in Debove's case, where the ulcer and the consequent constriction were situated 2 inches above the cardia. In such cases, however, the dysphagia and the symptoms of constriction are preceded by symptoms resembling those of ulcer of the stomach. Sharp pains are present at the pit of the stomach, and in the vertebral region, they radiate to the shoulders, around the thorax, or in other directions. Vomiting of blood, accompanied by pain, is the second prominent symptom of ulcer of the œsophagus. The hæmorrhage consists of red rather than of black blood. It may be profuse and recurrent. This first phase, which may last months and years, is succeeded by dysphagia and symptoms of stricture. These symptoms appear slowly, and the difficulty in deglutition increases gradually, until the catheter is passed, and the seat of the constriction is

established. The succession and the course of these symptoms allow us to eliminate the idea of cancer.

3. The third variety of cicatricial constriction is due to syphilis. We find neither the acute stage seen in corrosive strictures, nor the pain and hæmorrhage which precede constriction due to a simple ulcer. Cases of syphilitic stricture are rare, and Potain has been able to collect only seven cases. Virchow found breaking-down gummata in the scar of a stricture. Lubinski cites two cases of syphilitic constriction. In the first case a young man, twenty-nine years of age, had been syphilitic for some years, and the stenosis, which was almost complete, was probably due to an ulcerated gumma, and developed in about three weeks. Specific treatment brought about rapid improvement. Palmar psoriasis confirmed the diagnosis. The second case is that of a man who had had syphilis of the tongue. In syphilitic constriction the diagnosis will be made by the process of elimination. The constriction takes place either quickly or slowly, and is not preceded by the preliminary stage noticed in the other varieties of cicatricial constriction, and the patient has already had syphilitic troubles.

As we have just seen by this lengthy discussion of the symptoms, it is by elimination that the cancerous nature of the lesion will be recognized in difficult cases. It is at first difficult to diagnose, but the age of the patient, conditions of heredity, and careful study of the preliminary stage, will most frequently allow us to say whether it is a case of cancer or not.

In making a diagnosis, cachexia must never be relied on, for it may appear late, if nourishment can be taken. Vomiting of blood-stained matter mixed with particles of food is in favour of cancer.

The **use of the catheter**, which must be carried out with the greatest care, in order to avoid false passages and perforations of the canal, may furnish valuable information. In the case of spasmodic constriction serious resistance is sometimes met with. We can, however, pass a large olivary catheter more easily than a small one, and when the instrument has been passed the patient can usually swallow without difficulty—at any rate, for the first few moments. In cicatricial stricture, especially when annular, the catheter passes with a jerk, as through an obstacle which does not give way. On the contrary, the catheter when passing a cancerous constriction seems to go through a less rigid obstacle, which is more yielding, and it is often streaked with blood on withdrawal, even though the manipulation has been carried out with dexterity. In some cases the presence of supraclavicular glands or the appearance of phlegmasia alba dolens will help to prove the existence of cancer.

**Treatment.**—In the cicatricial strictures the use of the catheter has a twofold object: on the one hand, it is possible to feed the patient, and, on



the other, it leads to the gradual dilatation of the constriction. It is often preferable to perform gastrostomy, in order to feed the patient and allow time for systematic catheterization. I did this for a patient of mine and the result was excellent.

When there is a malignant stricture, a gastric fistula must at once be made ; the patient is fed through it (gastrostomy), and in a few weeks gains several pounds in weight, while life may be prolonged considerably.

I have seen several cases of this kind in patients of mine operated on by Routier and Marion.

## CHAPTER IV

### DISEASES OF THE STOMACH

#### I. GASTRIC DISTRESS—ACUTE CATARRHAL GASTRITIS.

THE terms “gastric distress” and “gastric fever” have been variously interpreted by different writers, because each has the drawback of referring to an ill-defined morbid condition. Some authors consider catarrhal gastritis as a local infection, whilst others class it amongst the group of fevers. It seems easy to me to reconcile these contrary opinions.

**Gastric Distress—Catarrh of the Stomach.**—The conditions united under the name of gastric distress or acute gastric catarrh may be the result of a local trouble; or the expression of a general condition, which is quite different. If an individual takes a heavy meal, drinks too much wine, eats rich food, alters his course of living completely for a few days, smokes too much, or keeps late hours, he will suffer from headache, loss of appetite, distaste for food, nausea, and vomiting. His tongue will be thick and coated, and he may have slight fever. This condition is due to local gastric trouble, and this acute catarrh of the stomach cannot be mistaken for the manifestation of a general condition. In other cases, in consequence of the ingestion of tainted meat, of “high” game, or indigestible food, the gastric disorder is the result of **auto-intoxication**. The digestion is impaired by the defective production of hydrochloric acid, fermentation occurs, and the absorption of toxic substances causes gastritis, with its train of symptoms (Bouchard). The ingestion of bad food may cause fatal ptomaine-poisoning (Brouardel and Boutmy).

The distress sometimes appears under quite different conditions. A person, without any appreciable cause, is taken ill with rigors, malaise, headache, and perhaps epistaxis. In the meanwhile the digestive troubles which I have just described supervene—complete anorexia, pain in the pit of the stomach, nausea or vomiting, constipation, ballooning of the abdomen, etc. The patient has acute catarrh of the stomach with fever, but in this case the trouble has been preceded by a period of invasion, and is accompanied by unmistakable symptoms. The remittent nature of the fever, the rapid loss of strength, the possible appearance of an exanthem,



and the slowness of convalescence, are evidence of a morbid condition in which the digestive troubles form only a part.

**Gastric Fever.**—Febrile gastritis owes to the seasons, to localities, or to the **medical constitution** of the moment, certain characteristics which permit the description of certain varieties. It is chiefly in spring and autumn, under the influence of sudden changes of temperature and atmosphere (changes which our predecessors called **circumfusa**), that we meet with epidemic gastritis. I shall sketch the principal types, some of which at least belong to the class of **infectious diseases**.

Acute catarrh of the stomach may last a few days, and, as it often appears in the remittent form, the disease has been called **remittent gastric fever** by some authors. In some cases it is accompanied by excessive secretion of bile (polycholia), and is then called **bilious gastric fever**. The skin and the conjunctivæ are yellowish, the hepatic region is tender, the liver is sometimes enlarged, the vomit contains bile, and the stools are liquid, fœtid, and often bilious. The pulse is soft, the headache is intense, and the fever is clearly remittent. This type of fever, which may be observed in the Mediterranean region, and more often in tropical countries, is generally the result of malarial poisoning.

In other cases gastric fever, especially in our climate, occurs in epidemic form, and is accompanied by headache, epistaxis, insomnia, intestinal troubles, and diarrhœa, so that the diagnosis is very difficult at first between these cases and typhoid fever. Certain writers have expressed the opinion, which I share, that this gastric fever is a mild form of typhoid fever. A fact in favour of this view is that such cases generally appear at the commencement of epidemics of typhoid fever. After all, the diagnosis, formerly so difficult, is now simplified by Widal's reaction.

**Prognosis—Treatment.**—The prognosis is not grave. The symptoms, however, may be complicated by palpitations, dyspnœa, or fits of choking, which are due to dilatation of the right heart. This dilatation is passive, due to the influence of gastric troubles, and disappears with them (Potain).

The **treatment** consists chiefly in the use of purgatives. In simple catarrh of the stomach saline purgatives are sufficient. Sulphate of soda is given for two or three days in half-ounce doses in vegetable broth; Birmenstorff water, Pullna water, etc., may be given. If the condition is more severe, we must employ emetics, such as ipecacuanha or ipecacuanha and tartar emetic, and the next day a saline purgative. The patient is dieted, and according to circumstances, is allowed acid drinks, cold broth, and cold milk, with Vichy or other alkaline water (Vals or Saint-Galmier).

## II. ACUTE GASTRITIS.

Acute, phlegmonous, and toxic gastritis demand notice here. Appendicular ulcerative gastritis will be dealt with in a separate section.

**Acute gastritis** is acute inflammation of the mucous membrane of the stomach. The mucosa is inflamed, and is sometimes the seat of erosions and superficial ulcerations. The vessels are dilated and form a fine network, while the glands are enlarged and the stomach is covered with mucus. The causes given under Gastric Catarrh apply to this form. The onset is sudden, and fever may be present; the patient complains of pain in the epigastric region, vomiting of mucus and bile comes on, and is accompanied by pain. The anorexia is complete, the thirst is great, the mouth is dry, the tongue is red or coated at times, and the urine is scanty and high-coloured. Cough is fairly common; it is frequent and dry or succeeded by the rejection of mucus from the stomach.

Acute simple gastritis is not dangerous, as complications do not occur, and the patient recovers after a convalescence which requires care.

**Phlegmonous Gastritis.**—This form of gastritis, which is also called **submucous gastritis**, is characterized by the formation of pus in the submucous tissue of the stomach. The pus may be spread out in more or less extensive sheets, or form abscesses varying in size from a lentil to a walnut. The abscesses are most common at the pylorus, and may be submucous or subperitoneal. The former may open into the stomach by one large opening or by several apertures, which are the orifices of the glands; and the latter may cause acute peritonitis, unless adhesions have previously formed between the stomach and the peritoneum.

The **symptoms** are those of acute gastritis, but the pain is severe, the vomiting is acute, and the fever is high; while dyspnoea, prostration, and icterus are also seen. Death may supervene before the opening of the abscess. If it opens into the stomach, the contents are vomited; if it opens into the peritoneum, it causes acute fatal peritonitis. The disease may end in recovery, but the scar-tissue sometimes causes stricture of the stomach or of the pylorus.

Phlegmonous gastritis may be primary or secondary, the latter form being observed in the infectious diseases (smallpox, pyæmia, or typhus).

**Toxic Gastritis.**—The mineral acids and caustic alkalis destroy the tissues of the stomach, but do not cause inflammation in the true sense of the word. Arsenic, phosphorus, the silver salts, and vegetable poisons cause necrosis of the coats of the stomach, but only after an acute inflammatory stage. We find hæmorrhage, ulceration, perforations, and eschars which may affect the mucous membrane or all the coats of the organ. The mouth, pharynx, œsophagus, and intestine show traces of the passage of the poison.



The symptoms are sudden, and their severity depends on the nature of the poison. Acids and caustic alkalis cause agonizing pain and absolute intolerance of the stomach, while the vomit often contains blood. The pulse is small, the skin is cold and covered with sweat, and the patient dies of collapse or of superacute peritonitis. When recovery does occur, it is only after a tedious convalescence, followed in some cases by stenosis of the œsophagus, stomach, or pylorus. When the patient has swallowed toxic substances which act on the whole organism (arsenic, phosphorus, vegetable poisons), the gastritis and the poisoning demand relief.

**Treatment.**—In simple acute gastritis leeches may be applied to the pit of the stomach, and the diet carefully regulated. The patient may suck ice; he may take cold drinks or small quantities of cold milk to which lime-water and cocaine are added. An ice-bag or cold compresses are applied to the epigastrium. The pain is relieved by injections of morphia. Similar measures may be employed in phlegmonous gastritis. The first indication in the case of toxic gastritis is to get rid of the poison by vomiting at the earliest possible moment, and then to administer an antidote. Treatment is only effective when it is applied soon after the intoxication.

### III. CHRONIC GASTRITIS.

**Ætiology.**—Chronic gastritis often follows acute or subacute gastritis, and even when it seems to be chronic from the first, it is preceded by dyspeptic symptoms, which indicate its gradual onset. Of all the causes which I have enumerated under Acute Gastritis, and which I might again mention in connection with chronic gastritis, alcoholism is the most important and the most frequent. The abuse of alcoholic drinks, especially of those of inferior quality, is the usual cause of chronic gastritis. By alcoholism I do not refer only to the excessive use of alcoholic drinks, but I also allude to the excess of alcohol taken by people who never get drunk, but who in the course of years poison themselves slowly, and so contract gastritis and cirrhosis. Amongst other contributory causes I may mention gout, tuberculosis, and most of the infectious diseases.

**Pathological Anatomy.**—The mucosa is red, pigmented, studded with hæmorrhagic erosions, thick and smooth, or mammillated. It sometimes has a polypoid appearance, especially at the pylorus. The submucous and muscular coats are thickened, giving a special firmness and resistance to the walls of the stomach. This hypertrophic sclerosis of the submucous layer is the chief lesion in some cases. Under the microscope these lesions are seen to be **glandular** and **interstitial**. As far as the glands are concerned, the epithelium is granular; the cul-de-sac is granulo-fatty, and some glands have undergone cystic dilatation.

The cells of the glands undergo a retrogressive change, become flattened, and have the appearance of cubical epithelial cells. These atypical cells block some of the ducts, and when we notice how closely the condition resembles epithelioma we are tempted to admit that there is no sharp line of demarcation between chronic gastritis, adenoma, and cancer. The adenomatous changes in the glands assume various forms. We find flat (Andral), pedunculated (Cruveilhier), or nodular adenoma, which somewhat resembles the convolutions of the brain. The interstitial and intermuscular tissue is hyperplastic. When the fibrosis chiefly affects the pylorus, it produces stenosis, and later dilatation of the stomach.

As the interstitial tissue increases the glands disappear, which explains the almost entire absence of free hydrochloric acid and pepsin. In other cases, however, the adenomatous degeneration is more marked than the formation of fibrous tissue, and as a result the chronic gastritis assumes the adenomatous type. Superficial ulcerations are sometimes found which do not extend beyond the mucosa. Small abscesses may also be present in the walls of the stomach.

**Symptoms.**—In very many cases the symptoms at first resemble those of simple dyspepsia so closely that a separate description is not possible. As the disease progresses the patient complains of epigastric pain, which is more acute after a meal. Loss of appetite, eructations, and ballooning of the abdomen are constant. Vomiting is frequent, and besides the food which has remained a longer or shorter period in the stomach, the patient brings up mucus, especially in the morning. This mucus is composed of the gastric secretions and the saliva swallowed during the night. Chronic gastritis is often accompanied by buccal catarrh, obstinate constipation, and congestion of the liver. Duodenitis, with or without ulceration of the mucous membrane, has also been noticed.

Examination of the epigastric region sometimes reveals induration of the stomach. While it simulates cancer, it is really due to the fibrosis of the submucous tissue. Hæmatemesis may also occur, as in a case quoted by Trousseau. When the stomach is dilated, the enlargement is revealed on percussion by a tympanitic note. If the stomach contains fluid, we can obtain a succussion splash by gently shaking the patient.

Wasting and loss of strength result from chronic gastritis. Under the influence of treatment improvement is often noticed, with long remissions and even cure may occur; in severe cases, especially when cirrhosis of the liver or Bright's disease is present, the disease runs a progressive course, and is generally incurable.

**Diagnosis—Treatment.**—The diagnosis of chronic gastritis from ulcer and from cancer of the stomach will be given later. The treatment will be discussed under Dyspepsia.



I must specially mention the diagnosis of chronic gastritis from the gastric manifestations of uræmia. Uræmic vomiting and gastritis are easily recognizable when they supervene in a patient with confirmed Bright's disease. In some cases, however, vomiting is the first apparent manifestation of Bright's disease, and if this cause is not thought of, an error in diagnosis will result.

Milk diet often gives excellent results in chronic gastritis. Gastrorrhœa and flatulence which resist medical measures are sometimes improved by lavage.

#### IV. DYSPEPSIA.

**Dyspepsia**, or **difficulty in digestion**, is a symptom common to many acute or chronic diseases. Even when this symptom becomes so prominent as to constitute a pathological species, it remains subordinate to morbid conditions which differ widely from each other (Trousseau). Dyspepsia, therefore, is only a symptom ; it is not a morbid entity.

Several classifications have been proposed. Dyspepsia has been looked upon as a kind of neurosis or as a chronic affection of the glands of the stomach. Glandular, mucous, or neuro-vascular dyspepsia, and dyspepsia *ab ingestis*, have all been described. Gübler has divided dyspepsia into painful, atonic, catarrhal, and inflammatory.

As I cannot enter here into full details, I must be content to mention its principal characteristics and its most important varieties, and I refer the reader to special monographs for fuller details.

**Pathogenesis.**—It is unnecessary to give details of the chemistry of the stomach, because authors are not unanimous in opinion. The question is at present somewhat obscure. I have had a recent proof of this in a patient whom I had sent to Vichy. Frémont wrote to me : "Striking disproportion between the chemical reactions, which are almost normal, and the dyspepsia, which is extreme."

In reducing gastric digestion to its simplest physiological expression we find two factors : (1) **movements**, (2) **secretions**. If the movements of the stomach become irregular or lose their force, if they become too slow or too quick, or if there is no harmony between the mechanical (movement) and the chemical acts (secretion), dyspepsia is the result. If the secretions of the stomach are changed in quantity or quality, if the acid and the pepsin of the gastric juice are no longer in the proper proportions, then the chemical act of digestion is imperfect, and we have dyspepsia. I hasten to state that the **chemical trouble** is the essential factor in dyspepsia.

The gastric juice owes its antiseptic action to the hydrochloric acid, which prevents abnormal fermentation in the stomach. Oxygen, nitrogen, carbonic acid gas, hydrogen, and sulphuretted hydrogen, may be set free

by abnormal fermentation, which is one of the most common causes of tympanites and of eructation of gas.

The bacilli of lactic and butyric acid fermentation, the spores of the *Mycoderma aceti*, and many other micro-organisms, have a useful or harmful action. They may irritate the mucosa, thus favouring distension of the stomach. They may also produce toxic matter, and neutralize the hydrochloric acid. When we remember that the various factors of digestion must act in perfect harmony in order that the function may be normal, and that, on the other hand, if one of these factors is altered, the digestive process as a whole is deranged, it is easy to see that many causes may bring on dyspepsia. According to Ewald's happy expression, there is "**gastric insufficiency.**"

"In the stomach," says Guyon, "the fermentations are of two types: in the one they result from organic stenosis, and are only an epiphenomenon which occasions no trouble; in the other, they exist apart from any organic lesion and distension. We then speak of fermentative dyspepsia, described formerly under the name of flatulent or acid dyspepsia." Naunym was the first to draw attention to these gastric fermentations. As A. Robin has proved, this form of dyspepsia may be primary, and lead to functional troubles, or secondary to hyperchlorhydria or hypochlorhydria. As a matter of fact, we do not know whether there are special types of fermentation, accompanied by special symptoms and characterized by special micro-organisms.

The **causes** of dyspepsia are at times easily grasped; in other cases they are but hypotheses. In the enumeration of the causes I do not include, of course, acute and chronic gastritis, dilatation of the stomach, or ulcer and cancer, where dyspepsia is merely symptomatic of the different morbid entities, and is only of importance as regards the diagnosis.

Amongst the most frequent causes of dyspepsia, some are local and the direct result of the inefficient action of the stomach, while others depend upon some general disease or remote lesion. Amongst the former we must place the question of foods. A person who has been in the habit of stimulating the stomach by means of highly-spiced foods or alcohol will suffer from dyspepsia if he suddenly stops the stimulant. So, too, indefinite stimulation will end in catarrh and gastritis. In large eaters and heavy drinkers the stomach becomes dilated and the muscular irritability is reduced. This muscular asthenia is a cause of dyspepsia. The opposite excess—viz., deprivation of food—also favours dyspepsia. Excessive work, sedentary habits, night work, and worry produce a like result, and produce a form of dyspepsia which we shall study later under the name of Gastroxia.

In the second category we must place dyspepsia due to disease of a more or less remote organ. Dyspepsia is often associated with diseases of the



liver or of the uterus, with pregnancy, and with diseases of the heart, the kidneys (Bright's disease), or the bladder and the urethra (Guyon).

To a third category belong the dyspeptic troubles, resulting from anæmia, chlorosis, tuberculosis, syphilis, gout, and neuroses, of which neurasthenia deserves special mention.

Each of these causes should be studied in detail. Thus, the dyspepsia which is associated with genito-urinary diseases may result from fever or from some infection (catarrh of the bladder, pyelitis). It may also be the consequence of Bright's disease and uræmia.

Dyspepsia in tubercular patients, which is often associated with lesions of the stomach, and is often so marked at an advanced period of the disease, may be present from the first, and exactly simulate dyspepsia of anæmic origin, although it is really a question of tuberculosis.

Gouty dyspepsia, so characteristic and so frequent that it may be said that the stomach is to gout what the heart is to rheumatism, shows various forms. It may precede the attack of gout or may exist apart from any joint trouble in gouty people.

Dyspepsia in neurasthenic patients seems to be rather the cause than the effect of the neurasthenia.

**Symptoms.**—The functional troubles may be divided into several varieties. These divisions, however, are artificial, and the different varieties are sometimes associated in the same patient.

As a rule, dyspepsia is accompanied by loss of appetite. The patient does not feel hungry, and even when fasting experiences a feeling of fulness in the stomach. He sometimes complains of cramp in the stomach, due in part to **spasms of the pylorus**. When he has begun his meal, he eats sufficient, but is careful to avoid certain foods, such as meat, fish, fats, and vegetables which do not agree. Digestion is retarded. It is sometimes accompanied by pains in the stomach, ballooning of the epigastric region, congestion of the face, drowsiness, pain, eructations, and in some cases by retching and vomiting. The pain and tympanites may not appear till two or three hours after a meal. In the morning the patient suffers from dryness of the mouth, bitter taste, and coated tongue. The epigastric region is sensitive; constipation is the rule. This condition of the digestive tract (gastro-intestinal dyspepsia) often affects the entire economy. The affection may be the result of reflex action, such as arrhythmia or dilatation of the right side of the heart (Potain), or of auto-intoxication (Bouchard). The dyspeptic complains of headache, vertigo, and inaptitude for work; anæmia, loss of flesh, hypochondria, and neurasthenia supervene. If the reader will refer to Neurasthenia, he will find the relation between neurasthenia and dyspepsia.

The general symptoms may in time become very severe. The patient

complains of palpitation and shortness of breath. The skin becomes pale and earthy, and the emaciation is so great that we are apt to suspect cancer of the stomach.

**Flatulent** dyspepsia is characterized by abundant formation of gas, which is associated with the other dyspeptic troubles. After a meal, gas forms in the stomach and the intestine, the swelling being so marked that the patient is compelled to loosen his clothes. Eructations are at times abundant, and afford relief. The gastro-intestinal **pneumatosis** is not exclusively due to abnormal fermentation, but results sometimes from a secretion of gas analogous to that seen in hysterical patients. It has also been stated that the gas forms in the intestine, and is driven back into the stomach by antiperistalsis (Leven).

In acid dyspepsia the vomit is acid, and causes a burning sensation in the throat (**pyrosis**). Acid vomiting is most pronounced in **gastroxia**.

In one form of dyspepsia, **boulimia** replaces loss of appetite. It is generally met with in women suffering from hysteria or gastralgia. The patient experiences a continual sensation of emptiness in the stomach, and is always hungry. Even when the hunger is satisfied, it is accompanied with a feeling of faintness. This form is not, as a rule, accompanied by eructations, flatulence, or constipation. Diarrhoea is more often present.

In gouty persons dyspeptic troubles are often associated with congestion of the liver and hypersecretion of bile (**polycholia**). These hepato-gastric troubles often alternate or coincide with eczematous eruptions.

Writers have described under the name of **gastroxia nervosa** a dyspeptic condition that comes on in fits, and is perhaps a variety of migraine. The attacks are most common during mental strain. They come on every month or two, and disappear under the influence of rest or of change of air. The health is excellent between the attacks. The attack is characterized by violent headache, burning pain in the stomach, and vomiting of **acid fluid**, which leaves an acrid feeling in the pharynx for several hours. Analyses have shown the presence of an **excess of hydrochloric and lactic acids**. The attacks may be checked by very hot water. Unless treatment is given, they last for a longer or shorter period.

**Course—Diagnosis—Treatment.**—Dyspepsia is essentially chronic, and subject to recurrence, especially in the gouty form. Though we have separated it from chronic catarrh of the stomach, it must be remarked that the symptoms of dyspepsia and of gastritis are so closely allied that we are frequently compelled to include both in the same description. The difficulty in diagnosis is not the recognition of dyspepsia; it consists in discovering whether the trouble is purely functional or whether it is the result or the cause of some nervous condition (neurasthenia), and also whether it is a manifestation of some remote lesion (tuberculosis, pyelitis, nephritis), or the



forerunner of disease of the stomach, such as ulcer or cancer. The problem is sometimes difficult to solve, because the symptoms of cancer are at times preceded by a long period of dyspepsia, which may be associated or not with chronic gastritis and adenomatous changes in the stomach.

The **treatment** varies with the nature and the cause. Analysis of the chyle may help to show whether the acid is in excess or not. Proper foods (milk, strong meats, white meats) and beverages (alkaline, bitter or fermented drinks), regular meals, and suitable exercise—or, in a word, a correct régime—play a great part in the treatment of dyspepsia. In many cases (simple or gouty dyspepsia) alkalis, lime-water, Vichy water, Pougès, Carlsbad, or Homburg waters are indicated. In gastro-intestinal catarrh mild purgation is needed (Birmenstorff or Pullna water). Belloc's charcoal is indicated in flatulent dyspepsia, which is often accompanied by acid eructations. Alkaline preparations are also of use if replaced in a few days by quassia, calumba, or other bitter tinctures. If anæmia is present, chalybeate waters (Spa and Forges) should be selected.

In my opinion, Trousseau's plan is the best. Flatulent dyspepsia is the most frequent form, and its usual symptoms are loss of appetite, constipation, foul tongue, ballooning of the abdomen, eructations after a meal, headache, congestion of the face, and vertigo. In these cases I advise the following measures :

1. Take every morning when fasting half a glass of Vichy water (Célestins).

2. Before the two principal meals take 3 drops of Baumé's bitter tincture in a spoonful of water.

3. During the meal take a cachet composed of—

|                     |   |    |    |    |              |
|---------------------|---|----|----|----|--------------|
| Prepared chalk      | } | .. | .. | .. | āā 2 grains. |
| Bicarbonate of soda |   |    |    |    |              |
| Magnesia            |   |    |    |    |              |

4. If constipation is severe, the cachets are replaced by the following :

|                     |    |    |    |           |
|---------------------|----|----|----|-----------|
| Bicarbonate of soda | .. | .. | .. | 4 grains. |
| Rhubarb             | .. | .. | .. | 3 grains. |

5. Take after the meal 1 or 2 tablespoonfuls of lime-water.

6. If pain is present,  $\frac{1}{10}$  grain cocaine is added to the lime-water.

7. In the case of constipation, take a mild purgative on going to bed—either a teaspoonful of Vichy laxative powder in half a glass of Évian water, or a cascara pill.

8. Eat foods that agree well. Drink at meals Évian, Alet, or Vittel water, with or without red or white wine.

9. Avoid alcoholic beverages and pure wine. Hot infusions after meals, such as tea, coffee, or camomile, are permitted.

10. Take a cure at Vichy or Pougès.

Some kinds of dyspepsia are not relieved by alkalis, and must therefore be treated with acids. The patient is given hydrochloric acid, diluted with water, after each meal.

Dyspepsia, accompanied by boulimia, sensation of emptiness in the stomach, and diarrhoea, which comes on directly after taking food, is relieved by small doses of opium. One or two drops of Sydenham's laudanum before each meal will suffice.

**Lavage of the stomach** is sometimes very useful, especially if the dyspepsia results from catarrh, with or without dilatation of the organ.

Frémont has suggested the employment of gasterine (animal gastric juice). I have found it useful, especially in obstinate dyspepsia. "Gasterine is an acid, watery fluid, with great digestive power. It is the type of stomachic opotherapy, and is applicable in all cases of insufficient secretion by the stomach, except cancer." Gasterine is administered in doses of 3 to 10 ounces daily, mixed with milk, beer, broth, water, or wine, either during, before, or after a meal.

## V. GASTRALGIA.

**Ætiology.**—**Gastralgia** (cramp in the stomach) is neuralgia of the nerves of the stomach. It is sometimes essential and comprises the whole disease, but at other times it is secondary and appears as a symptom of various morbid conditions.

It may be brought on by cold, fatigue, worry, or the use of stimulants. It is a frequent symptom in dyspepsia, hysteria, neurasthenia, anæmia, and uterine diseases. It is caused by phthisis, gout, malaria, and certain affections of the spinal cord, notably locomotor ataxy. In one case it was undoubtedly connected with the reduction of a varicocele.

**Description.**—Pain is the essential and at times the only symptom. It occurs in paroxysms, which are generally spontaneous and independent of the ingestion of food. The pain is sometimes preceded by eructations, nausea, or pyrosis. The attack of pain may be slight and limited to the epigastric region, with or without spasms of the pylorus. At other times it is acute, and accompanied by vomiting and syncope. The pain is of the most varied character; it may be stabbing or agonizing, and radiates to the back, the thorax, and the base of the chest. It may invade the abdomen and reach the hypochondria, the kidneys, or even the spermatic cord. In severe attacks the face is white and pinched, and the patient is obliged to press upon the painful region, seeking to diminish his suffering by assuming every possible position. The attack may last for some minutes, but in other cases the duration may vary from a quarter of an hour to an hour. It may return several times in the twenty-four hours, and on several consecutive days.



If gastralgia is associated with dyspepsia or gastric catarrh, digestive troubles are also present. In other cases digestion is normal, and the appetite is good, or at times increased. In hysterical and chlorotic women the taste is perverted (*pica*). In locomotor ataxy the attacks (**gastric crises**) have the characteristics which are described in the chapter on *Tabes*, and may be the first symptom. The duration of gastralgia is subordinate to its cause. It may be transient or persistent and subject to repetitions.

**Diagnosis.**—**Simple ulcer** of the stomach provokes pains like those of gastralgia, but the ulcer is recognized by other symptoms, such as intolerance of certain foods, the appearance or the aggravation of the pain after meals, the xiphoidal or spinal seat of the pain, and the presence of blood in the vomit (*hæmatemesis*). **Cancer** of the stomach causes less pain, but this lesion is sometimes ushered in by persistent epigastric pain, and the diagnosis must be made from gastralgia. The age of the patient, the gradual wasting, *hæmatemesis*, *melæna*, *phlegmasia alba dolens*, presence of gastric tumour, and hypochlorhydria, are important points in cancer. Gastralgia must not be confounded with superficial neuralgia of the skin or of the muscular layers of the epigastric region. **Epigastralgia** and **neuralgia of the intercostal nerves** are accompanied by hyperæsthesia, which is easily provoked by pressure at the exit of the nerve branches. Slight attacks of **hepatic colic**, particularly if jaundice is absent, are frequently mistaken by patients for gastralgia. In order to avoid this mistake we must localize the seat of the pain, which in hepatic colic is in the right hypochondrium, and frequently radiates to the right shoulder. The liver, which is often painful and enlarged, must be percussed, and the urine tested, as it often contains bile pigment. Hepatic colic, even if slight, is at times accompanied by vomiting, and followed, if not by icterus, at least by a subicteric tinge of the conjunctivæ.

It is not sufficient to diagnose gastralgia; the cause must also be ascertained, for the prognosis largely depends on it. We must find whether the gastralgia is purely nervous, symptomatic (ulcer, cancer, *tabes*), or an early symptom of pulmonary tuberculosis.

**Treatment.**—In the attack the first point is to relieve the pain. Ice-bags are applied over the stomach, aspirin or antipyrin is prescribed, and an injection of morphia in the epigastric region is administered, according to the duration and intensity of the attack.

In the intervals the cause must be treated: antispasmodics (*valerian*, bromide of potassium) in cases where the nervous element predominates, alkalis and bitters if the gastralgia is associated with one of the forms of dyspepsia previously described. Laudanum should be given before meals if the attacks of pain are provoked by the ingestion of food. Milk diet is also indicated. In many cases hydrotherapy renders valuable services.

## VI. ULCERATION OF THE STOMACH—GENERAL CONSIDERATIONS.

**Erosions and Ulcerations.**—**Ulceration of the stomach**, from a simple erosion to the complete destruction of the wall, occurs in widely different conditions. **Hæmorrhagic erosions** are found in alcoholic or uræmic gastritis (Treitz), and in gastritis of the new-born, which is called “**ulcerous gastropathy**” (Parrot).

1. If in an infant the skin dries quickly and becomes bluish, the eyes are sunken, and the face is pinched, and the vomit contains brownish flakes, ulceration of the stomach should be suspected. Post mortem, the stomach is found covered with a thick layer of mucus, and studded with blackish clots of blood, modified by the gastric juice. Beneath the mucus the erosions form circular ulcers, some being hardly visible to the naked eye, while others are more than a millimetre in diameter. They are chiefly found on the greater curvature and in the pyloric region. Histological examination shows that the ulceration affects the entire mucous membrane (Parrot). Sometimes it destroys the superficial part of the glands, and at other times the glandular layer. The venous congestion is marked.

2. **Hæmorrhagic erosions** of the stomach are also seen in venous stasis (obstruction in the portal circulation, diseases of the liver and heart). The erosions are generally small and rounded, with flat edges, but they sometimes follow, in a more or less capricious manner, lines or circles in relation to the submucous veins. Half a dozen are found, on an average, to a square centimetre, and the largest never exceed 2 millimetres in diameter. The lesion seems to be both inflammatory and necrobiotic. The blood-stasis and the inflammation succeed one another, or combine to bring about the death of the tissue.

3. **Tuberculosis**, which is essentially a destructive affection, does not always spare the stomach. Functional troubles are frequent, but **tubercular ulcerations** of the stomach are so rare that Marfan quotes but fourteen cases, and Letorey has collected only twenty-one authentic cases. The ulcers vary much in character. Sometimes they are as small as a pin's head; at other times they are 4 to 5 centimetres in diameter. The edges are ragged and irregular. Tubercles are often seen on the floor and the circumference of the ulcer. Bacteriological examination has several times revealed bacilli. Tubercular ulcers of the stomach are nearly always secondary, and develop in patients already suffering from tuberculosis. They are usually latent, and are found by chance at the post-mortem examination. The tubercular ulcer, unlike the simple ulcer of Cruveilhier, has little tendency to perforation. Hæmatemesis, though rare, is a possible complication. It may be fatal, as in two cases quoted by Letorey.



On microscopic examination the subglandular zone of the mucous membrane and the tunica submucosa are found to be infiltrated with tubercles; the tubercular infiltration also follows the vessels of these regions. The ulceration attacks the mucous membrane, as a rule, and rarely extends beyond the submucous layer. The peritoneum over the ulcer is thickened, but tubercles are rare. In exceptional cases the ulceration may involve the gastro-epiploic vessels, and produce fatal hæmorrhage, or cause perforation of the stomach, with acute peritonitis. Perforation of the stomach in the six known cases has always been produced from without inwards, by peritonitis, colitis, or tubercular adenitis.

F. Arloing, who has written an important monograph on tubercular ulceration of the stomach, comes to the following general conclusions :

“Tubercular ulceration of the stomach is not common in man and in animals. It is always accompanied by other tubercular lesions in the viscera. The different forms of tubercular ulcerations seen in man may be reproduced experimentally.

“Direct penetration of Koch’s bacillus through the healthy or diseased mucous membrane, either naturally or experimentally, seems doubtful. Clinically, it is probable that the infectious agent (bacillus or toxine) selects the blood-passages in order to reach the mucous membrane. The ulceration may be either histologically tubercular or toxi-infectious without histological manifestations.”

4. **Typhoid fever** may cause ulceration of the stomach (Chauffard) in very exceptional cases. Millard’s case is usually quoted, but in the section on Appendicular Ulcerative Gastritis we shall find that this case must be revised, because it has perhaps not been interpreted correctly. Ulceration of the stomach, though appearing in the course of enteric fever, may still persist as a sequela after recovery from the fever, as Cazeneuve’s observations prove.

5. Large **burns** on the surface of the body and contusions of the epigastric region may also be followed by ulcer of the stomach. Cases in which epigastric traumatism has induced **ulcus simplex** have been quoted.

6. **Syphilitic** ulceration of the stomach will be described separately. Gummatous ulcers are common, and often coincide with tertiary lesions in other regions.

7. The next three sections are devoted to the study of erosions and of acute ulceration of the stomach due to toxic poisoning. Hæmorrhage is frequent, and it may, indeed, be said that hæmatemesis is the chief symptom.

## VII. PNEUMOCOCCAL GASTRITIS WITH ULCERATION.

In the next three sections I shall discuss the following subjects :

1. **Pneumococcal gastritis.**
2. **Appendicular vomito negro.**
3. **Exulceratio simplex.**

Let us commence with the study of pneumococcal gastritis.

**Description.**—In the past “pneumonia” summed up the infection which we now call “pneumococcal infection.” Bacteriology, however, has revealed this infection in numerous organs where it had not been suspected. Pleurisy, pericarditis, peritonitis, both cerebral and cerebro-spinal meningitis, endocarditis, otitis, tonsillitis, and arthritis have been placed amongst the pneumococcal infections. These lesions are found not only as secondary complications of pneumonia, but also as **primary** affections, due to the pneumococcus (peritonitis, otitis, meningitis, etc.), and quite independent of pneumonia.

One form of pneumococcal infection has, however, passed almost unnoticed—viz., **pneumococcia of the stomach**. I give here two cases which I have already described in my lectures at the Hôtel-Dieu :\*

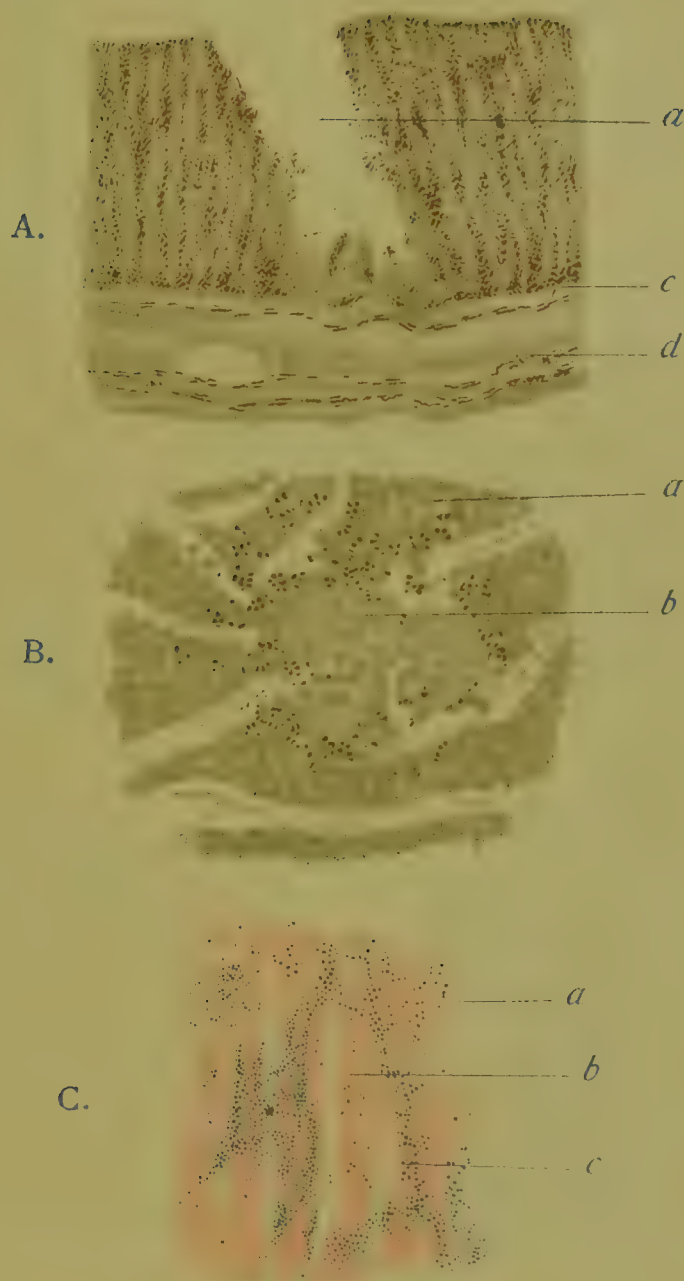
A patient was admitted to the hospital with dyspnœa, cyanosis, temperature of 105° F., respirations 42, and acute pain under the right breast. We found pneumonia at the right base; friction sounds showed that the pleura was also involved. The dyspnœa was so severe that it was impossible to auscultate the heart. The abdomen was painful and distended. He brought up rusty sputum, was restless at night, vomited, and had diarrhœa. The urine contained albumin, and urobilin. The abdominal symptoms of pain, tympanites, vomiting, and diarrhœa, which are rare in pneumonia, attracted attention. Next day the dyspnœa was worse; the abdomen was more painful and distended; the nausea and diarrhœa continued. What was the meaning of these abdominal symptoms? Was peritonitis the cause? Twelve ounces of blood were taken. He had a bad day, though the dyspnœa was improved by the bleeding. Next day great restlessness and complete insomnia. He complained of pains in the stomach and of a desire to vomit; at 5 a.m. he vomited about 1½ pints of blackish fluid, like coffee-grounds. In view of the severe hæmatemesis, I was at a loss for a diagnosis. There was no doubt that the patient was suffering from pneumonia, but why this severe hæmatemesis? As far as I know, hæmatemesis does not appear in pneumonia. Many theories were possible. Could he, prior to the pneumonia, have had an **ulcus simplex**, which had previously been latent? Or else, was not this a case of **exulceratio simplex**? He grew worse from hour to hour. The tympanites, pain, and diarrhœa became more marked; the diarrhœa, so common in primary pneumococcal peritonitis, made us think of peritonitis. Melæna was present—a point which agreed well with the hæmatemesis, but did not clear up the diagnosis. At noon slight hæmatemesis, and at three o'clock, after a severe attack, the patient succumbed, the temperature being 104° F. The chief feature was: severe attacks of hæmatemesis and gastro-peritoneal complications in a patient with pneumonia.

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\* “Gastérite Ulcéreuse Pneumococcique: Grandes Hématémèses” (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 11<sup>me</sup> leçon, p. 219).



Plate V.



A. — *a*, Erosion which affects the whole thickness of the glandular layer, and which stops on the confines of the muscularis mucosæ. The base of the erosion in the necrosed glandular tissue is in process of elimination. In the vicinity of the erosion the interglandular connective tissue has undergone slight embryonic infiltration. *c*, Intact muscularis mucosæ. *d*, Tunica muscularis.

B. — A tangential section of the mucosa on a level with the bottom of the erosion. *a*, the healthy glandular tissue, with a section of its tubes; *b*, the small glandular region, where the necrosis is taking place which will form the erosion. This is the earliest phase of the lesion.

C. — A section of the mucosa, perpendicular to its surface, passing through an erosion. The preparation has been stained by thionine and Gram's method. *a*, The edge of the erosion; *b*, The glandular tubes; *c*, Pneumococci. The microbes infiltrate, not only the edge of the erosion, but are found in numbers in the interglandular connective tissue at some distance from it. It might be called *pneumocorrhagia* — a name which appears to me the more acceptable as the capillaries of the mucous membrane are also infiltrated with pneumococci.





Post-mortem examination : On opening the thorax, the pericardium was found to contain 13 ounces of reddish fluid, rich in red corpuscles. The pericardium was covered with fibrinous exudate, villous and studded with bright spots ; the pneumococcus was the cause of the pericarditis. The heart was of normal size ; in the right cavities, post-mortem, clots. The orifices of the heart and the large vessels showed nothing peculiar. The right pleural cavity contained 10 ounces of reddish fluid, similar to that in the pericardium, but not so clear. At certain points the two layers of the pleura were adherent. The parietal pleura was slightly thickened, and covered with shreds of fibrin. The visceral pleura over the lung formed a kind of connective shell of a lardaceous aspect. Pneumonia of the right lung ; congestion of the left. The left pleura was healthy. The tracheo-bronchial glands were enlarged.

The abdominal cavity showed **peritonitis**. Turbid reddish fluid in the pelvis. The intestines were matted together, and covered with a viscid, non-purulent exudate, studded with granules, several of which did not exceed a pin's head in size. The pneumococcus was the cause of the peritonitis, and the abdominal symptoms present during life were explained : he had had secondary pneumococcal peritonitis. The spleen was enlarged ; the liver and pancreas were normal. The lesions of the stomach were noteworthy. When it was opened and cleaned, several brown points of the size of a pin's head were seen ; one of these points was as large as a small lentil. These brownish points were erosions, and showed clearly under the lens. The edges were clean-cut and adherent, and the floor was filled up with a small hæmorrhagic eschar. The diameter of the erosions was from 2 to 3 millimetres. They were scattered over the whole mucous membrane, but especially in the pyloric region, at the bottom of the folds and on the surface. Bacteriological examination showed that the man had succumbed to general pneumococcal poisoning. The pneumococcus was found in the fluid from the pleura, pericardium, and peritoneum.

The nature of the hæmorrhagic erosions that had caused the hæmatemesis had still to be cleared up. The histological and bacteriological preparations, reproduced in Plate V., were made by Jolly. I think this is the first case in which pneumococcal infection of the stomach has been seen in a complete form with pneumococci, hæmorrhagic erosions, and hæmatemesis.

The erosions were formed by the elimination of an eschar, and the process is clearly shown in Plate V.

The erosions were due to rapid and limited necrosis of a small portion of the mucous membrane, with an inflammatory reaction that was nil or trifling. They were caused by the pneumococcus, and were therefore part of the general pneumococcal infection. These erosions explained the severe hæmatemeses which had been so obscure during life. I have proposed for this condition the name of **pneumococcal hæmorrhagic ulcerative gastritis**.

By a strange coincidence, I had in my wards at the same time a similar case. A man was admitted to the Hôtel-Dieu on the sixth day of pneumonia. **Abdominal** symptoms appeared on the same day—diarrhœa, pain, and tympanites—pointing to pneumococcal peritonitis. He also complained of pain in his left wrist (pneumococcal arthritis). On the eighth day the abdomen was tympanitic and painful, especially at the pit of the stomach and in the right flank. During the night he vomited, and passed two loose stools. The diagnosis of pneumococcal peritonitis was clear. The abdominal condition became worse, and the arthritis of the left wrist more marked. A surprise occurred. The patient, like his neighbour opposite, was seized with pains in the stomach, nausea, and hæmatemesis, and vomited a quart of blackish fluid like coffee-grounds.

For two days peritonism was marked, with vomiting, hiccough, ballooning of the abdomen, and diarrhœa, which is the usual symptom in pneumococcal peritonitis. As the threatening peritonitis began to resolve, the vomiting ceased, the abdominal pain grew less, the ballooning disappeared, and the prognosis was more hopeful.

One morning he felt weak, and complained of violent headache, with buzzing in the ears. He refused nourishment, and lay on his right side in a state of coma. This symptom-complex pointed to meningitis. Death occurred on the nineteenth day of the disease.

The post-mortem examination revealed the following lesions: The lower lobe of the left lung was splenized and infected by the pneumococcus; the right lung was congested. The left pleura was adherent, and a similar condition was found in the interlobar fissure, in which three small abscesses were found. Adhesions and abscesses also existed in the diaphragmatic pleura. The abscesses, both interlobular and diaphragmatic, contained creamy pus, rich in pneumococci.

False membranes and a small quantity of liquid were found in the pelvis, in front of the rectum. Bacteriological examination revealed the pneumococcus, so that these lesions were the result of pneumococcal peritonitis, and showed the spontaneous curability of this secondary variety. On examining the heart, a small, prominent, friable vegetation, from 4 to 5 millimetres in diameter, was found at the insertion of the right sigmoid valve. The pneumococcus was found in smears taken from the vegetation. The symptoms of meningitis observed during life were also explained. On opening the skull, we found purulent pneumococcal meningitis and thick yellowish pus spread along the Sylvian arteries, and over the peduncles and upper surface of the cerebellum. There was nothing noticeable in the cavity of the tympanum.

The radio-carpal articulation and the sheaths of the extensor tendons contained about  $\frac{1}{2}$  ounce of creamy greenish pus. The articular surfaces of the ulna and radius, and the radial surface of the carpal articulation presented a velvet-like change, resulting in complete disappearance of the cartilage at certain points. Bacteriological examination proved the presence of the pneumococcus.

On opening the stomach, we found congestion of the mucous membrane, but no active erosions, as in the preceding case. The histological examination of the mucosa showed small hæmorrhagic foci, some in the submucous tissue, and others, more numerous, in the interglandular tissue. These foci (the origin of the hæmatemesis) pushed the glands aside, and formed distinct masses, that were separated from the exterior by a thin layer of mucous membrane. Around the foci no trace of inflammatory reaction was to be seen. The search for microbes was negative.

In these two cases, by a singular coincidence, we have two men, both suffering from pneumococcia, who are seized within a few days with peritoneal symptoms and severe hæmatemesis. The first patient had several attacks, and finally succumbed to hæmatemesis. The bleeding, as we have seen, was due to pneumococcal infection, with hæmorrhagic erosions.

In the first patient, who died while the gastric stage was active, we were able to surprise the infection in actual progress. In the second patient, who lived some time after the hæmatemesis, we found only the remains of hæmorrhagic foci in the gastric mucous membrane.

In an old woman who had suffered from pneumonia at the right apex, but had during her life shown neither gastric nor intestinal symptoms, Griffon found pneumonia with abscesses, the pus of which contained only pneumococci. In the first portion of the duodenum were two symmetrical ulcers. In the pyloric region there were two ulcers and several punctate hæmorrhages. Although the bacteriological examination did not show the pneumococcus in the ulcers, it is probable that they were due to a toxoinfection, caused either by the microbes or their toxins.

Gastric ulcers occurring in the course of pneumococcia have been studied experimentally in the guinea-pig. According to Bezançon and Griffon, the



pneumococcus is apt to cause hæmorrhagic lesions when its virulence has been increased by successive passages, whilst the fibrino-purulent changes are due to the pneumococcus of less exalted virulence. The peritoneum of a healthy guinea-pig was inoculated with a few drops of peritoneal exudation from a guinea-pig infected with virulent pneumococci. The inoculated guinea-pig succumbed in twenty-one hours, and the post-mortem examination showed petechiæ in the mesentery and the large intestine, and hæmorrhagic nodules in the spleen. The mucosa of the stomach was studded with about fifteen hæmorrhagic erosions. The histological examination of these erosions showed that the process affected only the mucous and submucous layers. Pneumococci were present in the ulcer, as in all the organs. These experiments are of value, in that they allow us to place clinical and experimental cases side by side.

**Summary.**—The stomach, like other organs, may be infected by the pneumococcus. This organism may perhaps localize itself from the first in the stomach, causing a primary gastritis. Why should we not find primary and secondary forms of pneumococcal gastritis, just as we do in meningitis? Pneumococcal infection of the stomach may be revealed by gastric symptoms. Still, nausea and vomiting of food or bile are of no great significance, because they may occur in simple pneumonia. On the other hand, attacks of hæmatemesis in the course of pneumonia point to pneumococcal gastritis with ulceration. Melæna has the same signification, whether it is due to ulcerative gastritis or enteritis.

Although the erosions are very small, there is nothing to show that they may not be more extensive. They may get well quickly (as in my second case), but it is equally probable that they may survive the gastritis. An individual, though cured of pneumonia and apparently of pneumococcal gastritis, may yet have an invading gastric ulcer, which will perhaps in time assume the form of Cruveilhier's **ulcus simplex**. In other words, it is not impossible that the **ulcus simplex** may in some cases arise from a pneumococcal erosion. This view of the transformation of infective gastric ulcerations into **ulcus** has gained ground during the past few years. Gandy has written a remarkable monograph on this subject, to which I shall refer under *Exulceratio Simplex*.

### VIII. APPENDICULAR VOMITO NEGRO.

Amongst the innumerable misdeeds of the appendix there is one which I shall now discuss—namely, vomiting of blood. A few years ago no one thought of hæmatemesis in connection with appendicitis, and until recent years text-books of medicine and surgery were silent on this point; and yet hæmatemesis resulting from appendicitis is far from being rare.

I do not refer to slight hæmatemesis, which is only an epiphenomenon, and may be lightly passed over. On the contrary, we usually find severe attacks of hæmatemesis. **The black vomit** may amount to  $\frac{1}{2}$  pint of blood, the attacks may be repeated, and the patient may die from hæmorrhage.

I have given the name of **appendicular vomito negro** to this tragic event, and in addition to a communication to the Académie de Médecine I have delivered a clinical lecture on it.\* The following cases will give us full details :

On June 6, 1900, in consultation with Gros and Cazin, I saw a young American lady who was suffering from appendicitis of four days' duration, complicated by peritonitis. She was operated upon by Cazin on the same day. In spite of the operation, her condition remained grave, and the fever persisted. Two days later jaundice was just beginning. Urine scanty and high-coloured. In spite of treatment, complete suppression (toxic anuria). The first severe attack of hæmatemesis now appeared. During the night, delirium, alternating with restlessness and coma ; the anuria persisted. Next day the jaundice had become general. Blood was repeatedly vomited. The delirium continued, and the patient died a few hours later, after an attack of copious hæmatemesis.

On October 8, 1900, I saw, with Sevestre and Quénu, a young girl of ten years of age, who had appendicitis. The attack had commenced forty hours previously, with vomiting and pain. When we arrived, at half-past seven, she was very restless. Her features were drawn ; her pulse was 140, and her temperature nearly 104° F. Suppression of urine since the morning. The abdomen was distended, and the region of the appendix painful. The prognosis was alarming, and we decided on immediate operation.

On opening the abdomen, diffuse peritonitis ; serous and foetid pus in the pelvis. The appendix lay behind the cæcum, and its tip was gangrenous. The restlessness persisted all night.

Next morning the general condition was bad ; temperature 103° F. ; pulse 122. Injections of serum given. Restless night. Next morning temperature rose to 104° F., and pulse to 130. Everything went well as far as the abdomen was concerned, but the child was poisoned, and complications soon appeared. At ten o'clock temperature was 104.3° F., pulse 140, and respirations 45. We saw her at noon, when she had an earthy complexion and small, irregular pulse. The urine was scanty and albuminous. From the vomit, in which I saw some shreds of **black blood**, I foretold an attack of hæmatemesis, which came on two hours later ; it was profuse. Ice was given, and the injections of serum continued, but her condition became worse every hour. Other attacks of hæmatemesis occurred at 6 p.m. and at 8 p.m. The breathing was quick ; the extremities were cold ; she passed several large foetid stools, and died at 9.15 p.m.

On January 13, 1900, with Bergeron and Widal, I saw a patient who had been operated upon by Delbet on the fourth day of an attack of appendicitis. Next day the patient was seized with hæmatemesis and died.

On October 30, 1900, I saw, with Charrier, a young man who for three days had been suffering from appendicitis. The yellowish tinge of the conjunctivæ and the condition of the urine showed general infection. The situation appeared to be most grave, and an operation was performed that evening by Segond. On opening the abdomen a foetid abscess and diffuse peritonitis were found ; the intestine was of a purple colour. The abscess passed up behind the cæcum, the limiting membranes being very friable. The appendix was perforated and gangrenous. Next morning he seemed a little better.

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\* "Vomito Negro Appendiculaire" (*Clinique Médicale de l'Hôtel-Dieu*, 1903, 10<sup>me</sup> leçon).



but in the evening he had an attack of **hæmatemesis**, and vomited about 3 ounces of dark blood, like coffee-grounds. The pulse rose to 140, and nervous symptoms of a toxic nature appeared—viz., stupor, loss of consciousness, and oscillations of the head, which lasted till death.

On January 27, 1900, I saw, with Segond and Ramond, a young girl who had been operated upon by Segond on the fourth day of an attack of appendicitis. During the next few days she had several attacks of **hæmatemesis**, which, fortunately, did not prevent recovery.

A youth was operated upon by Piéchaud on the sixth day of an attack of appendicitis; a few hours later he felt much better. At five o'clock in the afternoon he was *in extremis*. An injection of serum was administered, but the patient was seized with **fulminant hæmatemesis**, and died at one o'clock in the morning. At the post-mortem we found the appendix perforated at its tip. The stomach was distended with black blood, as the result of the hæmorrhage; the mucous membrane showed ecchymotic patches.

**Pathogenesis.**—Post-mortem, ecchymotic patches are found on the mucosa of the stomach, and erosions which are of hæmorrhagic origin and the result of the appendicular toxines. I have shown (*vide* Appendicitis) that the appendicular focus developed in a closed cavity causes not only infection, but also intoxication. It is the appendicular toxines that provoke the jaundice, albuminuria, anuria, and nervous troubles which I shall discuss under Appendicitis. It is this same toxi-infection which causes the gastric erosions and the consequent hæmatemesis. When the toxic syndrome is complete, as in the first case quoted, the jaundice and black vomit confirm the name of appendicular vomito negro.

Since attention has been called to this complication of appendicitis I have asked myself what gastric lesion was capable of producing such attacks of hæmatemesis. My researches on pneumococcal erosions and exulceratio simplex had taught me that the attacks of hæmatemesis consequent on these toxi-infections are due to ulceration of the arterioles ramifying under the muscularis mucosæ. I have found that the process is identical in the gastric erosions which follow strangulation of the gut, as in the following case:

On Saturday, June 16, a man was admitted for strangulated hernia, and was immediately operated upon. Next morning the conjunctivæ and the face were yellowish; during the night two severe attacks of **hæmatemesis** took place. On Monday the jaundice was more marked. Kahn, who examined the urine, found albumin, urobilin, and brown pigment. My prognosis was grave. During the day fresh hæmatemesis occurred. On Tuesday the urine was scanty and albuminous; he had a large hæmatemesis. On Wednesday he passed only 5 ounces of urine, containing brown pigment, urobilin, and albumin. He was delirious, and was seized during the night with several epileptiform fits. On Thursday fresh fits, complete anuria, and another attack of hæmatemesis. On Friday death. The results of the post-mortem examination were as follows: Cure of the strangulated hernia; nothing in the intestine or peritoneum. The liver (50 ounces) and the kidney (7 ounces) showed **acute degeneration of the epithelium**. The stomach contained blood in abundance, but the mucous surface was intact, except in the pyloric region, where there was a hæmorrhagic eschar as

big as a small lentil. This eschar was prominent, adherent, and surrounded by a furrow.

The histological examination made by Gandy is shown in Figs. 26-28.

**Summary**—Necrosis of an extensive portion of the mucosa in the form of an eschar, cutting the glandular layer cleanly, injuring the muscularis mucosa, the superficial part of the submucosa; after affecting other anatomical parts in the course of its progress, it finally reached the superior wall of an arteriole situated below the muscularis mucosæ, which gave way. In the neighbourhood of, and at a distance from, this focus of necrosis there were some submucous hæmorrhages and secondary inflammatory reaction, shown chiefly by embryonic infiltration in the form of large subglandular masses.

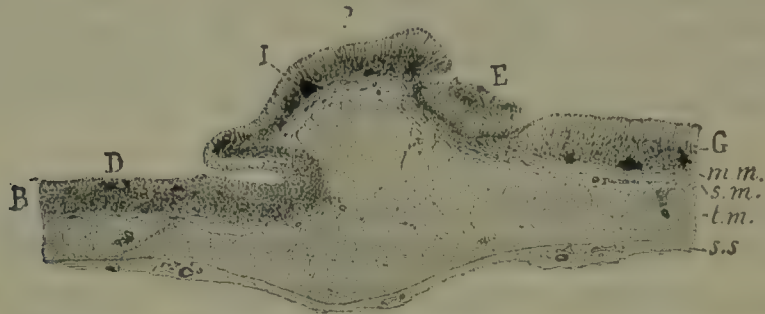


FIG. 26.—ONE OF THE SECTIONS (LOW POWER).

P, Gastric side of the pyloric valve; D, mucosa of the duodenum; B, Brünner's glands; I, inflammatory subglandular islets; E, eschar breaking up the glandular layer; G, glandular layer of the stomach; *m.m.*, muscularis mucosæ; *s.m.*, submucous layer; *t.m.*, tunica muscularis; *s.s.*, subserous layer.



FIG. 27.—ONE OF THE SECTIONS (HIGH POWER).

G, Glandular layer of the pyloric region; *m.m.*, muscularis mucosæ; *s.m.*, submucous layer infiltrated with fibrinous exudation; *t.m.*, tunica muscularis; E, eschar; *n*, invading zone of the necrosis; A, arteriole of the submucosa, injured by the necrosis at the base of the eschar.

The experiments made by Talma (Utrecht, 1890) on the pathogenesis of simple ulcer of the stomach show the part played by intestinal strangulation in the production of erosions and ulceration. Talma repeated his experiments several times. He ligatured a coil in an animal, or, in other words, established a closed cavity, and amongst other lesions he found erosions and ulceration of the stomach. The clinical and experimental



facts, therefore, agree. Similar gastric erosions existed in three cases reported by Charlot.

We have now gained some idea of the lesions which provoke hæmatemesis consequent on appendicitis and strangulated hernia. The toxic process is identical in both cases. The infection causes acute hæmorrhagic erosions and ulcerations of the stomach comparable to the ulcerations found in pneumococcal infection and to *exulceratio simplex*.

**Description.**—**Vomito negro** is part of the appendicular toxi-infection. I have often endeavoured to prove that appendicitis is not only a focus of infection, but also of intoxication.\* The toxins are responsible for changes in the liver, with jaundice, urobilinuria, and sometimes symptoms of icterus gravis. They are also responsible for changes in the kidneys, with albuminuria, oliguria, anuria, and sometimes symptoms of uræmia. The lesions

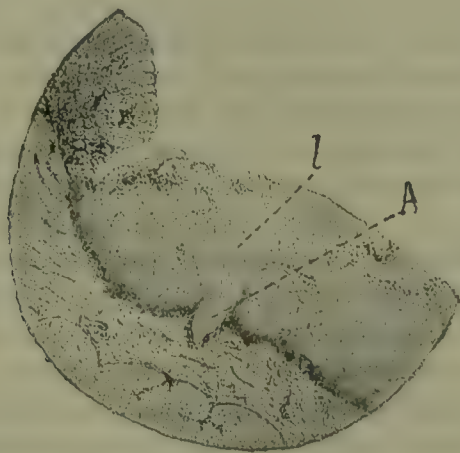


FIG. 28.—SECTION THROUGH SUBMUCOSA.

A, Arteriole with the superior wall opened, communicating with a large lacuna, l, made by the eschar.

of the stomach, with the attacks of hæmatemesis, therefore appear to me to form part of the **toxic syndrome of appendicitis**. Hæmatemesis is sometimes independent of other toxi-infectious manifestations, but at other times it is associated with them.

Jaundice of the conjunctivæ and skin, urobilinuria, and albuminuria often precede the appearance of hæmatemesis. In the first case quoted, the jaundice was general when the black vomit appeared.

Appendicular hæmatemesis may assume different forms. The attacks are sometimes preceded by nausea and vomiting. On examining the vomit, blackish streaks and clots like coffee-grounds, which are the forerunners of an attack of hæmatemesis, may be seen. Sometimes the attack comes on suddenly without prodromata, even when an operation for appendicitis had

\* Dieulafoy, "Toxicité de l'Appendicite" (communication à l'Académie de Médecine, 1899, et *Clinique Médicale de l'Hôtel-Dieu*, 1899, 17<sup>me</sup> leçon).

apparently averted complications. A single attack of hæmatemesis is rare; six or more may occur in a few hours. Sometimes the attacks are, so to say, fulminant, and carry off the patient in a few moments.

I have so far considered only hæmatemesis due to ordinary appendicitis. The paratyphoid form of appendicitis, which appears during the decline of typhoid fever, may also give rise to gastric ulceration and fatal hæmatemesis, as in the following case :

In 1876 Millard communicated to the Société Médicale des Hôpitaux the case of a patient who, during convalescence from typhoid fever, was attacked by acute peritonitis, and died a few days later, after attacks of very profuse hæmatemesis. At the post-mortem examination diffuse purulent peritonitis was found; it was not due to the intestinal lesions, for they had quite healed. It was necessary to seek elsewhere, says Mallard; and, indeed, he discovered "in the vermiform appendix the evident cause of the peritonitis. The appendix, which had perforated in its middle third, no longer communicated with the cæcum; the opening into the cæcum was completely obliterated."

The attacks of hæmatemesis from which he suffered during the course of the peritonitis were due to ulceration of the stomach near the pyloric region. One of the ulcers was oval, 2 inches in its larger diameter, and very deep, with irregular, clean-cut edges. Millard discusses the cause and origin of these ulcers, and recalls the excessive rarity of gastric ulcer in typhoid fever. With wise prudence, he refrains from coming to a conclusion, and, with great intuition, he asks himself if there is not some connection between the purulent peritonitis and the ulcerative process in the stomach. We know the connection, and we can explain the pathogenesis of the complications in this case: paratyphoid appendicitis, with its toxi-infection, had, as in common appendicitis, caused peritonitis on the one hand, and gastric ulceration, with hæmatemesis, on the other. This explanation at once clears up this case, which has been regarded as a typhoid ulcer of the stomach; for even supposing that the typhoid infection had played some part, it is the appendicular toxi-infection which claimed the larger share.

In **appendicular vomito negro** the prognosis is most grave. Some cases of cure, however, have been reported.

Absolute rest for the stomach, no liquid by the mouth, and large injections of serum, appear to me to be the rational treatment of this complication. Lucas-Champonnière, however, advises lavage.

The terrible complication just described darkens the prognosis of appendicitis, and is a further plea in favour of early operation. The more time the appendicular focus has to elaborate its poisons, the more threatening the situation; the less time the focus has to poison the victim, the less are the complications to be feared. We must, then, remove the focus. This statement is self-evident.

## IX. EXULCERATIO SIMPLEX OF THE STOMACH.

The two preceding sections have been devoted to hæmorrhagic erosions in which the course was rapid. There are, however, cases in which acute ulceration, with severe hæmatemesis, occurs, although we cannot find the



infectious origin of the gastric lesion, just as it often happens that the simple ulcer of Cruveilhier develops in the stomach without the original cause being found. I have reserved for these acute ulcers which supervene without apparent cause the name of **exulceratio simplex**. In future it will be necessary to describe **exulceratio simplex**, which is an acute lesion, and **ulcus simplex**, which runs a chronic course.

I have made a communication to the Académie de Médecine on **exulceratio simplex**, and I shall here quote two cases from my clinical lectures\* on this subject :

A man, twenty-seven years of age, was admitted under my care. His pallor at once pointed to severe hæmorrhage. He had had several attacks of hæmatemesis, and passed much blood *per anum*. He put the quantity of blood vomited at 3 pints. The pallor of the skin, the feebleness of the pulse, and the coldness of the extremities, all indicated that the bleeding must have been profuse. Although the classical signs of gastric ulcer were absent, I thought of a latent ulcer which had perhaps opened a large vessel. In spite of treatment, the bleeding recurred during the night ; next morning two basins were nearly filled with blackish blood and clots. The hæmatemesis had been followed by melæna, as on the previous day. Three pints of artificial serum were injected into the veins, and produced slight improvement. At one o'clock in the afternoon fresh hæmatemesis and death. He had lost about 7 pints of blood in less than thirty hours.

Here are the results of the autopsy : The exterior aspect of the stomach was normal. A ligature was placed around the cardia and the pylorus ; an incision was made in the stomach, which contained a point of blood, but, to our great surprise, we did not find the simple ulcer.

The mucous membrane was carefully washed, and we then discovered, half an inch from the cardia, a superficial circular **exulceration**.

The specimens made by Caussade showed the characters of the ulceration. It was so superficial as to affect only the tunica mucosa, with its muscularis mucosæ ; it had only broken down in places. At the bottom of the ulcer we saw the gaping arteriole which caused the fatal hæmorrhages. These details are shown in Fig. 29.

Fig. 30 shows miliary abscesses in the mucous membrane ; they open on the surface of the mucosa, and have made breaches of surface, which have left the muscularis mucosæ uncovered. This may have allowed the gastric juice to help in the work of destruction. The origin of the exulceration and of the fulminant hæmatemesis is thus clear.

It seems strange that this superficial ulcer and this small arteriole should have caused such profuse hæmatemesis. The calibre of an empty arteriole cannot, however, be correctly estimated in anatomical specimens, and the arterioles of the stomach, even those which are separated from the mucosa only by the muscularis mucosæ, are relatively of large calibre ; they equal in size the collateral arteries of the little finger. The ulceration had been latent and rapid in its progress, for our specimens showed recent lesions only. I was sorry on seeing this limited lesion in the stomach. I said to myself that surgical intervention might have saved the life of this man, and determined to seize the opportunity as soon as it presented itself.

The opportunity did present itself. On October 7, 1897, at 11 p.m., a young man, twenty-two years of age, brought up much blood. "I must have vomited," said he, "2 or 3 pints, for there was a regular pool on the pavement." The next day, "to pick

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\* "Exulceratio Simplex" (*Clinique Médicale de l'Hôtel-Dieu*, 1898, 1<sup>re</sup>, 2<sup>me</sup> et 3<sup>me</sup> leçons).

up strength," he ate well. About two o'clock next morning he again brought up blood. The vomited blood was brownish, part liquid and part clotted. On October 9 and during the following days he had neither pain nor vomiting, though he was very weak. He came to the Hôtel-Dieu on October 13. Next morning I was struck with

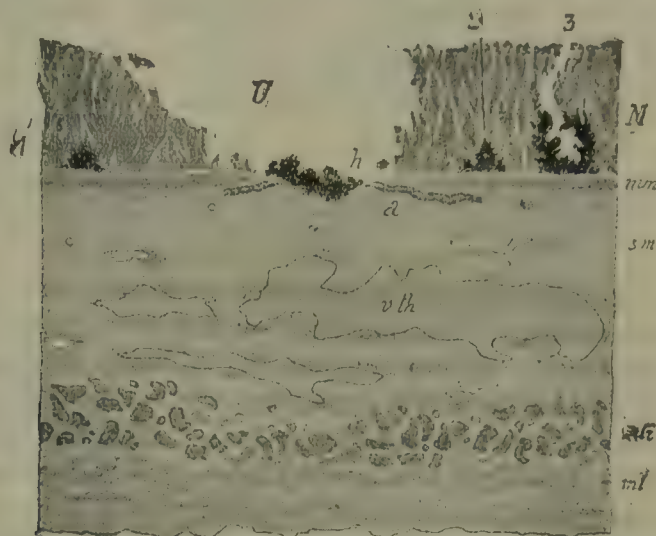


FIG. 29.—SECTION THROUGH THE WALL OF THE STOMACH.

U, Ulceration formed at the expense of the tunica mucosa M and of the muscularis mucosæ *mm*; *a*, small submucous artery destroyed at the point *h*, where a quantity of red blood-corpuscles are found *en masse* (it was from this spot that the fatal hæmorrhages proceeded); *v, th*, thrombosed vein; *sm*, tunica submucosa; *mtr* and *ml*, tunica muscularis; *s*, tunica serosa; 1, 2, 3, miliary abscesses, situated in the depth of the mucosa.

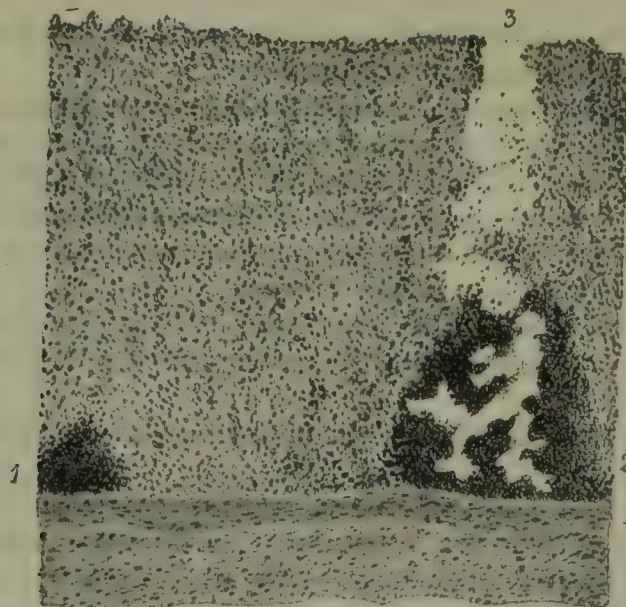


FIG. 30.—TUNICA MUCOSA, WITH ITS MUSCULARIS MUCOSÆ.

1, Miliary abscess in formation in the depths of the mucosa above the muscularis mucosæ; 2, miliary abscess, opening through the tunica mucosa, and communicating with the cavity of the stomach at the point 3.

the pallor of the skin and mucous membranes. The pulse was small, the temperature 100° F., and the patient complained of great weakness. I am sure that he spoke the truth when he claimed to have lost 5 pints of blood, for the blood-count gave only



1,600,000 red corpuscles. Though we had not verified the hæmorrhage, it was necessary to make a diagnosis, and to find out the origin of the bleeding. The case was evidently one of hæmatemesis, and not of hæmoptysis, because the blood had been vomited in streams, with enormous clots, and without the least cough ; moreover, the lungs were absolutely healthy. On the other hand, it was evident that the attacks were not due to the rupture of œsophageal varices, for he had no sign of cirrhosis. Some lesion of the stomach was therefore the cause. It was certainly not cancer, which is never ushered by violent attacks of gastrorrhagia. A simple ulcer, which so often causes hæmatemesis, remained ; he had never showed any symptoms of ulcer or other gastric trouble.

I felt sure that he had an exulceration like my other patient. The idea of surgical intervention at once entered my mind. We had not saved the first patient, in spite of intravenous injections, and therefore we must not let the second one die through indecision.

I commenced medical treatment, giving milk, which the patient vomited repeatedly. The analysis of the vomit showed that the hydrochloric acid was diminished.

Next day and the day following, the patient, who wished to get up, had a fainting fit ; fresh complications were arising.

When I arrived at the Hôtel-Dieu next day I was shown a basin containing 56 ounces of liquid blood with clots. During the night he had had a sudden attack of hæmatemesis. He was pale, drowsy, and prostrate. The pulse was almost absent. Further delay was impossible ; fresh hæmorrhage might come on, and the patient would assuredly die, as my other patient did.

After a preliminary injection of artificial serum Cazin operated. An incision 5 inches long, parallel to the edge of the false ribs on the left side, opened the peritoneal cavity. The stomach, partly hidden by the false ribs, was brought out of the wound and examined ; it appeared quite normal. Neither by sight nor by touch could we find any lesion ; ecchymosis, adhesions, and induration were absent. For a moment it appeared unnecessary to open the stomach, which, from the external examination, appeared healthy. Nevertheless, as the **diagnosis of exulceration had been made**, the operation was continued, and we showed the value of exact diagnosis and timely intervention. The contents of the stomach were squeezed into the intestine, and the viscus was clamped near the pylorus, to prevent regurgitation from the intestine. An incision 4 inches long was made in the anterior surface of the stomach, which was turned inside out to make the exploration easier. The stomach was empty.

Cursory examination seemed to show that the operation was ill-advised. The previous example, however, was not to be lost. When Cazin wiped the mucous membrane with sterile gauze, a bleeding spot appeared ; it was as big as a sixpence, and was situated in the mucous membrane.

When the ulcerated surface was gently rubbed with a tampon, the hæmorrhage reappeared over an area as large as a crown-piece. The case was one of exulceratio simplex in the mucous membrane, discernible only by careful search, and comparable to the exulceration which had killed my first patient.

I omit the details of the operation and subsequent treatment. Food was gradually given. Three weeks after the operation the patient's appetite was good ; he grew fat, and was discharged from the hospital five weeks after the operation, completely cured. The red corpuscles amounted to nearly 4,000,000. I saw him a year later, when he was in perfect health.

The third case I owe to the kindness of Michaux ; it is identical with the preceding ones. A healthy young woman suddenly vomited 2 or 3 pints of blood. During the night of October 22 and also next day she had fresh hæmatemesis and melæna. She was admitted into hospital on the 25th, and had lost so much blood that her skin and mucous membranes were colourless. As the hæmatemesis recurred, in spite of medical

treatment, and as danger was imminent, Michaux decided on operation. The stomach was opened and explored with care, but the operator did not find the simple ulcer which he expected. No adhesions; no induration; walls of the stomach supple. The patient was so weak that further manipulation was abandoned. During the next few days the situation became worse; the melæna persisted, and death supervened on October 31.



FIG. 31.—SHOWING THE ULCERATION.

The ulceration has taken place at the expense of the tunica mucosa *M* with the muscularis mucosæ *mm*; the other tunicae of the stomach, the tunica sub-mucosa *sm*, the tunica muscularis *m*, the tunica serosa *s*, are quite healthy. The superficial slanting arteriole *a* is destroyed at *h*.

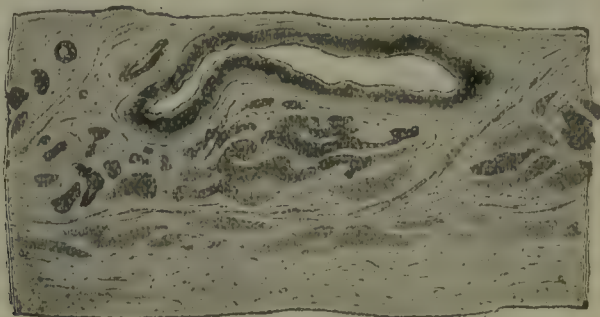


FIG. 32.—SECTION OF THE ARTERY (NO ARTERITIS) AT THE POINT WHERE IT REACHES THE ULCERATED SURFACE.

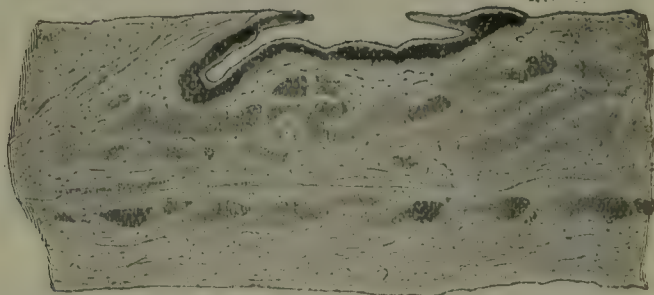


FIG. 33.—SECTION OF THE ARTERY AT THE POINT WHERE ITS WALLS, ERODED BY THE ULCERATIVE PROCESS, OPEN INTO THE CAVITY OF THE STOMACH.

Michaux has been kind enough to give me his specimens. The mucosa of the stomach shows a superficial circular ulceration, which has attacked the mucous membrane only, and is as large as a florin. In the centre there is a small cup-like erosion, traversed by an open arteriole.

Sections made by Du Pasquier show all the details. Fig. 31 represents the ulceration.

The arterial lesion which caused the hæmorrhage is seen in Figs. 32 and 33. The artery is healthy, and there is not a trace of arteritis; the vessel is seen running up to the tunica mucosa, until its walls are involved in the ulceration. It opens by a gaping aperture into the cavity of the stomach.



In this case, as in my first one, we find acute exulceration without previous vascular lesions. The ulcer has destroyed the mucosa and the muscularis mucosæ, and produced the fulminating hæmatemesis by erosion of one of the arterioles which ramify under the muscularis mucosæ.

In my clinical lectures on exulceratio simplex several cases are mentioned. They are absolutely identical with the preceding ones, and show that this lesion is far from being rare. Since my paper on this subject, several new cases have been published in France (Chauffard, Bichelonne and Petges) and abroad (John Lindsay Steven, Quintard, and Restis).

**Pathological Anatomy.**—The exulceration is generally circular; it may be elliptical or stellate. It often affects a large area, since it may be as large as a sixpence (my second case), a florin (Michaux's case), or more (my first case). We have not to deal here—at least, as far as appearance is concerned—with the small, punctiform, hæmorrhagic erosions described by Balzer and Pillet, and found in alcoholic, cardiac, or cirrhotic patients. In our cases, cirrhosis, heart disease, and alcoholism were not in evidence.

The exulceration may occur in any region of the gastric mucosa. It is supple; its edges are neither adherent nor thickened. They are fairly distinct from the surrounding healthy parts. The ulceration is in the mucosa, which must be smoothed out in order to show the lesion. Its depth is so little that in the living patient at the time of operation, and in the cadaver at the post-mortem examination, it may easily pass unnoticed without careful examination and a preconceived idea of its presence. In the neighbourhood of the ulceration reddish spots of an ecchymotic appearance are sometimes seen. In some cases it is possible to distinguish with the naked eye—or, better, with a lens—on the ulcerated part the open arteriole which has caused the bleeding.

I shall not dwell on the histological points of this lesion. Microscopic examination has always proved that the ulceration is due to the disappearance of the mucous coat, with its muscularis mucosæ. The rest of the mucosa of the stomach is generally healthy throughout its whole extent, and the arterioles of the ulcerated portion hardly ever show arteritis. In my two cases and in those reported by Brault and Giraudeau there was not a trace of it. The arterioles attacked by the ulcerative process were healthy, a proof that pre-existing arterial lesions are not needed to explain the ulceration and the resulting hæmatemesis.

Some authors consider that alcoholism is of importance as leading to simple ulcer, but “everyone is aware,” says Letulle, “that the simple ulcer is common in sober persons whose arteries are healthy.” This remark applies to most cases of exulceration, and amongst the cases which I have collected, I can only find alcoholic gastritis in two of them.

**Summary.**—Exulceratio simplex is not due to tuberculosis, syphilis,

alcohol, or uræmia. What is it, then? Everything considered, we must admit that it is the result of an acute toxi-infectious process, and I may add that in two of my cases the deep parts of the ulcerated mucous coat were the seat of small collections, probably little miliary abscesses, resulting from toxi-infection. These abscesses, as may be seen in Fig. 30, had destroyed the mucosa in places, and emptied themselves into the cavity of the stomach, leaving the underlying layers without any defence. Similar abscesses were found in the cases of Lépine and Bret. In one of Giraudeau's cases the exulceration did not extend beyond the muscularis mucosæ, and attacked an arterial branch. The vessel was not quite divided, and no **trace of arteritis** was present in the walls, which had resisted the ulcerative process. Furthermore, and an important point, in the neighbourhood of this vessel masses of leucocytes were seen representing true miliary abscesses, with numerous micro-organisms in little chains.

The process which ends in exulceration is rapid in its progress. The exulceration in my patient was of recent date, and the histological examination did not reveal any chronic lesion. It was the same in Michaux's patient, and the exulceration in Giraudeau's patient was likewise the result of a recent process. In the case reported by Luys everything leads to the supposition "that the lesion was of recent date," and Brault also insists that the exulceration which caused the death of his patient was "of recent date."

The toxi-infectious origin of exulceratio simplex and the rapidity of its course agree with what we know of the pathogenesis of acute ulcerations of the stomach. This fact was explained in Sections VII. and VIII. Letulle has expressed the opinion that "the ulcer is often the hidden relic of an infectious disease." In a woman who had died from puerperal fever Letulle found in the stomach two small clean-cut ulcerations. The underlying veins were thrombosed, and contained many streptococci. Following the same idea, Widal and Meslay have reported the case of a youth who had a corn on his right foot. After incision cutaneous abscesses and symptoms of toxi-infection soon appeared, and caused death. Post mortem, signs of general infection were found, abscesses or suppurative infarcts in the myocardium, kidneys, lung, and epididymis, due to *Streptococcus aureus*. In the stomach there was an acute ulcer of large size.

The experimental investigation of acute ulceration of the stomach is also in favour of a toxic origin. Letulle produced ulceration of the stomach in a guinea-pig by injecting staphylococci into the peritoneum, after having first filled the stomach with solution of bicarbonate of soda. Enriquez and Hallion have produced gastric ulcerations in the guinea-pig by injecting diphtheritic toxine under the skin of the abdomen, and Bezançon and Griffon have induced acute gastric erosions by the introduction of the pneumococcus into the peritoneum of a guinea-pig.



It was still essential to determine the origin of the toxi-infection. Was it preceded by infectious lesions, such as angina, furuncles, otitis, etc.? It was not possible to prove this fact in my patients. In them the exulceration supervened whilst they were in perfect health.

**Description.**—Events usually run the following course : A young person who has previously had neither gastric pain nor dyspeptic troubles, and is apparently free from any gastric lesion, is suddenly seized with malaise, vertigo, nausea, and weight in the stomach, and vomits 1 or 2 pints of blood, which may be liquid or partly clotted. The bleeding is followed by weakness, and if the first stools are examined, melæna will be found. The first attack is rarely fatal, but, on the other hand, it very rarely happens that the attack does not recur, and a few hours later or on the following day or the day after, a second, third, or fourth severe attack of hæmatemesis takes place, and is accompanied by vertigo and syncope, so that in twenty-four to forty-eight hours the patient vomits several pints of blood. You find him breathless and livid, with colourless mucosæ and inaudible voice. The pulse is small and quick, and the temperature is often raised ; but the patient feels no pain, and the stomach is not tender on palpation.

The exulceration is almost always rapid and latent ; it may, indeed, be said that **its clinical history commences with the hæmorrhage**. Let us review our cases, and it will be seen that, with the exception of two or three amongst them, who complained of some gastric symptoms, the others had experienced nothing that might draw attention to the condition of the stomach. They were not dyspeptic, and had had neither pains in the stomach, nor fits of vomiting, nor gastric intolerance. They were taken suddenly ill whilst in perfect health with these severe attacks of hæmorrhage. In my first patient who died, the attacks of hæmatemesis came on suddenly without previous warning ; my second patient, who was cured by the operation, was walking home when he commenced to vomit blood, without any previous gastric symptoms. A young woman whom we had in the St. Jeanne Ward was seized with severe and unexpected hæmatemesis. A young girl whom I saw in consultation with Grünberg had never had any gastric symptoms, when she was attacked, while walking, with fulminant hæmatemesis. The patient operated upon by Michaux had never had any fits of vomiting or gastric pains. The first symptom of the exulceration was hæmatemesis. The young girl who died whilst under the care of Brault had never had any stomach troubles when the severe attacks of hæmatemesis which ended fatally, suddenly appeared. A patient whom I had in the Hôtel-Dieu, and who had not the slightest gastric trouble previously, was seized with such severe attacks of hæmatemesis that the blood-count showed only 630,000 red blood-corpuscles.

Another fact has attracted my attention—namely, that hæmorrhages

in exulceratio simplex are severe from the first, and are not preceded by slight premonitory bleeding; they are not preceded by vomiting of blood like coffee-grounds, which is so frequent in ulcer simplex. From the very first they amount to a pint or more, and this severe hæmorrhage takes place in a few moments, and is at times followed by melæna. Whilst fatal gastrorrhagia is relatively rare in ulcer simplex, lightning-like hæmatemesis is much more frequent in exulceratio simplex, in which it constitutes the most important sign and the sole danger. Pathological anatomy will perhaps be able to enlighten us on these differences between gastrorrhagia in ulcer and in exulceration.

The chronic course of the ulcer lends itself better to vascular obliteration, whilst the acute process in the exulceration rapidly affects the muscularis mucosæ and the arterioles. It attacks the vessel and destroys its wall **laterally**. The hæmorrhage is much worse when an artery is opened laterally than when it is completely cut across. In the former case the retraction of the walls and the formation of the clot are not enough to arrest the hæmorrhage. Accordingly, in exulceratio simplex the hæmorrhage is terrible and in some cases fulminant. My first patient lost 7 pints in less than thirty hours; my second patient had lost 7 pints of blood when I advised operation. Michaux's patient lost several pints of blood, and the same may be said of Brault's case; Gilbert's patient died of fulminant hæmatemesis. Two young women, of whom I shall speak later, lost several pints of blood in forty-eight hours. The greatest peril of **exulceratio simplex** lies, then, in the quasi-fulminant attacks of gastrorrhagia, which show themselves by hæmatemesis and melæna.

We are ill acquainted with the chemical composition of the gastric juice during exulceratio simplex. It might, *a priori*, be supposed that here, as in ulcer simplex, excess of acid would be found, and yet in Lépine and Bret's case no free hydrochloric acid was discovered. In my second patient I found a condition of hypochlorhydria. I bring forward this fact, which does not agree with the theories, and which proves in any case that exulceration, like ulcer, may occur although the amount of hydrochloric acid is diminished. Another point to be noticed is the febrile condition of patients suffering from exulceration, and we find in almost every case a rise of temperature. Why this rise of temperature? The youth in St. Christopher Ward had a temperature of 100° F., and in Michaux's patient the reading was 102° F. In the case of the young woman in St. Jeanne Ward the temperature remained high for three days, and the young girl whom I saw with Grünberg had a temperature of 102° F. for several days. Another point to be noted is the predilection of exulceration for young people.

**Diagnosis.**—We have said that the evolution of exulceratio simplex is rapid and latent, revealing its presence suddenly by terrible attacks of



hæmatemesis, and but imperfectly heralded by weight in the stomach, with or without nausea, vertigo, and tendency to syncope. These attacks of hæmatemesis may be fatal, unless recourse be had to operation, so that the question resolves itself into making a correct diagnosis.

If the patient is seized with sudden and severe hæmatemesis, a double diagnosis must be made—firstly, to prove that it is a case of hæmatemesis; and, secondly, to ascertain the lesion that has provoked it. A patient tells you that he has been vomiting blood. Has he had an attack of hæmatemesis or of hæmoptysis? Hæmoptysis is sometimes so copious and the blood is brought up with such violence that the patient speaks of vomiting, though the blood really comes from the lung, and not from the stomach. The blood in hæmoptysis is brought up during fits of coughing; it is frothy and bright red, which is not the case in hæmatemesis. Furthermore, the pulmonary lesion which causes hæmoptysis indicates the cause and seat of the hæmorrhage. The opening of an aortic aneurysm into the trachea or bronchi must also be considered. In this case the patient vomits an enormous quantity of blood, which must be differentiated from hæmatemesis. It is true that death is the immediate consequence.

When hæmatemesis is proved, it is necessary to localize its origin. Blood may pass from the nose into the stomach and be vomited. I have seen two cases of this kind.

A bloodless patient came under my care for hæmatemesis, attributed to a lesion of the stomach. Examination showed, not a gastric lesion, but an angioma of the nasal fossæ. The blood ran down the œsophagus and accumulated in the stomach, whence it was rejected by hæmatemesis. The angioma was cauterized by Luc, and the bleeding at once ceased.

Œsophageal and gastric varices (Letulle) in cirrhosis of the liver may rupture and cause hæmatemesis.

A patient came under my care for cirrhosis of the liver. Whilst we were examining him, he was seized with nausea, and twice, at a quarter of an hour's interval, he brought up 2 pints of blood. In spite of treatment, he died the next day. At the post-mortem examination cirrhosis of the liver was found; at the lower end of the œsophagus there was a varicose plexus; one of the veins was ulcerated, and the blood had filtered into the stomach and had been subsequently vomited.

In such a case we have as a guide the symptoms of hepatic cirrhosis. If the cirrhosis is early, it is possible, thanks to "the minor signs of cirrhosis" (Hanot)—urobilinuria, subicteric tint, hæmorrhoids, epistaxis, alimentary glycosuria, and abdominal tympanites—to recognize the disease and the origin of the hæmorrhage.

Severe hæmatemesis with melæna has also been found as a complication of gall-stones. In this case the bleeding is due to an aneurysm of the hepatic artery, which opens into the large bile-ducts. I have seen a case of this kind.

It has been thought that hæmatemesis might be due to infective angiocholitis. Rondot has published the history of two patients who had symptoms of ulcer of the stomach and severe hæmatemesis. At the post-mortem examination an abscess of the liver was found in one patient and suppurative cholecystitis in the other. The report mentions that the stomach also presented traces of ulceration, which were examined microscopically. I therefore put the question whether the trouble must not be laid rather to the charge of the ulceration than to the angiocholitis?

Cancer of the stomach causes attacks of hæmatemesis, but they are preceded by many other signs, and, moreover, the bleeding is not copious. Severe hæmatemesis is seen in ulcer. If cancer of the stomach is accompanied by severe hæmatemesis, it is because the cancer is grafted on an ulcer.

From this discussion it is evident that, with a few exceptions recognizable by clinical examination (varices of the œsophagus and stomach, hepatic cirrhosis), profuse hæmatemesis is due to simple ulcer and exulceratio simplex. Let us attempt a differentiation.

In exulceration, hæmatemesis is generally the first symptom; previous gastric troubles are absent or slight. In ulcer, hæmatemesis is preceded or accompanied by the classical symptoms. The ulcer may, it is true, have been latent, but scrutiny shows that this so-called latent state is often only relative. Hæmatemesis in exulceration presents some interesting features. When I refer to my case-book, I find that the hæmatemesis is severe from the first, and is not preceded by the slight attacks of vomiting of blood like coffee-grounds which are so frequent in *ulcus simplex*. The latter, it is true, also causes hæmorrhage, which may become fatal on account of its abundance or its repetition. I consider, however, the hæmatemesis of exulceration as more to be feared than that of ulcer. I have had many patients suffering from ulcer, but, though several died from perforation, cachexia, or malignant changes, and others had severe attacks of hæmatemesis, fulminant hæmorrhage was exceedingly rare.

Let us see what happened in the ten cases of exulceratio simplex discussed in my clinical lectures. Seven died more or less quickly, after losing several pints of blood, from ulceration of an arteriole. Post mortem the same lesion was found in every case, the exulceration having attacked or destroyed the arteriole. As for the patients who recovered, they also looked as if the hand of death had been placed upon them in the midst of perfect health. The gastrorrhagia was so copious that the blood-count showed only 1,200,000 red corpuscles, and in a recent case at the Hôtel-Dieu the corpuscles were only 630,000. I am therefore correct in stating that hæmatemesis in exulceration is more acute and more sudden, as a rule, than in ulcer. In a word, sudden hæmatemesis, which is threatening from the very outset,



and often fatal, is the dominant symptom in the clinical picture of exulceratio simplex.

**Prognosis.**—I have just explained how death from exulceration comes about, and I should like to mention those cases that recover. What is their future? Are they freed, once and for all, from this terrible eventuality? Does exulceration predispose to a second attack, like ulcer, or is it not the initial stage of Cruveilhier's *ulcus simplex*, arrested in its course by fulminant hæmatemesis or by appropriate treatment? To prove this hypothesis, it would be necessary to find a typical ulcer and an exulceration in the same stomach. It would, then, be rational to suppose that we had before our eyes the simple ulcer in the various stages of its development. With this idea in view, I have succeeded in finding a case which seems clear. The case is as follows :

A man who had had no previous gastric symptoms was admitted into hospital with signs of internal hæmorrhage—viz., pallor, cold sweat, and tendency to syncope. After dinner he suffered from severe malaise, was seized with fulminant hæmatemesis, and died. At the post-mortem examination three ulcers were found, placed one behind the other, on the lesser curve of the stomach. The middle ulcer was a typical *ulcus simplex* ; it was the size of a shilling, round, and had prominent, clean-cut edges. The floor was reddish, mammillated, and a divided artery, obliterated by a clot of fibrin, was found on section. To the right and left of this ulcer there were two slight ulcerations, one of which was of the size of a lentil, while the other ulceration was superficial, elliptical in shape, and about as large as a halfpenny. Although no histological examination was made, it had all the characteristics of an *exulceratio*. The floor was traversed by a vessel, with a lateral opening visible under water and under the lens. On reading of this case, I thought I had found a case in which the lesion existed both in the stage of confirmed ulcer and of limited exulceration.

We may, then, admit that, until the contrary has been proved, *exulceratio simplex* may be an initial stage of Cruveilhier's *ulcus simplex*. Moreover, Cruveilhier, in his excellent description of simple ulcer, has rightly said that "In the first degree the ulceration is limited to the mucous membrane, and is generally a follicular erosion."

I believe that *exulceratio simplex* is not subject to recurrences. A patient who is cured of exulceration need not fear a return thereof, because the toxi-infectious process which produced the ulceration is extinct.

**Treatment.**—Let us take the situation in a case analogous to one of those which we have just described. You are called to a patient who has been seized with severe hæmatemesis while in good health. The blood is there, liquid or in clots, and the quantity is alarming, for 2 or 3 pints have been brought up at one time. The patient is anxious, ready to faint, and extremely pale. The pulse is quick, and the temperature slightly raised. You make inquiries, and formulate the following diagnosis : The patient has just had a terrible attack of hæmatemesis, in consequence of ulceration of the stomach, and everything points to *exulceratio simplex*.

What are you going to do? Are you to advise immediate operation? As you know that death has followed the first or second attack of hæmatemesis, be ready in case surgical intervention should become urgent, but first try medical treatment. The immediate indication is to keep your patient absolutely at rest, with an ice-bag on the abdomen. In my opinion, the patient should take absolutely nothing—not even a spoonful of water nor a morsel of ice. I consider that anything which may induce movement of the stomach or secretion of the gastric juice is harmful. An arteriole has been opened, and the hæmorrhage has been stopped for the moment by a clot which will later obliterate the vessel. Do not give anything which may hamper the process of repair. Fluid by the mouth provokes contraction of the stomach and secretion of the gastric juice, conditions which are unfavourable to the solidification of the occluding clot. I reject, therefore, in the most absolute manner, preparations of gelatine and hæmostatic draughts. All medication must be carried out hypodermically or per rectum. The patient must be fed by means of nutritive enemata of peptones, eggs and milk, while ergotin or ergotinin is given hypodermically; and even then these remedies, though most efficacious in uterine hæmorrhage, are here of less utility. Artificial serum should be given by subcutaneous injection. It may be asked theoretically if these injections do not affect the process of coagulation in the vessel, by raising the arterial tension, or if too large a quantity of serum, rapidly introduced, has not a bad effect on the small quantity of blood still remaining in the body? Experience proves that careful injections of serum are of service. It is not necessary that they be intravenous. The injections may be repeated several times a day, and 10 to 15 ounces of serum may be given at a time.

The young woman whom I saw with Grünberg took some 26 pints in twenty-nine days. You may employ a serum containing 0·7 per cent. of chloride of sodium (Malassez's formula), or a serum containing 75 grains of chloride of sodium and 150 grains of sulphate of soda (Hayem's formula), to 35 ounces of water. The serum which I employ contains 105 grains of chloride of sodium and 2 grains of benzoate of caffein to 35 ounces of water. I have held to this formula, because it seems to me to answer all purposes. (For further details, see Appendix on Therapeutics.)

Inhalations of oxygen and injections of ether are of benefit in case of syncope. The above treatment is carefully watched. The patient must take **no food** for three or four days; after that time a few spoonfuls of water, and then a few spoonfuls of milk diluted with water, are given, the amounts being gradually increased. You rarely succeed in stopping the hæmorrhage at first, and fresh attacks of hæmatemesis may occur. In certain cases the situation becomes dangerous: the restlessness increases, the pulse becomes quicker, the pallor more pronounced, and the fainting-



fits succeed one another. You do not dare to leave the patient, and are rightly afraid of fatal syncope. You ask yourself whether fatal hæmatemesis may not occur at any moment, and you think of surgical intervention.

The difficulty is great, for you must now take the responsibility of choosing the right moment for surgical intervention.

What, then, is the right moment in the case of severe hæmatemesis due to exulceratio simplex? It is certainly true that the above treatment is of great value, but it has its limits. My first patient succumbed, although he had received 3 pints of serum. Brault's patient succumbed, though large and repeated injections, including an intravenous one of 3 pints, were given. Medical treatment is therefore at times powerless, and surgical methods have a better chance of success if they are applied before fatal syncope is imminent. What, then, are the indications showing that the operation is urgent? Is it necessary when the number of red corpuscles has been reduced to a certain number? Can it be said, for example, that an operation is necessary as soon as the number of red corpuscles has been reduced to 1,200,000? I do not think that this is the sole criterion, for Lépine and Bret's patient succumbed when he had 1,300,000 corpuscles; and, on the other hand, one of my patients at the Hôtel-Dieu recovered, though he had only 630,000 corpuscles. These cases show that a blood-count alone is no guide.

I do not know of any exact sign by which to decide when operation is urgent. The decision must rest upon a survey of all the facts. Account must be taken of the state of the pulse, the strength of the patient, and his powers of resistance. In short, we must not only know when to wait, but also when to decide. I am convinced that operations for hæmatemesis due to exulceratio simplex will give brilliant results, for they are **infinitely more simple** in the case of exulceration than of ulcer. In the case of exulceratio there are no adhesions, no perigastric lesions, and no large ulcers in the stomach. On the contrary, there is a very limited superficial lesion which lends itself to operation. The lesion is so superficial, and at first sight so little apparent, that it may escape notice if previously unsuspected. Remember what happened to operators until the anatomical and clinical history of exulceratio came to be written.

If a patient was seized with severe and repeated attacks of hæmatemesis, he was generally thought to be suffering from ulcer simplex, and was handed over to the surgeon. The surgeon, certain of finding an important lesion, such as adhesions, induration of the walls of the stomach, or a large tumour with thick edges, opened the stomach, and was much surprised at the absence of the expected lesion. He believed that there had been an error in the diagnosis, and closed the stomach, or else performed gastro-enterostomy, without accounting for what had really happened. A similar mistake

has been noted in several cases. Michaux opened the stomach, with the full conviction that he would find an ulcer; but he did not find it, and the exulceratio simplex, which was the cause of fatal hæmorrhage, passed unnoticed, and the mistake was only found at the post-mortem examination. Hartmann opened the stomach, expecting to find an ulcer, and failed to discover the exulceratio simplex which gave rise to the fatal hæmorrhage, and was only found later at the autopsy. In the case of the patient upon whom Cazan operated at my request, the stomach was opened, and at first sight appeared healthy. As I felt certain that an exulceratio simplex was present, it was looked for, found, and ligatured, with the result that the patient recovered.

It must not be said, as has been incorrectly advanced, that it is impossible to find an exulceratio simplex at the time of operation, and I protest against such an assertion. Until recently we were not familiar with exulceratio simplex. No monographs had been written, and it had attracted so little attention that when the stomach was opened **it was not seen, because we had not been taught how to look for it. It passed unnoticed, because its existence was unknown.** We have therefore a new chapter to add to our accounts of ulceration of the stomach. In future the physician will make his diagnosis, and when the surgeon has opened the stomach for hæmatemesis due to an exulceration, he will know that the stomach may at first sight appear perfectly healthy, and that he must look for the exulceratio simplex with the most minute care. He must examine the mucosa, cleansing and smoothing it out, and, if necessary, using a lens, when he will find the exulceration, which may vary in size from a lentil to a shilling. He will discover it the more easily as the exulceration, no matter how superficial it is, generally occupies a certain surface and is sometimes surrounded by ecchymoses, which serve as a guide.

## X. SIMPLE ULCER OF THE STOMACH (CRUVEILHIER'S DISEASE).

Several questions concerning simple ulcer have been discussed under Exulceratio Simplex; they will also be mentioned in Section XIII. I would therefore ask the reader to study these sections together.

**History.**—We have already seen that the acute ulcerative lesions of the stomach are numerous, and that their origin is multiple. The present description refers to a chronic variety which has been called **simple ulcer** (Cruveilhier), **perforating ulcer** (Rokitansky), **ulcus rotundum** (Niemeyer); it is peculiar to the stomach, œsophagus, and duodenum, and was first described by Cruveilhier.

In the year 1830, he described the simple chronic ulcer, and separated it from cancer of the stomach, with which it had previously been confounded. He completed his work in 1838, and has left us so complete a clinical and anatomical picture of the disease that this malady rightly bears his name. Rokitansky's monograph is dated 1839. I



may be permitted to state that the clinical distinction between ulcer and cancer of the stomach is not always absolute. Cruveilhier himself noted the coexistence of both lesions in the same stomach, and during the past few years several cases have been noted which prove that gastric ulcer is sometimes only the prelude of cancer, or, rather, cancer frequently grafts itself on the ulcer or its cicatrix.

The name *ulcus rotundum* is suitable; the ulcer, however, has not always a rounded form, and the name of *perforating* is no better, because the ulcer does not always end in perforation. The name *simple ulcer* should therefore be preserved.

**Pathological Anatomy.**—According to statistics collected by Rosenheim and Brinton, the situation of ulcer is as follows :

|                               |    |    |    |
|-------------------------------|----|----|----|
| Posterior wall of the stomach | .. | .. | 83 |
| Pyloric region                | .. | .. | 36 |
| Lesser curvature              | .. | .. | 37 |
| Anterior wall                 | .. | .. | 26 |

The ulcer is generally rounded. It has a semilunar or circular form when it embraces the pyloric region, but is large and irregular when several ulcers have coalesced.

The ulceration tends to destroy successively the mucous, fibrous, muscular, and peritoneal coats, and the depth of the ulcer varies, therefore, according to the coat destroyed. As regards its anatomical course, the simple ulcer commences as an erosion, forms an ulceration, and ends by cicatrization or by perforation.

On the mucous aspect, the ulcer appears funnel-shaped, the orifice of the ulcer being wider than the bottom. Its edges are not ragged, but clean-cut, like a punch-hole. On the edges and the floor of the ulcer we often find arterioles cut through, while the lumen has been obliterated by a plug of embryonic tissue. When the lesion is old, the edges of the ulcer may become so indurated and thickened as to simulate cancer. The dimensions of the ulcer are variable. Some are hardly as big as a sixpence, while others may be larger than a crown. The ulcer may be single or multiple. According to Brinton it is double once in five times, while as many as five have been found in the same subject (Rokitansky). Post mortem, it is no uncommon thing to discover the scars of healed ulcers. The mucous membrane is absent over these areas.

Recovery is frequent, but sometimes occurs at the expense of scars, which constrict the pylorus or produce an hour-glass stomach. When the ulcer terminates in perforation, the peritoneal opening, which varies in size, is as clean-cut as the gastric opening. The perforation in some cases opens into the peritoneal cavity, and if adhesions are absent fatal peritonitis is the result, unless an early operation is performed. In other cases the perforation is limited by the adhesions to a neighbouring organ, which forms a tampon, and in turn becomes ulcerated. In this way the floor of the ulcer is formed, as the case may be, by the diaphragm, liver, spleen, pancreas,

mesenteric glands, or sternum (Barth). In other cases the perforation establishes a fistula between the stomach and the duodenum, colon, bronchi, pericardium, or skin. The perforation may give rise to subphrenic abscess, which we shall study later. As the ulceration does not spare the arteries of the stomach (coronary, splenic, gastro-epiploic vessels), fatal gastrorrhagia may result.

The ulcer, even in the absence of perforation, causes adhesions between the stomach and the neighbouring organs. This fact often complicates the technique in the case of surgical intervention. The microscopic examination of the tissues bounding the ulcer shows the lesions of gastritis. The glands show a tendency to disappear, the cells are affected by fatty degeneration, and masses of embryonic cells appear under the mucosa in the cellular tissue, while the submucous muscular tissue is broken and the subjacent tunicae musculares are invaded.

**Ætiology—Pathogenesis.**—Is simple ulcer of the stomach a distinct morbid entity, or is it only the end of the various erosions and ulcerations which may at a given moment pass into simple ulcer? Both opinions have been upheld. Whichever be the theory preferred, how can we explain the process which takes the chief part in the formation of this ulcer? It has been held that the ulceration at its commencement is only an ecchymotic patch caused by blood-stasis, due to fatty or atheromatous degeneration of the small vessels. Superficial necrobiosis of the mucosa is the result, and the gastric juice, by digesting the walls of the stomach, becomes the chief factor in the morbid process.

The action of the gastric juice appears the more likely to many writers, because patients suffering from simple ulcer of the stomach, often suffer from hyperchlorhydria or excess of acid, during digestion, and from hypersecretion, or continuous excess of hydrochloric acid, even apart from digestion. It must be admitted that hyperchlorhydria, hypersecretion, and ulcer of the stomach seem to belong to the same pathological family. Patients begin with hyperchlorhydria or hypersecretion and end with ulcer. These facts would decide in favour of those who hold that the gastric juice may cause the ulcer by digestion of the tissues (autodigestion). On the other hand, how are we to reconcile this hypothesis with the fact that simple ulcer is found in the œsophagus—that is to say, in a region where the gastric juice is absent? and, above all, how are we to reconcile this hypothesis with the fact that in the same stomach two ulcers may run opposite courses, the one burrowing, thanks to the action of the gastric juice, and the other cicatrizing, in spite of it? This fact is not favourable to the theory.

The germ theory may explain the formation of the ulcer, which is at its commencement only an exulceration. As I have discussed this question



under *Exulceratio Simplex*, I shall not refer to it again. The theory that compares gastric ulcer with perforating ulcer of the foot is ingenious, but not proven. Alcoholism has been looked upon as the chief cause of gastric ulcer. This view is wide of the truth, as Letulle has pointed out, because in most cases the patients are not alcoholic and the arteries of the stomach are not atheromatous.

According to Gilles de la Tourette, hysteria has an important place in the causation of ulcer.

Simple ulcer is fairly common, especially in England and Germany. It is found more frequently in women than in men, and chiefly in young adults. Young women of the chlorotic type are prone to it. Traumatism (blows, contusions of the epigastrium) may perhaps be the cause in a certain number of cases (Potain). In short, the real cause of *ulcus simplex* frequently escapes us.

**Symptoms.**—In the classical form the symptoms are as follows: After a varying period of dyspepsia, and often of hyperchlorhydria, the cardinal symptoms of pain, vomiting, and hæmatemesis appear.

The **pain**, which is generally limited to the xiphoid point, is almost always accompanied by a corresponding pain in the spine, on a level with the first lumbar vertebra (rachidian point, Cruveilhier). This gnawing and boring pain returns several times a day, or at more distant intervals. I have seen patients in whom these pains, like those of severe burns, were so acute that the relief could only be obtained by the excessive use of morphia for months at a time. The pain is increased by pressure or palpation of the stomach. It is generally brought on by the ingestion of food, and persists during the whole period of gastric digestion, being most severe at this time. It must not be forgotten that pain is sometimes absent, and that similar pain may be present in certain cases of gastralgia, especially with hyperchlorhydria. In some cases the gastric intolerance is absolute, and milk or even water is rejected.

**Vomiting** is frequent. Vomiting of food takes place more or less rapidly after meals; indeed, the pain may only cease with the vomiting. Some patients vomit phlegm tinged with bile during the day, and especially in the morning. The vomit is often very acid, because in ulcer of the stomach the gastric juice usually contains hydrochloric acid in excess. It is the presence or excess of hydrochloric acid in the chyme which in difficult cases is one of the signs of diagnosis, though by no means an absolute one, between ulcer and cancer.

Vomiting of blood (hæmatemesis) varies according to the case. If the hæmorrhage into the stomach is copious, and if the blood is quickly vomited, it is red and liquid. If, however, it has remained for some time in the stomach in contact with the gastric juice or the food, it is voided in clots,

or more usually in the form of a blackish liquid, like coffee-grounds or soot diluted with water. This is called the **black vomit**.

Sometimes a portion of the blood passes from the stomach into the intestine, and the patient voids tarry stools. This is called **melæna**. If the hæmorrhage is not copious, and occurs slowly in small quantities, the blood may pass into the intestine, and the melæna is then the only sign of the gastric hæmorrhage.

Attacks of hæmatemesis are also possible when the ulcer has cicatrized, and a fatal hæmorrhage takes place by a fresh lesion of the cicatrized tissue, as the following case proves (Bonnet) :

A woman who had had no previous illness showed all the symptoms of ulcer of the stomach, including xiphoid and spinal pains, increased by the ingestion of food and vomiting. During the course of the illness severe hæmatemesis supervened, and on the following day a second hæmorrhage, more copious than the first one. The patient was put on a rigid milk diet, and at the end of nine months she was completely cured of her trouble. Eight years later the patient was admitted into the Croix Rousse Hospital, having just been seized with copious hæmatemesis and melæna. She complained of pains in the pit of the stomach. She was put on a milk diet, and ice-bags were applied to the abdomen. At eight o'clock in the evening the pains increased, and the patient was seized with a terrible attack of hæmatemesis, amounting to about 4 pints of blood. An injection of ergotin was given, then one of serum, but the patient died at ten o'clock. At the post-mortem examination large clots were found in the stomach, and on the small curvature there was the cicatrix of the old ulcer, in which there was seen a small conical projection, terminating in a gaping orifice; the latter was the opening of an arteriole, the walls of which had been attacked. (It might be called an exulceration grafted on the cicatrix of the ulcer.)

**Dilatation** of the stomach frequently accompanies ulcer. It is not due only to organic stricture of the pylorus, but is also found associated with ulcers of other regions. The spasmodic reflex contraction of the pylorus is, without doubt, an important factor. This contracture is said by Doyen to play a considerable part in the symptoms of gastric ulcer, and is held to be the origin of the cramps in the stomach. It is also said to be one of the factors in the dilatation and vomiting, as well as an obstacle to the healing of the ulcer by provoking dilatation of the organ and consequent dragging on the edges of the ulcer.

**Headache** is a symptom which, I notice, is nowhere mentioned, but which I have often met with in gastric ulcer. I am not quite certain of the cause, but from a semeiological point of view I mention its importance. The violent and obstinate headache is often as distressing to the patient as the epigastric pain, and seems to me to be of value in diagnosis, for it accompanies ulcer, but not cancer.

The patient wastes quickly, as the result of the pain and vomiting. Dysmenorrhœa and amenorrhœa are found in women. The disease finally produces cachexia.



The possible terminations of simple ulcer are as follows: complete recovery, recovery with cicatrices or with extragastric adhesions; death from hæmorrhage, perforation, peritonitis, collapse, cachexia, or malignant change. Recurrences are fairly frequent, and the symptoms may appear again after some months or years of cure. Fresh ulceration sometimes appears in an old cicatrix.

**Complications.**—Amongst the terrible and fatal complications let us mention first **hæmorrhage**, which, like perforation, may result from a latent ulcer. I saw a case of this kind with Caussade.

A woman, who had shown no symptoms of gastric ulcer, was seized with severe hæmatemesis. The bleeding ceased, but she died, a few weeks later, from perforation. In one of Bazy's cases the patient died in a few hours from hæmorrhage, and at the post-mortem examination three ulcers were found in the lesser curvature; the ulceration had destroyed the gastric artery. In Caillard's case the patient was carried off in a few hours by hæmorrhage. At the post-mortem examination an ulcer of the lesser curvature, involving the pyloric artery, was found. In a case reported by Litten the patient died of repeated hæmorrhages from the stomach, and post mortem a perforation in the middle of the posterior wall and ulceration of the splenic artery were found.

**Peritonitis.**—**Perforation** and **acute general peritonitis** are most common in ulcers on the anterior surface of the stomach, because this region is more mobile and more accessible to traumatism, and because adhesions do not form. The seat of election differs, therefore, in ulcer and in perforation, the ulcer being more common on the posterior surface of the stomach. As regards statistics, perforation is forty times more common in anterior ulcer than in posterior ulcer. Ulcers on the anterior wall are, therefore, of exceptional gravity.

**Supercute peritonitis**, due to the perforation of the stomach, will be described in the next section. This form of peritonitis is rapidly fatal, unless surgical intervention is prompt. In the more favourable cases the perforation causes partial or encysted peritonitis. The adhesions have had time to unite the stomach to the diaphragm, the abdominal wall, the liver, or the transverse colon, and enclose an anfractuous abscess cavity, which is usually situated in the epigastric and hypochondriac regions.

These perigastric abscesses may be classified in several categories:

The **gastro-splenic** abscess is seated between the fundus of the stomach and the spleen, in the left hypochondrium, and is due to a perforation of the greater curvature of the stomach.

The **gastro-subhepatic** abscess extends towards the right hypochondrium, being limited above by the left lobe of the liver, below by the lesser curvature of the stomach and the pylorus, and in front by the adhesions which unite the edge of the liver to the stomach. It is due to a perforation in the pyloric region.

The **gastro-abdominal** or antegastric abscess is placed between the front of the stomach and the abdominal wall. It may cause inflammation of the anterior abdominal wall, with gastric fistula, and is due to a perforation of the anterior wall of the stomach.

**Subphrenic Abscess.**—The gastro-hepato-phrenic abscess, called also **subphrenic abscess**, or subdiaphragmatic pyopneumothorax, is the most common variety, and is usually due to a perforation of the posterior wall of the stomach. It forms a **subphrenic empyema**, which, like the preceding varieties, contains gas, putrefying fragments of food, and sloughy shreds. From a topographical point of view, the abscess is constituted in the following manner : Its superior wall is formed by the diaphragm, which, under the pressure of liquid and gas, may be pushed up as high as the third intercostal space. The lung is in its turn displaced, and the subphrenic collection, ascending into the right or left side of the chest, may simulate pleuritic effusion or pyopneumothorax. The inferior wall of the abscess is formed by the liver, stomach, and transverse colon, which are adherent and covered by false membranes. The right wall of the abscess is formed by the suspensory ligament, which joins the convex surface of the liver to the diaphragm and abdominal wall. The left wall is formed by the spleen adhering to the stomach and by the vertical portion of the diaphragm, while the posterior wall is formed by the coronary ligament, which extends from the posterior edge of the liver to the corresponding part of the diaphragm. The abscess, which contains pus and gas, occupies the epigastrium and the hypochondria, especially the left. The thoracic organs may be displaced.

The earliest symptoms are often epigastric pain and vomiting, but as these symptoms much resemble those of ulcer the onset of phrenic abscess may not be recognized. In other cases the abscess forms in an insidious manner, and either passes unnoticed or is masked by the urgent and painful symptoms of ulcer. Some patients complain of pains which radiate into the shoulder when the abscess is forming, while others suffer from rigors and fever, which indicate suppuration.

A subphrenic abscess, when it has formed, gives rise to **abdominal** and **thoracic** symptoms. The abdominal symptoms are pain, bulging, and deformity of the epigastrium and left hypochondrium. They are localized to the **supra-umbilical portion** of the abdomen, whilst the subumbilical portion remains supple, and shows no abnormality. This point is valuable in diagnosis.

The thoracic symptoms comprise enlargement of the base of the thorax and signs of pleural effusion or of pyopneumothorax. As the diaphragm may be pushed upwards, the signs of pleural effusion and also of pyopneumothorax are seen. The difficulty in diagnosis is apparent, but I



think that it may be thus overcome. Bulging, deformity, and dullness in the epigastrium and the hypochondria do not exist in pleural effusions nor in true pyopneumothorax. There are, however, cases in which effusion into the left pleura may deform the left hypochondrium and abolish the resonance in Traube's space. This happens when there is considerable effusion, amounting to 3 or 4 pints at least. With such an effusion, however, the heart is displaced outwards, and the dullness reaches the clavicular region, which is not the case in pseudo-pleural effusions of abdominal origin. There are also cases in which an effusion of the right pleura may depress the liver and cause bulging in the right hypochondrium, but here also it is a case of large effusions which cause dullness up to the clavicular region. This is not the case in pseudo-pleural effusions of abdominal origin.

The peritoneal abscesses just described may terminate in different ways : (1) they may bring about suppuration in the abdominal wall, with gastro-abdominal fistula ; (2) they may empty into the small intestine or into the colon, causing foetid diarrhoea and gastro-intestinal fistula, which may close up ; (3) they may open into the peritoneum and cause general peritonitis ; (4) they may perforate the diaphragm, and open into the pericardium, pleuræ (causing empyema and pyopneumothorax), or into the bronchi and lungs (causing gangrene or bronchopneumonia).

**Latent Ulcer.**—The description of gastric ulcer, as given above, holds good in the majority of cases. Dyspeptic troubles first appear, and are followed by pain, vomiting, and attacks of hæmatemesis. The disease may last months and years (**two years being the average**), with alternate improvement and relapse, which, with proper treatment, often ends in cure. The ulcer does not, however, always present the same form. In certain cases it is **latent**, or only betrays itself by slight dyspepsia, and an apparently healthy person is **suddenly** stricken with perforation of the stomach, acute peritonitis, ulceration of a large vessel, or violent hæmatemesis, which endangers life in a few days (rapid variety) or in a few hours (fulminant variety) (Jaccoud). These terrible complications are found in the ordinary chronic ulcer, **but then they are foreseen**. I cannot lay too great stress on this latent form, which causes such great surprises. Fatal peritonitis from perforation is more common than is generally supposed in cases where the ulcer has made progress, although pain, hæmorrhage, and vomiting are absent or **almost latent**.

The following cases will illustrate the point :

**CASE 1.**—An anæmic girl, who had never had stomach trouble, was taken suddenly ill during the night, with violent epigastric pain and all the signs of superacute peritonitis except vomiting. She died in fifteen hours from peritonitis, due to perforation of an ulcer on the anterior surface of the stomach.

CASE 2.—A healthy young woman was suddenly seized with very sharp pain in the left hypochondrium, and the temperature fell to 96° F. in a few hours. The abdomen was distended and very painful. The extremities grew cold, and the respiration was greatly quickened; the pulse could not be counted, and the patient died in twenty-four hours. At the post-mortem examination double perforation of the stomach was found on the anterior and posterior walls, the two perforations being almost opposite one another.

CASE 3.—A young girl, eighteen years of age, was suddenly seized with sharp pains in the abdomen. Symptoms of general peritonitis soon appeared. An operation was performed on the third day; the patient succumbed. At the post-mortem examination an ulcer with a large perforation was found.

CASE 4.—I saw, with Tisné, a young girl who had been suddenly taken ill with terrible pain in the abdomen; no vomiting. Next morning we diagnosed peritonitis from perforation, and I sent the patient to the Necker Hospital for operation. Her condition became so bad that the operation was not completed; she died twenty hours after the onset of complications. At the post-mortem examination we found two large rounded perforations on the anterior and posterior surfaces of the stomach. They were absolutely superposable, as if one ulcer had produced the other. The ulcers were of the latent type. The patient had never experienced any gastric symptom. I made a minute inquiry into the case. On the previous evening she had been out walking, and had made an excellent dinner.

CASE 7.—A young woman, twenty-six years of age, was admitted by Fernet for superacute peritonitis, which had come on soon after a meal. The woman, who was in good health, had felt some slight dyspeptic troubles, ascribed to chlorosis. She died twenty-four hours after the onset of the symptoms, and at the post-mortem examination two ulcers were found to have perforated through the anterior surface of the stomach.

These cases of latent ulcer allow me to draw the following conclusions: Latent ulcer of the stomach is especially common in young women, with or without chloro-anæmia. It is usually situated on the anterior surface of the stomach. The more latent the ulcer is, the more is it to be feared, because the patient follows no treatment, as she has not been warned by any symptom of the presence of the disease, which may kill her as by a flash of lightning.

**Supercute peritonitis** from perforation of a simple ulcer of the stomach will be discussed in the next article. We shall see that, while its symptoms are severe and sudden, they differ much from those of early appendicitis. In its suddenness and its immediate gravity perforation of a gastric ulcer free from adhesions is only comparable with that of simple ulcer of the duodenum.

**Diagnosis.**—Violent pains at the xiphoid and spinal points; vomiting, with hyperchlorhydria, following more or less quickly after taking food; hæmatemesis, which is more or less copious and repeated, with or without melæna, and the absence of an epigastric tumour, are all in favour of simple ulcer. These symptoms are, however, not always clearly marked, and some of them may be wanting, so that in certain cases the diagnosis is very difficult.

As pain in the stomach may be early, frequent, and intense, thus form-



ing the chief symptom in simple ulcer of the stomach, we must review the various diseases in which attacks of pain may simulate ulcer.

The pains of **hepatic colic**, from their position and violence, may simulate those of gastric ulcer. In hepatic colic the pain is independent of digestion, but in ulcer the pain is at once excited by food, and becomes most intense two or three hours after a meal, when the quantity of free hydrochloric acid attains its maximum. Hepatic colic provokes vomiting of bile, while ulcer causes vomiting of food, mucus, or dark blood. Hepatic colic is a passing episode, often accompanied by jaundice and urobilinuria. Nothing of a similar nature is seen in gastric ulcer.

The **gastric crises of tabes** may, from their situation and intensity, simulate the pains of ulcer; the likeness is the greater as the crises in tabes may be accompanied by hyperchlorhydria and bloody vomit. In the tabetic patient, even in the pre-ataxic stage, some stigmata of tabes will always be found if carefully looked for—lightning pains in the legs, retardation of sensibility, abolition of the patellar reflexes; eye troubles, ptosis, strabismus; urinary troubles, Romberg's sign. Finally, in the intervals between the gastric crises the stomach performs its function well, without dyspepsia and hyperchlorhydria.

**Gastric uræmia**, with its gastralgia, intolerance of food, repeated and at times blood-stained vomit, may simulate simple ulcer of the stomach. The vomit in uræmia is very rarely acid, and frequently contains urea and carbonate of ammonia, while the patient usually has albuminuria, and always shows signs of Brightism or of confirmed Bright's disease.

**Hysteria** simulates gastric ulcer. Acute gastralgia, intolerance, vomiting, hyperchlorhydria, and hæmatemesis may be present, so that it is only by careful search for the stigmata of hysteria, such as pharyngeal anæsthesia, hemianæsthesia, ovarian pain, contraction of the field of vision, hysterogeneous zones, etc., that it will be possible to make a diagnosis.

The diagnosis of ulcer from **hyperchlorhydria** is very difficult, because in both cases the pains caused by digestion reach their maximum two or three hours after a meal; but the pyrosis and acid regurgitation in hyperchlorhydria are not usually accompanied by vomiting of food. Hypersecretion and ulcer have as common symptoms paroxysmal and continuous pains, but nocturnal vomiting of several ounces of liquid and the presence of hydrochloric acid in the stomach, apart from digestion, are special to hypersecretion. The diagnosis of ulcer and cancer will be discussed under Cancer of the Stomach.

The diagnosis of the site of the ulcer is of importance. The anterior ulcer, which most often leads to perforation, causes pain towards the left hypochondrium. It is made worse by the prone position and relieved by the supine posture. The posterior ulcer, which causes severe hæmorrhage

(splenic artery), gives rise to great pain, which is especially acute in the spinal region. The pain is relieved by the prone position. The pyloric ulcer has its centre of pain to the right of the median line.

The **prognosis** is always grave, even when the disease has a benign appearance. It is grave because the ulcer exposes the patient to attacks of hæmorrhage, perforation, peritonitis, or stenosis of the stomach, and also because the ulcer is sometimes rebellious to treatment and subject to return. Finally, the prognosis is grave because **cancer may graft itself on ulcer**.

**Treatment.**—The patient must take milk, fresh or boiled, hot or cold, according to taste, at equal intervals—every two hours, for instance—so as to make up 3 or 4 pints in the twenty-four hours.

I had in the Hôtel-Dieu a young woman suffering from ulcer of the stomach, with hæmatemesis, acute pain, uncontrollable vomiting, and loss of flesh. The intolerance of the stomach was such that cow's milk, even in very small doses, was immediately vomited. I therefore prescribed **ass's milk**. In two months she gained 25 pounds in weight, and was quite restored to health.

The milk diet, in order to be efficacious, must be as rigid as possible. At first, if the milk does not agree well, each glass is mixed with a tablespoonful of lime-water, to which small doses of morphia and cocaine are added. Lime-water, with the addition of **very small doses of morphia and cocaine**, is, in my opinion, an excellent remedy in all diseases where the abnormal excitability of the stomach provokes pain and vomiting. Injections of morphia should be reserved for cases in which the gastralgia is acute and obstinate. Nitrate of silver, in doses of  $\frac{1}{2}$  grain or more daily, with opium and subnitrate of bismuth, or alkalis in large doses (Debove), is of service. Hæmorrhage must be treated with perchloride of iron, ergotin, and iced drinks. The continuous application of an ice-bag to the epigastric region is excellent, both for hæmorrhage and also for the pain and vomiting. In severe hæmatemesis I advise large subcutaneous injections of artificial serum, repeated several times a day.

If the ulcer be situated on the anterior surface of the stomach, I would advise absolute rest in bed, in the dorsal position. The convalescence must be carefully watched, and we must not forget that the disease may recur.

In some cases medical treatment fails. If the hæmorrhage, pain, and vomiting are persistent (in the absence of syphilis), recourse must be had to surgical treatment.

Marion sums up the question as follows: The reports of intervention for hæmorrhage are still too few to give exact information as to surgical treatment. Gannat, who operated upon a patient for hæmatemesis, was alarmed by the perigastric adhesions, and closed the abdomen. The post-



mortem examination revealed the presence of a simple ulcer, which involved the pancreatico-duodenal artery.

Mikulicz operated four times for hæmatemesis. In the first case he cauterized the ulcer, but the patient died of collapse fifty hours after operation. In the second case he attempted to suture the bleeding area, but the patient died from collapse the same evening. In the case of a woman he curetted the ulcer and perforated the wall of the stomach, which he sutured. The patient recovered. Finally, in the fourth case he sutured the stomach at the bleeding spot, and in the evening death supervened from collapse. Küster operated on a young girl for hæmatemesis. He cauterized the ulcer, and completed the operation by gastro-enterostomy, to obviate any ulterior contraction of the pylorus due to cicatrix of the ulcer. The patient recovered. Roux performed partial gastrectomy, followed by suture of the three layers, in a patient with hæmatemesis following simple ulcer which had eroded the gastric artery. The double ligature of this artery, with the excision of the ulcerated portion, saved the patient.

In cases other than those of hæmorrhage numerous and important successes have been recorded (Doyen). If an ulcer reveals itself by frequent and repeated attacks of hæmatemesis, incessant vomiting, symptoms of spasm or pyloric constriction, or signs of cachexia, as these cases resist medical treatment, gastro-enterostomy should be done. Each operator modifies the procedure according to his liking, and according to the adhesions, the dimensions, the extent, the depth, and the situation of the ulcer.

## XI. PERFORATION OF SIMPLE ULCER OF THE STOMACH— SUPERACUTE PERITONITIS.

In the preceding section I described the consequences of perforation of the ulcer when adhesions limit the lesion. I shall now consider perforation followed by **superacute peritonitis**. As a type, I will quote the following case, taken from one of my clinical lectures : \*

One morning, at the Hôtel-Dieu, I saw a boy whose **pale** face expressed the most intense suffering. He walked with difficulty, his back bent and his stomach retracted, while his hands were held forward, as if to protect the abdomen and avoid contact with anything which might increase the pain. He was suffering, so he told us, from terrible pains in the belly ; they came on suddenly in the morning at six o'clock, " as if the intestines were being torn." These awful pains were followed neither by vomiting nor by hiccough. The belly was flat, the pulse 68, and the temperature 98.5° F. He had only one symptom—pain. I formed a bad impression of the case, the features being drawn and the nose pinched. It was necessary to decide the question of immediate **surgical intervention**. Was the pain due to appendicitis or peritonitis from perforation

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\* " Perforation de l'Ulcère Simple de l'Estomac : Péritonite Suraiguë " (*Clinique Médicale de l'Hôtel-Dieu*, 1898, 5<sup>me</sup> leçon).

of an ulcer of the stomach or duodenum? Had we to do with internal strangulation or acute entero-colitis? Was it a case of renal, hepatic, or lead colic? In such cases an exact diagnosis can only be made by careful consideration of the signs and symptoms.

Pain was here the chief symptom, and therefore demanded prompt investigation. The pain commenced at six o'clock in the morning, being sudden and acute from the first. It was unexpected, the patient having gone to bed the previous evening feeling quite well. The suddenness and intensity of the pain recalled in every point what I have named "**the peritoneal dagger-thrust**," or the special symptom of perforation of a gastric or duodenal ulcer. The sudden onset eliminated the idea of appendicitis. We no longer admit the erroneous idea that appendicitis can commence suddenly with terrible pain due to perforation. At the risk of repetition, I must again state that in appendicitis (if we look closely) events do not run this course. No matter how painful the onset of appendicitis may be, the pains **increase gradually**. Question your patients carefully, and try to obtain from them precise answers, and you will be convinced that it is only after one or several hours that the pain reaches its maximum, and even then it is very rarely excessive. In peritonitis from perforation the pain is as sudden as it is violent, and the comparison with the peritoneal dagger-thrust, which is here correct, is not applicable to appendicitis. In short, the onset of perforation bears little or no resemblance to that of appendicitis.

Furthermore, the **localization** of the pain is important in diagnosis, and, even supposing that the entire belly be painful, you can still, by methodical examination, locate the region in which the pain began and the spot where it became most acute. In appendicitis (even if the pains extend elsewhere) this spot occupies the centre of a line drawn from the umbilicus to the right antero-superior spine of the ilium. It is at McBurney's point that the pain of appendicitis commences. On palpation, you will find the greatest tenderness there, and the defensive contraction of the underlying muscle. Finally, gentle stroking of the skin in that region will reveal hyperæsthesia, shown by reflex movements in the belly wall and the neighbouring parts.

In my patient, examination of the belly showed the principal point of pain was not in the appendicular region. The whole belly was painful, but the spontaneous pain of the onset, the tenderness on palpation and percussion, the defensive contraction, and the hyperæsthesia had their maximum of intensity, not in the appendicular region, but in the subhepato-gastric region above and a little to the right of the umbilicus.

The lesion, therefore, was in this region, where only three organs are liable to perforation—the gall-bladder, duodenum, and stomach. There was no question here of the gall-bladder, because he had never had any symptoms of gall-stones. I therefore made the following diagnosis: perforation of a latent ulcer of the pylorus or duodenum, the signs and symptoms of perforation being identical in the two cases.

Although the peritonitis had only existed four hours, his complexion was already becoming ashen. He had the peritoneal facies, which is an important index in diagnosis. I diagnosed superacute peritonitis, due to perforation, though he was free from fever, acceleration of the pulse, vomiting, hiccough, and ballooning of the belly, which are the classical signs of acute peritonitis. I had also to admit a latent ulcer, as the patient had, prior to the perforation, never shown any symptom of ulcer. There was not a moment, then, to be lost, for perforation with general peritonitis is always fatal, and the only chance of recovery lies in immediate operation. The peritoneal cavity was opened by a median incision. Peritonitis was at once evident; the coils of bowel were injected, and brownish liquid came out from the wound. A sponge introduced into the upper portion of the incision towards the stomach was, on withdrawal, soaked with puriform fluid. No trace of food. The sponge had therefore been in the neighbourhood of the lesion. The lower edge of the liver was pulled upwards and outwards, so as to expose the pylorus. About an inch from the pylorus, on the anterior surface of the stomach, a circular punched-out opening of the size of a lentil was found.



His condition was satisfactory until the next day, when two attacks of vomiting occurred, and he became very collapsed. He became worse on the third day: the pulse was 116, the temperature rose to 104° F., the vomit was porraceous, and the eyes were sunken. He became rapidly worse, and died. At the post-mortem examination the sutures employed to close the perforation were in exact apposition. Death was caused by the general suppurative peritonitis.

**Analysis of the Symptoms.**—On admission, the peritonitis, which was but four hours old, presented certain peculiarities. To begin with, the belly was flat and fairly hard, and tympanites was not present. Tympanites is generally given as one of the symptoms of peritonitis caused by perforation, but it may be absent. It is absent in cases of perforation when the peritonitis from the first produces such reflex contraction of the abdominal muscles that the belly is flat, hard, and retracted, instead of being tympanitic. The abdomen was flat and hard in a young girl whom I saw with Tisné, although two symmetrical ulcers of the stomach had just perforated. The abdomen was tight, “flat, and as hard as a board,” in a case reported by Utudjian. We shall see later that the same remark applies to super-acute peritonitis following perforation of a duodenal ulcer.

We are too apt to think that peritonitis caused by perforation is followed by meteorism from intestinal paralysis or the passage of gas through the perforation. This is not always the case, and it would be quite wrong to exclude peritonitis because the abdomen is flat and hard. These two conditions may, however, follow one another. A patient, for instance, whose abdomen is flat and hard shortly after the perforation will very likely show tympanites on the next day. The tympanites may be so marked in the symptom-complex that perforation of the stomach is wrongly taken for obstruction of the gut. I am acquainted with several cases in which the surgeon operated for strangulation and found peritonitis. It seems to me that this error might have been avoided, for pain is always a marked symptom, and should lead to a correct diagnosis. I know that, if the abdomen is tympanitic in consequence of a perforation of the stomach, sensibility is sometimes dulled, and that all the desired information cannot be obtained from the patient. If, however, the commencement of the complication be **stated precisely**, it will be seen that the perforation was ushered in by sudden severe pain in a person who had passed a free motion the night before. Intestinal occlusion does not begin in this manner. Its symptoms, including meteorism, come on more slowly, and “the peritoneal dagger-thrust,” which points to gastric or duodenal perforation, never marks the commencement of intestinal occlusion, whatever be its cause.

Our patient had **neither hiccough nor vomiting**, two signs which occupy an important part in the general description of acute peritonitis. These two signs are often wanting in peritonitis from perforation of a gastric or duodenal ulcer, especially in the period following the perforation.

A fantastic theory has been put forward that the patient does not vomit because the contents of the stomach pass into the peritoneum through the perforation. Another unexpected fact for those not well acquainted with the question is that the pulse was normal and the temperature only 99° F. It seems surprising that a patient suffering from superacute peritonitis of four hours' duration should have no increase in the pulse-rate and no rise or fall of temperature. Nothing of the kind occurred in the case before us, and similar facts have been noticed by other writers. My patient, therefore, had not tympanites, fever, vomiting, or hiccough, in spite of superacute peritonitis from perforation of the stomach. No previous gastric symptoms were present as a guide, and yet it was possible to make an accurate diagnosis. In speaking thus, I only wish to prove the importance of a well-conducted semeiology. One sign carefully examined—viz., pain—was sufficient to lead to the diagnosis.

We see, too, that the old description of acute peritonitis must be remodelled. It is impossible to give a dogmatic description of peritonitis, for the same symptoms are far from being applicable to all cases. Thus peritonitis in typhoid fever does not resemble that due to perforation of an ulcer of the stomach or the duodenum; the latter form does not resemble appendicular peritonitis, which, again, is quite different to primary pneumococcal peritonitis, etc. It may be said that the researches of recent years have quite changed the description of the medico-chirurgical diseases of the abdomen.

**Treatment** here is purely surgical. In superacute peritonitis following on ulcer of the stomach, operation is imperative. We must, however, decide and act promptly, for the chances of success are the greater the sooner the operation is performed.

In a communication on perforations of ulcer of the stomach, Le Dentu has entered into minute details, and collected the following statistics concerning the results of operation:

Michaux's statistics for the year 1894: In 25 cases, 5 cures and 20 deaths.

Chapt's statistics for the year 1895: In 27 cases, 6 cures and 21 deaths.

Chapt and Maclaure's combined statistics: In 45 cases, 15 cures and 30 deaths.

Houzé's statistics for the year 1895: In 67 cases, 20 cures and 47 deaths.

Parisier's statistics: In 99 cases, 33 cures and 66 deaths.

These figures show recovery in a third of the cases, a good result in a disease which is fatal in the absence of surgical intervention. Furthermore, it may be laid down as an axiom that the earlier the operation, the more certain the cure. Out of nineteen cures reported by Comte, eleven were due to the fact that the operation was performed within ten hours of the perforation. Out of Parisier's thirty-three cures, twenty-



three were due to the fact that operation was performed ten to fifteen hours after the perforation. On the other hand, the chance of recovery decreases in proportion as the operation is performed at longer intervals after the perforation.

## XII. PERFORATING ULCERS OF THE STOMACH AND DUODENUM CONSECUTIVE TO APPENDICITIS.

In Section IX. I described the erosions and the exulcerations of the stomach consecutive to appendicitis. I shall now prove that the appendicular infection may also give rise to deep ulcers of the stomach and the duodenum, which run a rapid course and end in perforation. This question is discussed in a clinical lecture I have recently delivered upon perforating ulcers consecutive to appendicitis. To quote examples :

**Clinical Cases.**—An enfeebled man, about sixty years of age, was admitted on January 16, 1905. His history was so vague that accurate diagnosis was not possible. Abdomen somewhat swollen ; diffuse dullness in the right iliac fossa, and acute tenderness on deep pressure in the right hypochondrium.

He died next day, and his wife gave the following information : for some time he had had pain in the belly ; acute pain then appeared suddenly in the region of the stomach, and he decided to come to hospital.

Post-mortem results : peritoneal adhesions in the right flank, circumscribing a retrocæcal abscess of appendicular origin.

On opening the stomach, numerous capillary hæmorrhages were found, chiefly at the cardiac end. The duodenum showed an unexpected lesion : on the posterior wall of the first portion an ulcer had perforated the gut ; it was the size of a shilling, and evidently of recent date, because the edges were not indurated, as in a classical ulcer. It was about  $\frac{1}{2}$  inch from the pylorus and rested against the posterior abdominal wall, which formed the floor and acted as a tampon, preventing general peritonitis.

In short, he had had subacute appendicitis, with retrocæcal abscess and adhesions ; the infection had led to hæmorrhagic erosions in the stomach, and to an acute perforating ulcer in the duodenum.

*Lediard and Sedgwick's Case.*—A man aged fifty-six was taken ill with pains in the right iliac fossa. One year later they came back over the appendix. In April, 1904, a fresh attack. Diagnosis : appendicitis. Operation on May 8. On opening the belly, the appendix was found to be large and covered with old exudate. It was removed and the wound was closed. During the next few days there was no fever. Slight jaundice of the skin and conjunctivæ. He then passed bloody stools. On dressing the wound, the stitches were found to be loose ; foul-smelling pus flowed out of the wound. A week after the operation more and more blood appeared in the stools, the pulse became intermittent, and the general pallor indicated abundant loss of blood. What was the cause and the seat of the bleeding ? Hæmatemesis was also present, and death occurred on June 29. The autopsy was made on the same day. On opening the abdomen, no peritonitis ; the stomach was healthy. On the second part of the duodenum, near the ampulla of Vater, there was an oval, crateriform, perforating ulcer, that was 1 inch in length and  $\frac{1}{2}$  inch in breadth ; its long axis was parallel to that of the bowel ; the edges of the ulcer were smooth, and the floor was obliterated by the pancreas, which formed a tampon, preventing acute peritonitis, which would otherwise have followed the perforation.

The pancreatic tissue showed early ulceration ; two open vessels (probably branches of the inferior pancreatico-duodenal artery) had given rise to the fatal intestinal hæmorrhage. A section through the edge of the ulcer and the adherent pancreatic tissue showed complete destruction of the bowel wall, and also of the adjacent pancreatic tissue.

*Milward's Case.*—A girl aged seven years had been ill for two weeks. On admission the right iliac fossa was painful and the abdomen distended. Operation: On opening the belly in the appendicular region, 2 ounces of pus flowed out from a cavity external to the cæcum and shut off from the general peritoneum by adhesions. The appendix was not found ; possibly it was destroyed or buried under the adhesions. After the operation, rapid pulse, high temperature, and diarrhœa. On the eighth day bulging in the epigastric region, which was resonant. The bulging increased slowly, and appeared to vary with the condition of the intestines, being more marked when the patient was constipated.

The tumour became so large on the twenty-fifth day that an operation was decided upon. The epigastrium was incised, and the operator found near the stomach a cavity containing gas, pus, and gastric contents. It was shut off from the peritoneal cavity by adhesions, which fixed the stomach to the abdominal wall. The anterior surface of the stomach showed a perforating ulcer of the size of a threepenny-piece. The perforation and the abdominal wound were sutured, but feeding became more difficult, the situation grew worse, and the patient died five days after the operation.

The autopsy showed that the iliac and epigastric lesions were independent. The course of the disease had been as follows: Appendicitis of a fortnight's duration, with encysted pericæcal abscess. Acute ulceration of the stomach, and formation of adhesions between the stomach and abdominal wall. The perigastritis had prevented the onset of acute peritonitis. The gas, which made its way through the perforation, caused bulging of the epigastric region.

*Warren Low's Case.*—On April 14, 1904, a girl of sixteen was admitted to St. Mary's Hospital. She appeared to be suffering from general peritonitis, and was very weak. Her history was as follows: On the evening of April 9 she felt pain in the right iliac fossa. Next day she remained in bed. On April 12 pain in the epigastric region. Next evening at five o'clock she was taken ill with vomiting and very acute pain in the region of the appendix. On admission she was collapsed, with pale and drawn face. Abdomen distended and sensitive; pain on palpation over the lower part of the belly, especially on the right side. The collapse improved with the aid of warmth and stimulants. Next morning an incision was made over the appendix; some odourless pus came out. The appendix was removed; it contained two calculi, and extravasated blood was present in its walls. The case seemed to belong to the relapsing type. A median incision above the umbilicus revealed adhesive peritonitis above the transverse colon. A perforation as large as a crown was exposed; it involved the front of the lesser curvature of the stomach, near the cardiac end. The ulcer was sutured; both wounds in the abdominal wall were drained. Recovery.

*Cheyne and Haydock Wilbe's Case.*—A boy aged thirteen years complained of pain in the stomach. Next day the pain was more severe and extensive. Dr. Wilbe, who saw the child in the afternoon, found him much collapsed. He found muscular rigidity and general hyperæsthesia; the pain was most marked in the right iliac fossa.

Mr. Watson Cheyne saw him the same day. The abdomen was hard, distended, and tympanitic; the hyperæsthesia was general, and the pain most marked to the right and above. Perforation of the appendix was diagnosed, and an operation performed.

An incision was made over the appendix. As soon as the peritoneum was opened, an escape of gas indicated perforation of the alimentary canal. Serous fluid was present in the peritoneal cavity. The cæcum and the appendix were examined. The latter was long and inflamed, and contained a calculus. The operator felt sure that



some other lesion was also present. As no fæces were free in the peritoneal cavity, and there was no intestinal perforation, a fresh incision was made in the epigastric region. Turbid fluid and bubbles of gas were seen over the front of the stomach near the cardiac end. A rounded perforation was shown up by reflected light about 1 inch from the cardiac end of the stomach; fluid and gas were escaping from it. The perforation was about as big as a lead-pencil; its edges were not thickened. No other lesions in the vicinity. The edges of the ulcer were sutured. Rapid improvement followed, and the patient was discharged cured.

*Pfihl's Case.*—A boy was taken ill with appendicitis. At the operation a few days later a pericæcal abscess was opened, but the appendix was not found. He died from profuse hæmatemesis twenty-three days after the operation. The autopsy showed a perforating ulcer on the anterior surface of the stomach four fingers' breadth from the pylorus; the perforation was circular, punched out, and as large as a sixpence.

**Discussion.**—In each of these six cases a perforating ulcer of the stomach or duodenum was associated with appendicitis. Careful study shows the order in which the lesions appeared. Appendicitis preceded by a more or less lengthy period the symptoms of ulcer.

In these cases there is no suggestion that the appendicitis was preceded by the classical signs of ulcer—epigastric and spinal pain, vomiting, hæmatemesis, and melæna. On the contrary, the scene opened with appendicitis.

We find in each case that acute, subacute, or relapsing appendicitis was in progress when the gastric or duodenal complication appeared suddenly, and ended in perforation. When we consider the chronological order of the different lesions, we may justly admit that the ulcer was secondary to the appendicitis.

Does this chronological order justify the view that the perforating ulcer is dependent upon the appendicitis? We know that the duodenum, and especially the stomach, may in the course of appendicitis be attacked by erosions, exulcerations, and perforating ulcer.

We have seen in Section VIII. that erosions and exulcerations occur in the stomach. In the following case, quoted by Dupont, deep ulceration, with acute necrosis of the gastric mucous membrane, was present.

In November, 1904, a soldier was taken ill with toxic appendicitis. The prognosis rapidly became grave: the heart was irregular, the breathing was jerky, the nose pinched, and the eyes hollow. The patient did not consent to operation till the fourth day of the disease. Naturally no improvement resulted, and he vomited dark blood on two occasions. Vomito negro is nearly always of evil omen. During the next few days restlessness and delirium set in, and he died in coma.

Post mortem: On opening the stomach, ecchymoses of variable size were found in the greater curvature and the pyloric region; at the pylorus, two ulcerations of the size of a lentil; in their centre the mucous membrane was raised up around a crater as big as a pin's head. Nattan-Larrier's examination revealed the course of the lesion. He found coagulation necrosis of the cells, converting the tissue into a hyaline mass and affecting the glands in an irregular way, so that normal and degenerated glands were found side by side. The necrosis was set up by the appendicular toxins.

Experimental research also gives us information regarding the pathogenesis of gastric and duodenal ulcers resulting from appendicitis. Talma's experiments on simple ulcer of the stomach show the action of intestinal strangulation in causing gastric erosions and ulcerations. He ligatured a loop of bowel, and found among the lesions present hæmorrhagic erosions and ulcerations of the stomach.

Roger has shown that experimental infections of the cæcum produce in the stomach ecchymoses like those of purpura. "It is difficult to explain," says he, "how the cæcal affection can react upon the stomach. The fact has none the less a certain importance, especially if we consider it in relation with Dieulafoy's observations, which show clearly the existence of gastric hæmorrhages due to appendicitis. Experimental pathology, therefore, confirms clinical observation."

Nattan-Larrier and Lœvy have made the following experiments: In two dogs an inch of small intestine was shut off by two ligatures. The animals died in five days from acute peritonitis. A small quantity of the fluid from the ligatured loop and from the peritoneum was injected into a ligatured loop in two other dogs. They died in three days from acute peritonitis. A small quantity of fluid (taken as before) was then injected into a fifth dog, which died in forty-eight hours without any trace of peritonitis. Its stomach, however, contained two spoonfuls of blackish fluid (gastrorrhagia); two clean-cut exulcerations were found, involving the mucous membrane and the sub-mucous tissue. One of the exulcerations was on the greater curvature, the other on the first part of the duodenum.

The urine contained bile pigment, albumin, and fatty casts. The cells in Henle's loops took the stain badly, and were studded with fine fatty granules, the nuclei being indistinct. In the liver the cells took the stain better, although they showed fatty degeneration. Three inoculations in series, therefore, modified the results. In the first two series ligature of a segment of gut simply gave rise to septic troubles, but in the third inoculation the animal showed toxæmia, with gastro-duodenal, hepatic, and renal lesions.

These lesions, produced experimentally, confirm the views I expressed at the Académie de Médecine regarding the toxicity of appendicitis. Prior to this occasion, appendicitis was considered to be simply an infective focus. I have shown, however, that poison is manufactured in this focus, and thence invades the economy. We have, therefore, a new variety of appendicitis, which gives rise to toxæmia; and I have called this condition "appendicæmia." We have also seen that the appendicular poison has a marked preference for the stomach, liver, and kidney.

In the kidney the toxic nephritis excites albuminuria, hæmaturia, oliguria, anuria, and at times symptoms of uræmia. In the liver the lesions cause jaundice, urobilinuria, and in some cases symptoms of icterus gravis. In the stomach the erosions and exulcerations give rise to hæmorrhage, which may be fulminant.

In some cases of appendicitis the ulcers in the stomach and duodenum



run a rapid course, ending in perforation; in other cases the ulcers are accompanied by erosions and ecchymoses in the neighbourhood of the ulcers. In my patient the stomach was the seat of capillary hæmorrhages, while a perforating ulcer was present in the duodenum. As a general rule, it seems that the stomach and duodenum are organs prone to ulceration and perforation. This fact is shown by the pneumococcal erosions, the exulcerations, and the simple ulcer. The appendicular toxines play a large part in this ulcerative process, which may end in erosion, exulceration, or perforation.

While it may be hard to distinguish between appendicitis and perforating ulcer of the stomach or duodenum, the distinction is much more difficult if the two lesions succeed one another and exist together. The diffuse pain from two separate foci, and the possible existence of acute peritonitis, so complicate the situation that a diagnosis may be almost impossible. The surgeon must discover both foci, and act accordingly. In the cases quoted two patients were saved by a double operation, which involved the appendix and the gastric ulcer. Similar operations are described later, under the Association of Appendicitis and Cholecystitis.

### XIII. TRANSFORMATION OF SIMPLE ULCER OF THE STOMACH INTO CANCER.

The transformation of ulcer of the stomach into cancer is not uncommon. Cruveilhier, in his excellent work on simple ulcer of the stomach, deserves great credit for elucidating a question which was previously obscure. Ulcer and cancer were confounded, and the simple ulcer, which is rightly called "Cruveilhier's disease," had not become a morbid entity. After Cruveilhier, people perhaps went too far; the division between ulcer and cancer was too absolute, and the fact, that Cruveilhier himself had noted the co-existence of the two lesions in the stomach, was forgotten.

This coexistence of carcinoma and ulcer, says Rokitansky, leads to the supposition that the former supervenes on the latter. Dittrich, of Prague, states positively the termination of ulcer in cancer. In 1848 he published the statistics of 160 cases, in which ulcer of the stomach was eight times associated with cancer. Sometimes both ulcer and cancer were present in the same stomach, but at other times the cancer was grafted on a healed or an active ulcer. Lebert considers that in 100 cases of cancer of the stomach, nine are preceded by ulcer. In 1882 Hanser studied histologically the lesions of the mucous membrane in the vicinity of the ulcer. He noted the development of adenomatous tissue, and the tendency for cylindrical cells to take the place of the glandular epithelium. This was the first stage, which ended later in the invasion of the muscularis mucosæ and tunica muscularis. Rosenheim admits that ulcer is complicated with cancer in the proportion

of 6 per cent., and Sonicksen found that in 156 cases of cancer of the stomach, examined in the Kiel Institute, ulcer had preceded cancer in the proportion of 14 per cent. Pignal has collected several cases of the transformation of ulcer into cancer, observed by Bouveret and Lépine. Mathieu has published three cases.

I have devoted a clinical lecture\* to this question. The following case will serve as an example :

A man came under my care for cancer of the stomach. Cachexia, marked loss of flesh, and straw-coloured complexion justified the diagnosis of cancer at first sight. I questioned him, and learnt that the disease had made its appearance fifteen months previously, with pains and vomiting, which had since been the chief symptoms. During this period he had had one attack of hæmatemesis and several of malæna. On examining the epigastric region, I was surprised to find that the slightest pressure was so painful as to prevent a complete examination. He had not only deep-seated pain, but also acute cutaneous hyperæsthesia. He then described his pains. From the commencement of the disease they supervened after meals, persisted during digestion, and did not cease when the stomach was empty. They were sometimes tearing in character, and were seated principally in the pit of the stomach, but they also radiated towards the spine. The fits of vomiting were frequent, and the vomit was so acid as to cause a lasting taste of vinegar in the mouth. It was evidently a case of hyperchlorhydria. The stomach, which did not appear to be dilated, was so intolerant that water and milk were at once vomited.

The severity of these symptoms led me to doubt the diagnosis of cancer. Cancer of the stomach is not, as a rule, accompanied by such acute pains and uncontrollable vomiting. Although pain, dyspepsia, and vomiting occupy a most important place in cancer of the stomach, I have not seen a case in which they were so severe ; the symptoms belong especially to simple ulcer.

I had the vomit examined by Du Pasquier, and instead of finding hyperchlorhydria, which must have been present before when the vomit was as acid as vinegar, he found marked hypochlorhydria. The hydrochloric acid was present in the proportion of 0.18 instead of 1.74 per 1,000. The results obtained after a test meal were practically the same.

The diagnosis, therefore, was difficult. The violence of the pain and the frequent vomiting, with the intolerance of the stomach and the previous acidity, were all in favour of simple ulcer. On the other hand, the actual hypochlorhydria and progressive emaciation of the patient, his cachectic look, and the presence of inguinal glands, were in favour of cancer. Exploration of the epigastric region did not settle the question, for there was neither tumour nor induration ; moreover, the presence of induration is of little value, for the tumour may escape notice, and also an ulcer, if surrounded by indurated tissues, may resemble cancer.

My previous experience, combined with the course of the symptoms, led me to think that the patient had originally had an ulcer of the stomach on which cancer had been grafted.

As it was necessary to relieve, if not to cure, him, I ordered the following mixture, which I recommend in all cases of painful dyspepsia :

|                       |    |    |    |    |       |
|-----------------------|----|----|----|----|-------|
| Lime-water            | .. | .. | .. | .. | ̄iii. |
| Cocaine hydrochlorate | .. | .. | .. | .. | gr. ½ |
| Morphia hydrochlorate | .. | .. | .. | .. | gr. ⅓ |

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\* "Transformation de l'Ulcère Stomacal en Cancer" (*Clinique Médicale de l'Hôtel-Dieu*, 1897, 13<sup>me</sup> leçon).



A tablespoonful of milk with a teaspoonful of the mixture was administered every hour. An ice-bag was placed on the pit of the stomach. Next day the pain in the stomach had diminished. The milk and lime-water were then increased. In about a fortnight the pains had almost disappeared and he took daily 3 pints milk without vomiting. His general condition improved, sleep returned, the loss of flesh was less marked, the weight even increased a little, so that the patient became hopeful, and I was asked if the diagnosis of cancer was not wrong, and if it was not necessary to substitute the diagnosis of simple ulcer.

Although true improvement is rare in cancer of the stomach, it is not uncommon to see a period of arrest in the course of the disease. Early lavage may cause an improvement, which does not suffice to exclude the primary idea of cancer. I did not, therefore, modify the diagnosis. The course of events justified my prophecy. After a month of relative improvement, the pains again became so severe as to demand frequent injections of morphia. He refused food, and died from cachexia.

This mode of death occurs chiefly in cancer. Ulcer of the stomach kills by peritonitis, hæmorrhage, or by secondary peritoneal abscess, while cancer kills slowly and progressively by cachexia, and probably by its toxine.

The post-mortem examination showed that it was a case of cancer grafted on ulcer. The lesion comprised a huge ulcer, perpendicular to the long axis of the stomach.

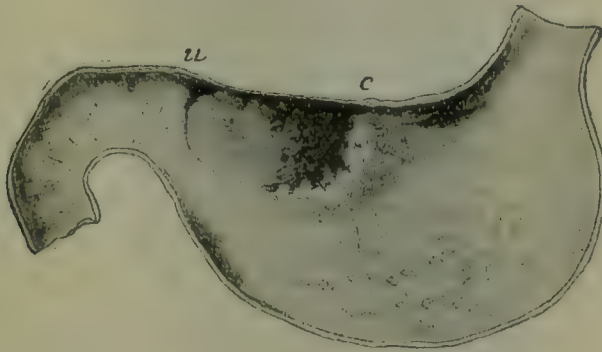


FIG. 34.—ULCER: FLOOR FORMED BY THE LIVER.

*u*, Indurated flat edge; *c*, raised edge showing malignant change.

The ulcer measured 2 and 4 inches in its two diameters. It occupied the lesser curvature and encroached on the anterior and posterior surfaces of the stomach. To the left it extended to within 2 inches of the œsophagus, and to the right it was bounded by the pylorus. It was a metatypical epithelioma.

In a general way, ulcer and cancer of the stomach may coexist in different combinations. The most frequent combination is that seen in my patient—the cancerous degeneration occurring whilst the ulceration is active. In other cases the cicatrix of a healed ulcer becomes the starting-point of the cancerous change; and in other cases, again, we find cancer and ulcer clearly distinct, which leads to the belief that the cancer has arisen in a former ulcer. The varieties met with include cylindrical epithelioma, metatypical epithelioma, encephaloidal carcinoma, scirrhus and colloid cancer. Of the different varieties, the metatypical epithelioma seems to be the most frequent.

The transformation of ulcer of the stomach into cancer is comparable

with the change seen elsewhere in cicatrices. Chaintre has collected a number of cases—Poncet : epithelioma in the scar of an amputation stump ; Mollière and Laroyenne : epithelioma in the cicatrix of an old cautery wound in the deltoid region ; Marcin : cancerous change in the scar of a burn on the lower part of the thigh ; Poncet : epithelioma in a urinary fistula ; Jansion : epithelioma grafted on old ulcers of the leg, etc.

Epithelioma may appear in a patch of buccal, labial, or lingual leukokeratosis, or in the cicatrix of patches of leukokeratosis which have been operated upon. If, in the present state of our knowledge, and in spite of its frequency, the epitheliomatous change is not considered as an inevitable



FIG. 35.—SECTION THROUGH (FIG. 34).

*v*, Malignant vegetation ; *cs*, culs-de-sac of the glands ; *m*, muscularis mucosæ ; *s*, submucosa ; *cm*, muscular layers ; *a*, discs of cancer cells ; *f*, floor of ulcer ; *z*, zone of transition between growth and healthy parts ; *e*, healthy mucosa ; *p*, thickened peritoneum. The lighter half of the figure is healthy tissue.

phase of the disease, it must be held that the change is determined by an inherent predisposition in the leukokeratosis itself (Le Dentu).

**Analysis of the Symptoms.**—It has been shown that ulcer of the stomach undergoes cancerous degeneration in relatively numerous cases, especially when heredity is concerned. The following problem has, then, to be solved : How are we to tell whether ulcer of the stomach, which is often curable, is undergoing malignant change, which is always fatal ?

Several forms may present themselves. In one case the symptoms of ulcer are so pronounced that diagnosis is impossible, and the presence of the cancer is unnoticed. A patient with the classical signs of ulcer is taken ill with perforation of the stomach, acute peritonitis, and fatal hæmorrhage.



The post-mortem examination reveals an ulcer, and also a malignant growth, which has not yet had time to leave its mark on the affection.

On the other hand, a patient shows signs of malignant cachexia—loss of flesh, anorexia, pallor of the skin, hypochlorhydria, and enlarged glands in the groin or in the clavicular region, pointing to cancer of the stomach. What signs will show that the cancer is grafted on an ulcer—or, rather, what signs will help us to eliminate the hypothesis of cancer, and retain

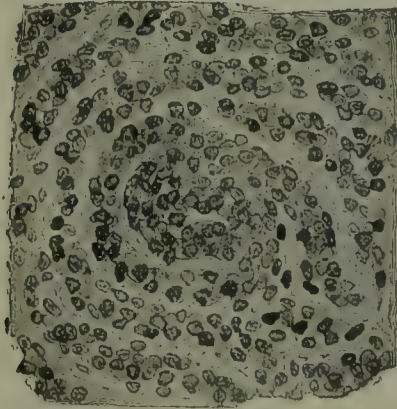


FIG. 36.—SECTION THROUGH A (FIG. 35); COIL OF CANCER CELLS.

that of ulcer? Cancer and ulcer of the stomach do not run the same course. It is not sufficient to make a thorough examination of the patient; we must also review his past, and study the onset and import of each symptom. The general rule is: Violent pain in the stomach and profuse hæmatemesis are found, not in cancer, but in ulcer. An individual who has the appearance of cancerous cachexia, and suffers, or has at some period of his illness suffered, from acute gastric and intrascapular pains, which are increased by the ingestion and digestion of food, has perhaps a cancer

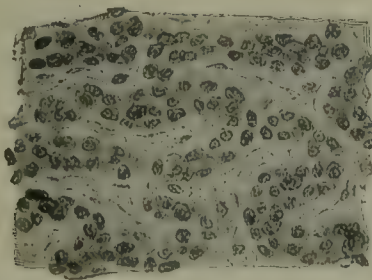


FIG. 37.—SECTION THROUGH COILS OF CANCER CELLS AT Z (FIG. 35).

grafted on the ulcer, but he certainly has an ulcer; indeed, there may be only an ulcer, and no cancer.

A similar argument is applicable to vomiting of blood. Some cancers of the stomach give rise to hæmatemesis and melæna. This cancerous gastrorrhagia, even though it may be profuse, is not comparable with the vomiting of blood seen in ulcer. An individual who has the appearance of cancerous cachexia, and suffers, or has suffered during some period of his

illness, from repeated and profuse hæmatemesis, has perhaps a cancer grafted on the ulcer. He certainly has an ulcer, and cancer may be absent.

On reading again the cases mentioned by Trousseau in his lectures on the diagnosis of cancer and ulcer of the stomach, it seems to me that two of the patients whom he considered to be suffering from cancer had really an ulcer on which the cancer was grafted.

I believe that, had Trousseau known of the coexistence of cancer and ulcer, he would perhaps have modified his opinion. One of his patients in whom the gastric lesions had been present for seven years—a very long period for cancer—had had the cardinal symptoms of ulcer: acute pains in the stomach and profuse vomiting of blood. Furthermore, the man, whose mother had cancer, had, I believe, grafted on his ulcer a cancer, which formed in the anterior wall of the stomach.

The statement regarding the acute pain and profuse hæmatemesis accompanying ulcer, with or without cancerous degeneration, is also applicable to suppurative perigastritis, which has often been set down to cancer. Feulard and Brechoteau have collected cases of anterior phlegmonous perigastritis, periumbilical phlegmon, and umbilical and gastro-cutaneous fistulæ, resulting from cancer of the stomach. Fournier enunciates the opinion that suppurative perigastritis, if closely looked into, is attributable, not to cancer, but to ulcer—or, at least, to an ulcer on which a cancer has been grafted.

“In cancer of the stomach, suppurative anterior perigastritis commonly assumes the form of periumbilical phlegmon followed by gastro-cutaneous or gastro-colic fistula, and is a complication which is almost exclusively seen when the cancer develops in an ulcer. Everything seems to confirm Bouveret’s opinion that perforation of the stomach is more common when the cancer is grafted on an ulcer. It is especially when the cancer is developed in an ulcer that it tends to grow larger and to invade the neighbouring organs” (Fournier). These assertions seem to be absolutely justified. It is the ulcer, and not the cancer, which eats away and perforates the stomach. A cancer that destroys the stomach to such an extent as to take the liver and the pancreas for its walls, or provokes phlegmonous perigastritis, is almost certainly associated with a perforating ulcer. Every individual, therefore, who has gastric troubles, with cancerous cachexia, and also suppurative perigastritis, which may or may not involve the abdominal wall and cause gastro-cutaneous fistulæ, has perhaps a cancer grafted on an ulcer, but he certainly has an ulcer. It is possible that there is only an ulcer, and not a cancer.

It follows, therefore, that it is generally possible to diagnose ulcer, but that it is at times difficult to diagnose the transformation of ulcer into cancer; and yet this diagnosis is of the greatest importance, for the question of cancer decides the prognosis as to death or recovery. I do not know,



however, of any signs or symptoms which in difficult cases will absolutely confirm the hypothesis of cancer. In many cases a confident diagnosis of cancer has been proved erroneous by laparotomy. Hypochlorhydria and anachlorhydria, though signs of some value in the case of cancer, are here of less importance, for the presence of an ulcer seems sufficient to increase the amount of hydrochloric acid. The presence of induration in the epigastric region has by no means the value which might be supposed, because in non-cancerous ulcers the indurated tissues may feel like a tumour, and, on the other hand, some cancers of the stomach cannot be felt, or do not form tumours. This view also applies to the other signs, including the transitory improvement which treatment may cause in the progress of the cancerous cachexia. Finally, the diagnosis is complicated by the fact that in some patients apparently cancerous cachexia is really the result of ulcer.

This study adds to the gravity of the prognosis in ulcer of the stomach. An ulcer is serious by reason of the pain and vomiting, which may induce loss of strength; it is serious, too, because of such complications as fulminant hæmorrhage, perforation, frequency of recurrence, and the possibility of malignant changes.

#### XIV. GASTRIC POLYADENOMA.

The growth formerly called **gastric polypus**, or **polypous gastritis**, is now more correctly termed an **adenoma**. And as gastric adenomata are multiple (we may find from thirty to several hundred), Brissaud has proposed the term **gastric polyadenoma**. The question of the gastric polyadenoma is entirely anatomical, for the ætiological conditions are still unknown, and the affection has practically no symptoms.

On opening the stomach, the adenomata are seen chiefly in the pepsinogenous regions, such as the great cul-de-sac, inferior border, and greater curvature. They may be as large as a lentil, pea, cherry, or hazel-nut, and the remarkable thing is the polypi are of the same size. It seems as if it were a case of an eruption, in which all the elements are of the **same age** and at the **same stage of growth**. At the commencement the growth is formed by a simple elevation of the mucous membrane; later it tends to become pedunculated. The growths are mobile, like the mucous membrane itself, and never extend beyond its deep layers.

In other cases the adenoma assumes the form of *mammillæ*, which are disposed in lines, and somewhat resemble the convolutions of the brain.

The adenoma is of glandular origin. It is met with in chronic gastritis, ulcer of the stomach, and cancer. It may, indeed, be asked whether the adenoma and the polyadenoma are not intermediate between chronic gastritis and cancer.

## XV. CANCER OF THE STOMACH.

**NOTE.**—In order to avoid repetition, I would ask the reader to study this section in conjunction with the two preceding sections. These three sections are complementary to each other. Pathological anatomy, semeiology, and diagnosis are there treated from slightly different points of view.

**Ætiology.**—In point of frequency, cancer of the stomach ranks first with cancer of the breast and uterus. It is hereditary in one-sixth of the cases, more frequent in men than in women, and usually appears between fifty and sixty-five years. The growth is generally primary, unlike cancer of the liver, which is almost always secondary. It has been asserted that grief, arthritism, and herpetism have some influence on its development, but these hypotheses do not appear likely. Chronic gastritis, ulcer of the stomach, and cancer, have such close connections, that the cancer seems in certain cases to graft itself on the other lesions of the stomach. This question has been discussed in the preceding section.

**Pathological Anatomy.**—The most common varieties are the cylindrical-celled epithelioma, and the encephaloid, scirrhus, and colloid cancers. The pylorus and the lesser curvature are usually affected, and the posterior wall is more frequently invaded than the anterior wall. On opening the stomach, we must not always expect to find a tumour; we may find an ulceration, patch, or ring.

The cancerous tumours form in the interior of the stomach a projection like a donkey's back, which becomes more marked as it extends in area. These solitary or multiple tumours are larger, more fungus-like, softer, more vascular, and richer in milky juice in the encephaloid than in the scirrhus variety. The mucous membrane which covers them is thickened or ulcerated. The ulcerations are of variable size; they may occupy the whole lesser curvature of the stomach, and surround the pylorus in the form of a ring. The ulcer in encephaloid cancer is fungating, with everted edges, and made up of a rose-coloured tissue, which is soft and rich in milky juice. In certain cases the floor of the ulcer is bleeding and fungous, or else, if the whole cancerous growth is deeply destroyed, the tunica muscularis appears denuded, and in part destroyed. There may even be a perforation. These cases resemble at first sight the simple ulcer, and caused confusion prior to the researches of Cruveilhier. Cancer *en cuirasse* spreads in the thickness of the coats of the stomach, without forming a tumour. The annular cancer occupies the pylorus or the cardiac end; in the former case it has little tendency to spread to the intestine, but is often accompanied by dilatation of the stomach; in the latter case it almost always attacks the œsophagus, and as a rule causes contraction of the stomach. The pylorus is by far the most common seat of cancer, and then come the lesser curvature, the cardiac end, the anterior and posterior surfaces.



Perforation of the stomach, acute peritonitis, adhesions, local peritonitis, fistulæ, and ulceration of the vessels, are far less common in cancer than simple ulcer. Cancer readily spreads from the stomach to the neighbouring organs, the dissemination taking place through the subserous tissue, blood-vessels, or lymphatics. The peritoneum, the glands in the gastro-hepatic omentum, great omentum, and mesentery, the liver (according to Brinton, in one-fourth of the cases), lungs, kidneys, sternum, and vertebræ, may be the seat of secondary growths. In some cases adhesions are formed between the stomach and the abdominal wall, and a phlegmon develops in the umbilical region. The abscess often communicates with the cavity of the stomach and with the exterior by means of an umbilical fistula. In some cases, however, we find that the abscess communicates only with the exterior (cutaneous fistula). It is a very rare complication in cancer of the stomach, and Feulard has only been able to collect fourteen cases.

Cancer of the stomach commences in the submucous tissue and in the glandular layer. The glands undergo a lengthening, which is due to the growth of the connective tissue between them. The muscular coat of the stomach is always hypertrophied in the neighbourhood of the cancer, and the hypertrophy may become general. The hypertrophied muscular coat owes its size to the thick lamellæ of connective tissue that divide the muscular bundles. These changes in the glands and muscular tissue are not peculiar to cancer, but are also met with in chronic gastritis.

**Symptoms.**—Cancer of the stomach begins, as a rule, with slight and intermittent dyspepsia; true anorexia is seen rather at a more advanced stage. It commences in some cases with persistent pains in the epigastric region, so that in the first period, which may last for months, it is difficult to say whether the case is one of painful dyspepsia or early cancer. If the dyspepsia is, however, obstinate, or accompanied by rapid loss of flesh and pallor of the skin, or if it supervenes in an old person, especially one born of cancerous stock, the prognosis is serious, even before the appearance of other symptoms.

Pain, either early or late, is seldom absent in cancer of the stomach, but it is less severe than in simple ulcer, and has not a predilection for the xiphoid and spinal regions. It is more diffuse, and tends to radiate towards the hypochondria. Vomiting appears in some cases from the first, together with the other dyspeptic troubles. It may be present throughout the entire disease, but at other times it shows itself only in the last stage, or may be completely absent. The vomited matter is of all kinds: it may be mucous, almost watery, or, very rarely, bilious, and may be rejected in the morning, whilst fasting, or in the daytime. It may consist of food-stuffs. The vomiting follows soon after the ingestion of food if the cancer is at the cardia, but later if the growth is at the pylorus. The vomit often

contains fragments of food and undigested meat, because the hydrochloric acid is deficient both in quantity and in quality. There is but little combined hydrochloric acid, and no free acid. Butyric fermentation gives an odour of rancid butter to the vomit, while putrid fermentation, due to insufficient digestion of the proteids, gives an odour of putrefaction.

**Vomiting of blood** (hæmatemesis) is of great importance, being frequent (42 per cent., according to Brinton), and showing itself under divers aspects. Rejection of pure blood is less common than in simple ulcer, and the vomited matter is generally blackish, like coffee-grounds or soot diluted with water. The colour results from the contact of the blood with the acids of the stomach and with the food-stuffs. In some cases the hæmatemesis is so slight that it may pass unnoticed unless the vomit is carefully examined. If the blood passes into the intestine, it is voided in the stools in the form of melæna. In a fair number of cases hæmorrhage from the stomach is revealed by melæna without hæmatemesis. Gastrorrhagia is usually a late symptom; it is due to the ulceration and softening which invades the vessels of the cancerous mass. "The degeneration and destruction are often complicated by the presence of excrescences and of fungoid growths, which increase still more the quantity of blood supplied." More rarely the hæmorrhage is caused by ulceration of large vessels in the walls of the stomach.

In addition to late gastrorrhagia, we sometimes see early hæmorrhage, which supervenes during good health "as the first symptom of a malady which will certainly carry the individual to his grave" (Trousseau). I think that these early and profuse attacks of gastrorrhagia are rather the result of an ulcer on which cancer has been grafted.

At some period of its development (80 per cent., according to Brinton) cancer of the stomach forms a tumour, which is more or less easily discerned, according to its situation. The growth is easy to feel when it occupies the anterior surface, the greater curvature, and the pylorus, but difficult when it is situated at the cardia, or on the posterior surface and lesser curvature. Tumours of the greater curvature are seated to the left of the median line, while a growth of the pylorus is felt to the right, near the umbilicus. The stomach is sometimes so dilated and drawn down that the tumour is situated at or below the umbilicus.

In some cases the cancer infiltrates the coats of the stomach without forming a tumour (cancer *en cuirasse*), and there is then a feeling of diffuse induration. Sometimes, too, neither induration nor tumour can be felt. The exploration of the abdomen for a tumour may be rendered difficult by the contraction of the recti muscles, which may hide a tumour lying beneath, or cause a phantom tumour. For this reason it is necessary to relax the



muscles, and the patient must flex the thighs and breathe with the mouth wide open.

The cancerous tumour, although but little painful to the touch, is nevertheless more sensitive than the neighbouring parts. When the growth constricts the pyloric orifice, it causes **dilatation of the stomach** and exaggerated resonance in the epigastric region.

The tumour may vary in position, according as the stomach is full or empty. On abdominal palpation the tumour most often appears mobile, but this apparent mobility cannot be relied upon, for cancer of the posterior surface may be adherent to the pancreas, and a growth in the anterior surface may be fixed to the abdominal wall. Cancer of the pylorus often adheres to the pancreas, gall-bladder, duodenum, small omentum, liver, or glands. In 300 cases of laparotomy for cancer of the stomach, the growth, when pyloric, was free from adhesions in only fourteen cases. When, therefore, a cancer of the stomach is explored and thought to be mobile, the mass which moves includes both the cancer and its adhesions. It is important to remember these facts from the point of view of operation.

Enlarged glands are more common in cancer of the stomach than in cancer of other viscera, not only in the subclavicular triangle, but also in the groin and axilla. The glands are hard, painless, and movable.

The **general symptoms**, which are not pronounced at the commencement of the disease, become characteristic. Pallor of the skin is followed by a straw-coloured tint; the patient shows a dislike for food; the strength fails, and diarrhoea is frequent. Fever is often seen; the loss of flesh becomes extreme; the voice grows weak; the skin becomes dry and cracks, and the stage of cachexia appears. During this stage the intellectual faculties remain intact, and the patient is conscious of his decay. Oedema and dropsy appear; the feet and legs become infiltrated, and the infiltration reaches the thighs, scrotum, and sometimes the hands and face, though there is, as a rule, no trace of albumin in the urine. This general oedema must not be confounded with the local and sometimes early oedema due to venous thrombosis (**phlegmasia alba dolens**). Trousseau was the first to note the relation between obliterating phlebitis and cancer. We shall refer to this point under Diagnosis.

**Complications.**—Certain complications, though frequent in ulcer, are exceedingly rare in cancer. We find fatal hæmorrhages are exceptional, whilst they are relatively frequent in ulcer. Perforation of the stomach and superacute peritonitis are practically unknown in cancer, whilst they are far from being rare in ulcer. Cancer, like ulcer, may cause purulent and gangrenous fistulæ. Adhesions are formed in the region of a cancerous ulceration, which sometimes results in perforation and local peritonitis. The peritoneal fistula thus formed may open into the colon (gastro-colic

fistula), or may involve the umbilical region, as I have previously remarked.

Amongst the complications secondary deposits in other organs must be noted. Sometimes they result from direct propagation, and it is through adhesions that the cancer attacks the liver, glands, spleen, pancreas, intestine, and abdominal wall. Sometimes there is general cancerous infiltration in the true sense of the word, by means of the lymphatics or bloodvessels.

**Diagnosis.**—I have just described the usual course of cancer of the stomach, with its insidious onset, accompanied by dyspeptic troubles, gradual wasting, vomiting, hæmatemesis, and anorexia, as well as the appearance of the tumour and the cachectic stage. Cancer of the stomach, however, does not always run such a regular course. In one case the usual signs are absent, and the disease is latent. We find neither vomiting, hæmatemesis, nor tumour. The patient has malignant cachexia, but the seat of the lesion is undecided. Or, again, we may find a tumour, but no symptoms of cancer. In another case, while the cancer of the stomach is at an early stage of its evolution, secondary deposits in the liver attract attention, and carry off the patient, masking the lesion in the stomach. In some cases the patient has anorexia, hæmatemesis, cachexia, and epigastric tumour, so that the diagnosis points to cancer of the stomach; but in a few months recovery gives the lie to the diagnosis. In the preceding section I have dwelt on this point in diagnosis at some length.

The following summary shows the errors which may occur in regard to cancer of the stomach :

Vomiting, hæmatemesis, tumour, and cachexia are the classical signs of cancer of the stomach; but they may be due to extensive ulcers, with thick, indurated edges. This fact holds good both in simple (Trousseau's and Rommelaere's cases) and in tubercular ulcers (Bréchemin's case).\*

The above signs, though classical of cancer, may exist in chronic gastritis, with thickening of the walls (hypertrophic submucous sclerosis), as in a remarkable case reported by Trousseau, where chronic gastritis was mistaken for cancer.

Vomiting, hæmatemesis, and cachexia, may exist in dilatation of the stomach, and cause an erroneous belief in the existence of cancer (Dujardin-Beaumetz).

Vomiting, hæmatemesis, epigastric tumour, and cachexia, may exist when the stomach is healthy. In such a case the tumour is formed by a cancer of the omentum, pancreas, or mesenteric glands, or by a peritoneal thickening (Leube). The cachexia is due to cancer in one of the above-

\* Whilst I was house-physician to Potain, one of his patients had hæmatemesis, progressive cachexia, and a tumour in the epigastric region. The diagnosis pointed to cancer of the stomach. At the post-mortem examination we found cancer in the glands. He had been operated on two years previously for cancer of the left testicle.



mentioned regions, and the hæmatemesis proceeds from the stasis in the gastric circulation, caused by compression of the gastric veins.

Doubt may also exist in certain **periumbilical phlegmons**, as mentioned under the pathological anatomy. In the case quoted\* the phlegmon was evident, when the symptoms of cancer were not appreciable.

This list proves how difficult—I might almost say impossible—it is in some cases to diagnose cancer of the stomach. Let us consider the signs of cancer of the stomach one by one, and see what is their respective value in diagnosis.

Acute epigastric pain, which pierces the patient through and through (xiphoid and spinal points), and comes on in paroxysms after meals or during digestion is caused by ulcer of the stomach. It may also be seen in cases of acid dyspepsia. The pain is less common in cancer; it is also less acute, more diffuse, and appears later.

**Vomiting** of bright or dark blood, with or without melæna, after a longer or shorter period of gastralgia, is caused rather by ulcer than by cancer; but hæmatemesis is of all the symptoms the most uncertain in diagnosis. Hæmatemesis in cancer is, it is true, less common and less profuse, and the blood is more mixed with food, and more of a “coffee-ground colour” than in ulcer; but these signs are inconstant, and in ulcer, gastritis, or dilatation of the stomach, attacks of hæmatemesis may supervene, and be exactly alike in character.

If we find epigastric swelling or induration in a patient who has vomiting, hæmatemesis, loss of flesh, and cachexia, the tumour is, as a rule, considered the most important point in the diagnosis of cancer. And yet I do not hesitate to say that the tumour most often causes the error in diagnosis. As long as the patient, though suffering from other symptoms, has no tumour, we think of ulcer; when the tumour appears, cancer is diagnosed. The following cases are a sufficient proof:

In 1888 Kolatschewsky performed pylorotomy on a boy with a hard, movable tumour of the pylorus, which was as large as an apple, and was looked upon as cancerous. The operation revealed a healed gastro-duodenal ulcer, surrounded by glands. The patient recovered. Billroth diagnosed cancer of the pylorus, and performed pylorotomy. Salzer, who reported the case to the Medical Society of Vienna, in December, 1887, proved that it was a case of ulcer of the stomach. Orthmann diagnosed cancer of the pylorus in a woman forty years of age. He performed pylorotomy on May 17, 1889. The operation revealed a cicatrized ulcer, with indurated edges. In June, 1884, Southa operated for cancer on a patient, who presented a hard and mobile tumour in the neighbourhood of the umbilicus, with all the symptoms of cancer. The patient succumbed, and a fibrous constriction of the pylorus was found, but not a trace of cancer.

In one of Chaput's cases the patient showed symptoms which might have been due to ulcer or cancer of the stomach. Brissaud thought, from the epigastric tumour,

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\* Dieulafoy, “Diagnostic du Cancer de l'Estomac,” *Sem. Méd.*, January 4, 1888.

that it was a case of cancer, and the patient was operated upon. It was then found that the tumour was due to an abscess of the pancreas, following a perforating ulcer of the stomach.

A woman came under the care of Terrier for gastric trouble, which presented the complete picture of cancer of the stomach, with epigastric tumour. Terrier performed laparotomy, and found adhesions between the stomach, liver, and anterior wall of the abdomen, but not cancer. These adhesions (probably due to ulcer of the stomach) were broken down, and the patient recovered. On this point Terrier states that Landerer has published three cases of laparotomy for cancer of the stomach. The operation showed that the tumours were due, not to cancer, but to adhesions, the excision of which brought about a cure. Doyen has reported several cases in which an ulcer with adhesions has been taken for a cancerous tumour.

This series of cases shows, I think, that the presence of an epigastric tumour is often a cause of error in diagnosis.

Progressive cachexia, with loss of appetite, straw-coloured complexion, and œdema of the legs, is caused by cancer of the stomach. Similar symptoms, however, may be present with ulcer, gastritis, and dilatation of the stomach, with or without spasmodic contraction of the pylorus, in which case the cachexia is caused by the hæmatemesis and vomiting of food, with consequent malnutrition.

Phlebitis obliterans is a valuable sign noticed by Trousseau. "When you are in doubt between chronic gastritis, simple ulcer, or carcinoma of the stomach, **phlegmasia alba dolens** of the leg or arm is a positive indication in favour of cancer." Trousseau later proved this fact in his own case; indeed, the appearance of phlegmasia in the leg led my venerated master to affirm the existence of cancer of the stomach, from which he died eight months later. Although **phlegmasia** has been found by Bouchard in dilatation of the stomach, it remains, none the less, one of the most valuable signs of distinction between cancer and ulcer.

Rommelaere believed that it was possible to base a diagnosis of cancer on the **diminution of the urea**. The amount of urea is lowered in cancer, but as similar diminution exists in many disorders of nutrition, this sign loses its value.

**Enlarged glands** above the clavicle are common in abdominal cancer, and notably in cancer of the stomach. They are found on the left side five times as often as on the right side. Whatever explanation is given for this distant metastasis of the primary lesion, it is none the less true that this sign is of value. In a doubtful case, however, it does not prove the existence of cancer of the stomach, because enlargement of these glands has been found in ulcer of the stomach.

It has been suggested that **examination of the chyme**, taken during digestion, might furnish useful information; and at the present time this analysis is commonly made, but it has not led to any fixed results.

The amount of hydrochloric acid is of great value in diagnosis. It is



present normally in the gastric juice in the proportion of 1·74 per 1,000, but it is absent when the stomach is at rest. The other acids of the gastric juice, and amongst them lactic acid, are formed from the food-stuffs. Regarding their formation, Ewald divides the process of digestion into three stages : In the first stage, which lasts from ten to thirty minutes, lactic acid is found in the stomach ; in the second stage, free hydrochloric acid exists side by side with the lactic acid ; but in the third stage, which commences half or three-quarters of an hour after the beginning of digestion, the lactic acid has generally disappeared, and hydrochloric acid alone is found. The hydrochloric acid must therefore be looked for during this stage.

The method requires care. The patient is first given a **test meal**, consisting of two small rolls and a cup of tea, without sugar or milk, taken when the stomach is empty. An hour or an hour and a quarter later the chyme is withdrawn by means of Debove's tube, or the siphon-tube may be first filled with a little water, and the hydrochloric acid is tested for in the chyme thus withdrawn. The acid may be detected by various reagents, such as methyl-violet or Congo red, which changes to a blue colour. Lépine prefers *vert brilliant*.\* This stain, diluted with water, loses its greenish colour and becomes blue. If 2 or 3 drops of a concentrated solution of *vert brilliant* be mixed with a few centimetres of filtered chyme containing **hydrochloric acid**, the mixture changes from blue to green, if the proportion of hydrochloric acid be from 0·18 to 0·19 per 1,000. The mixture becomes yellow, if the proportion of hydrochloric acid be from 0·19 to 1 per 1,000. This reaction is important, because lactic acid has practically no action on *vert brilliant*.

To have its full value, the test should be followed by a quantitative estimation of the free hydrochloric acid and other elements (acids of fermentation, combined chlorine), as well as of the pepsin and rennet ferment.

From our present point of view, **hypersecretion** of the hydrochloric acid, which may amount to 3 and 4·5 per 1,000, instead of 1·7, occurs in simple ulcer, in the gastric crises of ataxia, and in certain cases of hyperchlorhydric dyspepsia. It is never seen in cancer of the stomach, unless a cancer is grafted on an ulcer. **Diminution** or **disappearance** of the hydrochloric acid has been found in amyloid degeneration of the vessels of the gastric mucosa, chlorosis, most cachectic conditions, and alcoholic gastritis ; but it is in **cancer** of the stomach that the **disappearance of hydrochloric acid** is almost the rule. This disappearance is probably due to changes in the gastric juice, caused by the cancerous secretion (Riegel's experiments).

These facts do not show that the absence of hydrochloric acid excludes cancer absolutely, for the acid has been found in cancer. Nevertheless, the numerous cases in which the absence of hydrochloric acid has

\* *Vert brilliant* is probably identical with emerald, smaragd or malachite green.

prevented errors in diagnosis prove that the estimation of hydrochloric acid is a valuable method. To differentiate between the anachlorhydria of cancer and that of chronic gastritis it is important to test for the ferments. Total disappearance of the pepsin and rennin point rather to chronic gastritis; the disappearance of the pepsin alone would be in favour of cancer.

Examination of the blood and diminution in the colour index in cancerous patients have not yet given definite results.

I have tried to show in this detailed and critical survey the extreme difficulty at times experienced in making a diagnosis of cancer of the stomach, and I cannot dismiss the question without mentioning dyspepsia in neurasthenic patients. We have all seen neurasthenic patients who suffer from dyspepsia or gastralgia, with or without hyperchlorhydria or hypochlorhydria. They lose their appetite, vomit, grow thin, and are firmly convinced that they have cancer of the stomach. Careful examination of the neurasthenic symptoms will eliminate the idea of cancer.

The **diagnosis** of the **situation** of the cancer must now occupy our attention. Cancer of the cardiac orifice is generally confounded with cancer of the œsophagus, because the growth is rarely limited to the cardiac orifice. The food is arrested at the constriction, and is rejected soon after ingestion. Exploration with the bougie shows the seat and extent of the constriction. Cancers of the cardiac orifice and of the lesser curvature are very difficult to palpate. Cancer of the pylorus often causes stricture and secondary dilatation of the stomach. The vomiting comes on some time after meals; the loss of flesh is rapid, and the cachexia appears early. The tumour, which can be felt, remains fixed in the same region. Cancers of the **curvatures** and **surfaces** of the stomach are less rapid in their progress than those of the **orifices**, because they do not affect the passage of food. Vomiting is not so common, loss of flesh occurs late, and cachexia is slow to appear. Cancer of the greater curvature is remarkable for its mobility, and alters its position according as the stomach is full or empty. If the stomach is much distended, the tumour may occupy the most varied positions in the abdomen.

**Duration.**—Cancer of the stomach has an average duration of some twelve to eighteen months, but it may last even longer if it does not involve the orifices, and allows the passage of food. In young subjects—under thirty years of age—its course is usually rapid. Death is the termination of cancer, and is due to cachexia, repeated hæmatemesis, or secondary growths. Perforation of the stomach and peritonitis, which are relatively frequent in ulcer, are exceptional in cancer.

**Treatment.**—We now come to the treatment. The early dyspepsia must be treated with alkalis, lime-water, Vichy water, and prepared chalk. Milk diet, associated with easily digested food, is indicated in the early stages. Vanilla and coffee ices, or ices containing 2 ounces of meat-juice, agree well.



The vomiting and pain may be checked by small doses of **morphia** and **cocaine** in solution. If the pain does not yield, it must be quieted with injections of morphia. Hæmorrhage is treated with astringents, perchloride of iron, and iced drinks. Careful lavage is of service by preventing decomposition, and favouring the tolerance of the organ for food. The stomach is washed out every morning with luke-warm water, to which bicarbonate of soda has been added. When the fluids in the stomach undergo decomposition, a solution of choral (5 to 10 parts per 1,000) may be employed. If the stomach perform its functions badly, and if anorexia or a tendency to vomiting be present, meat-powders, mixed with milk or chocolate, may be given. This latter operation may be performed by means of a tube, shorter than the one used for lavage, because there is no need for it to pass into the stomach. When feeding by the stomach becomes impossible, either from intolerance or from constriction of the cardiac or pyloric orifice, nutrient enemata should be given. One glass of milk, with the yolk of an egg, 2 spoonfuls of liquid peptone, 5 drops of laudanum, and 15 grains of bicarbonate of soda (Dujardin-Beaumetz), make an excellent formula.

The **surgical** treatment of cancer of the stomach gives fairly satisfactory results. It consists in performing partial or entire resection of the organ. The object of gastro-enterostomy is to unite a portion of the posterior surface of the stomach near the pylorus to the first part of the jejunum. In order to be efficacious, surgical treatment must be early.

The surgical treatment varies according to the case. If the lesion is not very extensive, and especially if it occupies the pylorus, and has not invaded the neighbouring organs, pylorectomy should be performed. The earlier the operation is done, the better the chance of success. Statistics show that many patients have enjoyed excellent health for several years after operation: Wölfler's cases, four, five, and seven years; Löbker's cases, five and seven years; Hahn's cases, four and seven years. These facts are encouraging, and prove that excellent results may follow operative measures in cancer of the stomach.

When resection is not possible, we must be content with gastro-enterostomy (Roux's method). The jejunum is cut through, about 3 inches from the duodeno-jejunal angle; the lower end is joined to the posterior surface of the stomach through an opening in the transverse mesocolon; and the upper end of the cut jejunum is then joined to the intestinal loop about 2 inches below the anastomosis with the stomach. By this means the food passes from the stomach into the jejunum, and, on the other hand, the bile and pancreatic juice pass from the duodenum into the jejunum. There is no risk of a vicious circle. The peptic ulcer of the jejunum which sometimes follows in gastro-enterostomy has never been seen in gastro-enterostomy for cancer of the stomach.

## XVI. SYPHILIS OF THE STOMACH.

The following case from my clinical lectures\* is typical :

A man had suffered for eighteen months with the classical signs of gastric ulcer. He complained of pains in the epigastrium and spine, which became more severe after meals, and were frequently followed by vomiting of food. He was thought to be suffering from *ulcus simplex*, and strict milk diet was prescribed. Milk, kephir, ice, and morphia were given, while cupping and the actual cautery were applied to the epigastrium. A few weeks afterwards the patient, as he did not improve, left the hospital. The pain, however, being just as bad, he soon sought further advice. The symptoms had not changed. He suffered from the same epigastric and spinal pains, gastric intolerance, and vomiting of food. He was again treated for *ulcus simplex* with milk diet, alkalis, bicarbonate of soda, counter-irritants, and frequent cuppings to the epigastric region and to the back. After treatment extending over a period of three months, he left the hospital without appreciable improvement, but soon came back, because the gastric pain was more severe, and the vomiting of food was frequent. One night he had a profuse hæmatemesis, the clots being so large that he pulled them out of his mouth with his fingers.

During the eight months that he was in hospital, he had most careful treatment. Milk diet and alkalis were again tried, but as this régime did not give the expected results, a diet composed partly of eggs and partly of meat-powder was prescribed. The stomach was washed out every day for three months, and counter-irritation was applied in various forms, including dry- and wet-cupplings, tincture of iodine, five blisters, and many applications of actual cautery. As the disease was so stubborn, the question of neurosis was raised, although the patient did not show a single sign of hysteria. Douches, medicated baths, and electrical treatment were also prescribed, but all the remedies made no difference.

He left the hospital, but soon came back under another physician in the annex of the Hôtel-Dieu. He still showed all the symptoms of *ulcus simplex*, and a second hæmatemesis, more profuse than the first, occurred. He wasted, suffered continually, and got no sleep. As the means hitherto employed had failed, and the ulcer was so rebellious to treatment, he was advised to submit to surgical treatment.

He now came under my care. I found him squatting on his bed, stupefied by pain, his eye wan and his expression dull. The symptoms left no doubt as to the diagnosis of *ulcus simplex*. The pain was clearly marked at the xiphoid and spinal points. He said that this pain stabbed him through and through, and deprived him of rest. It was most severe after meals, and came on even after drinking a little milk. The stomach was so intolerant that milk and food were rejected half an hour later. I could find no dilatation, but the pit of the stomach was very tender on pressure, and the patient obtained slight relief only when lying on his right side. During my examination I noticed on the legs some scars suggestive of syphilides. He stated that he had had syphilis three years previously. He was at that time in the St. Louis Hospital, under Fournier, for syphilides of the skin, mucosæ, scrotum, and mouth, and ulcerated syphilides of the legs. He remained only a fortnight under Fournier, and it was ten months after these syphilitic lesions that the first symptoms of gastric ulcer appeared. It was therefore reasonable to suppose that the gastric symptoms were of a syphilitic nature. This hypothesis, already put forward by Kahn, was the more likely as the milk diet and other measures which generally improve or cure *ulcus simplex*, had given in this case no result after a year and a half.

Before prescribing specific treatment, I wished to satisfy myself as to the condition

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\* "Syphilis de l'Estomac" (*Clinique Médicale de l'Hôtel-Dieu*, 1898, 4<sup>me</sup> Leçon).



of the patient. I therefore ordered milk diet, but no medicine. The pains in the stomach and the vomiting continued as before, and the milk was vomited in a liquid state or in clots. I then ordered a daily injection of biniodide of mercury. The symptoms persisted for five days, but after the sixth injection the pains diminished; after about a dozen injections the pain and the vomiting disappeared. The patient slept, although his insomnia had been of such long duration. His features changed from day to day. To prove how much better he felt, he hit his stomach and turned in his bed without feeling the least pain. He was now able to take 4 or 5 pints of milk without vomiting, and his health was much better than it had been for the past eighteen months.

A few days later I added iodide of potassium to the mercurial injections. The pain in the stomach and the vomiting reappeared no more. The patient could not satisfy his hunger, and besides his usual four meals, he asked for extra rations. He put on flesh so quickly that he gained 10 pounds in five weeks. His recovery was complete.

This therapeutic success is attributable, in my opinion, to the specific treatment adopted. For a year and a half milk and kephir had been administered without result; alkalis in large doses had been given in vain, and daily lavage of the stomach for three months at a stretch had done no good. Counter-irritants had been employed, douches had been used, and a prolonged course of baths had been tried, as well as electrical treatment, without changing the condition of the patient. The violent pain, insomnia, vomiting, hæmatemesis, and the loss of flesh resisted every measure employed; surgical intervention seemed to be the only hope, and was all but put into execution. Mercurial injections, however, changed the situation completely, and brought about what a year and a half's treatment for the so-called *ulcus simplex* was unable to accomplish. Under the influence of the specific treatment the improvement was rapid, and the person most astonished was the patient. In such a case it is probable that the process of repair in the lesion is similar to that which we can observe in ulcerating gummata which are accessible to view.

I had the opportunity of observing a similar case at the Hôtel-Dieu:\*

A man was seized one day with profuse hæmatemesis without any apparent reason. He went to bed and took ergotin, but on the following day he had several attacks of hæmatemesis, one after the other. He reckoned the total loss of blood to be about 3 pints. He then came into the Hôtel-Dieu. He had had syphilis; the testes had been involved, and he had at the time suppurating gummata on his neck. All these complications gave way to mercurial treatment. They returned and ceased anew with such regularity that the same origin was evident in each one.

Furthermore, syphilis of the stomach is not so rare as was formerly believed, as the following cases prove:

**Anatomical Cases.** — Gailliard borrows the following case from Murchison:

A man who had contracted syphilis was seized five years later with attacks of nausea, profuse hæmatemesis, and melæna. He died, and the liver was cirrhotic and nodular. An ulcer was found in the stomach, and in the centre of the ulcer an artery had been opened.

Cornil has recorded the following case:

A woman who had pain in the stomach and was unable to digest food, died from pulmonary complications. Post mortem, gummata were found in the stomach and liver.

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\* *Journal de Médecine et de Chirurgie Pratiques*, December 10, 1902.

Along the lesser curvature and in the neighbourhood of the pylorus several dark tumours stood out in relief under the mucous membrane, which was thinned and adherent. The gummata were situated in the glandular layer of the mucous membrane.

In Klebs's case it was a question of syphilitic ulceration of the stomach in a man who had syphilitic ulcers of the skin and throat, with syphilomata of the tongue, liver, and intestine. A rounded ulceration, as large as a shilling, was seen on the mucosa, and the other layers of the wall of the stomach were thickened. The base and edges of the ulcer had a gummatous structure.

Weichselbaum's case :

A man, aged twenty-five, died of facial erysipelas. He had syphilitic lesions of the skull, pharynx, nose, larynx, and liver. The stomach showed a radiating white cicatrix and two ulcers, the base of which was formed by a cicatricial tissue, evidently of gummatous origin.

Birch-Hirschfeld has reported three cases of syphilis of the stomach :

(1) A new-born child had cutaneous syphilides and gummatous nodules in the liver and lungs, as well as a gummatous patch at the pylorus. (2) A woman, forty-five years of age, died, after suffering for four years from gastric trouble. The post-mortem examination revealed on the left lobe of the liver a gumma of the size of an apple, and on the right anterior wall of the stomach, in the pyloric region, a slightly ulcerated gummatous patch. (3) In a man gummatous patches were found in the intestines, the œsophagus, and the stomach.

Wagner has reported the case of a man of fifty-eight, in whom at the autopsy syphilitic lesions of the larynx and of the stomach were found.

Chiari systematically examined the stomach in 243 cases of syphilis. In 145 cases the disease was hereditary, and in 98 cases it was acquired. He often noticed such lesions of the stomach as ecchymoses, hæmorrhagic erosions, cicatrices of ulcers, and ulcers in active progress. An ulcer was found in a man of forty-six years of age who had had syphilis, and died of an attack of hæmatemesis. The coronary artery was eroded by an ulcer.

Fränkel has reported a very interesting case :

A man who had had syphilis was attacked by a gummatous ulceration, which perforated the stomach and caused fatal peritonitis. This case proves that syphilitic ulceration can produce peritonitis. At the post-mortem examination gummata were found in the stomach and intestines.

**Clinical Cases.**—Two clear cases of syphilis of the stomach are found in Andral's "Clinical Medicine" :

CASE 1.—A woman was taken ill with acute gastric symptoms ; the pain and vomiting were incessant, and, in spite of all treatment, the disease made rapid progress. Andral almost gave up hope, but one day the patient complained of difficulty in swallowing. He discovered on the posterior wall of the pharynx an ulcer which appeared to be syphilitic. It was, therefore, a question whether the affection of the stomach which was killing the patient was not due to syphilis. Andral ordered mercury, and improvement was soon manifest. He then prescribed mercurial inunctions. "After the



twelfth rubbing the condition of the patient was no longer recognizable." Recovery was rapidly effected. It is evident that this was a case of syphilitic lesions of the stomach.

CASE 2.—A patient had had the following syphilitic troubles: periosteal nodes, osteoscopic pains, and cutaneous pustules. He was subsequently taken ill with symptoms of phthisis and gastritis, frequent cough, hoarseness, pain in the pharynx, short and hurried breathing, anorexia, pain in the epigastrium, and frequent vomiting. Syphilitic periostitis of the tibia supervened, and it was a question whether the other complications were not also syphilitic. Mercurial inunction was prescribed, and recovery followed.

In this case, too, we must admit the syphilitic nature of the lesions in the stomach, for the gastritis and vomiting yielded to mercurial treatment.

Fournier communicated to the Académie the following cases, of which I give a résumé:

Some thirty years ago I attended a beautiful girl suffering from syphilitic rupia of the back. She recovered rapidly. Ten years afterwards she sent for me, and I found her moribund. By her side was a basin full of blood. For the past three or four months she had been vomiting blood, in spite of all the usual remedies. I prescribed iodide of potassium. A dramatic change took place and recovery was rapid. Six or seven years after this a veritable spectre came into my consulting-room. It was this woman. She had just come from Italy, where she had been seized with fresh attacks of hæmatemesis. She asked for iodide of potassium, which the doctors declined to administer. I prescribed it, and witnessed a resurrection.

Fournier's second case is similar to the first:

A Russian suffering from severe syphilis was taken ill with vomiting of blood, which was cured by specific treatment. He left off treatment, and then suffered from rupia and hæmatemesis, which yielded to iodide of potassium.

The following is a résumé of Dubuc's case, in which syphilis of the stomach simulated cancer:

A man contracted a hard chancre, followed by roseola and tubercular syphilides. Ten years later Dubuc noticed in the epigastric region an indurated projection of the size of a pigeon's egg. There was not the slightest doubt that the tumour was in the wall of the stomach. He grew thinner, digestion was retarded, and there was dull pain in the affected region. It was difficult not to think of the possibility of cancer. In consequence, however, of the antecedent syphilis, treatment with mercury and iodide was prescribed and brought about recovery.

**General Considerations.**—Anatomically, the syphilitic lesions of the stomach are: erosions, ecchymoses, gummata, gummatous infiltration and ulceration, and cicatrices. Clinically, these lesions show themselves by symptoms which, according to their nature and grouping, resemble dyspepsia, gastralgia, ulcer, and cancer of the stomach. One patient has dyspepsia, and is sent to Vichy, Pougues, or Capvern, when the proper treatment is injection of biniodide of mercury.

Another patient, suffering from loss of appetite, retching, vomiting of mucus, and gastric intolerance, is wrongly looked upon as alcoholic, in

spite of every assurance that he has always been temperate. Sometimes, as in one of my cases, there is a group of symptoms which so clearly counterfeits *ulcus simplex* as to cause mistakes. Sharp pains in the stomach, which is worse during the process of digestion, localization of pain to the xiphoid and spinal points, intolerance of the stomach, vomiting of food, and hæmatemesis are present. Moreover, hæmatemesis is not uncommon in syphilis of the stomach. My patient had two severe attacks of hæmatemesis, and Fournier's patients had similar attacks, which yielded to specific treatment.

In some cases the lesion assumes the mask of *exulceratio simplex*. The patient is seized with fulminating hæmatemesis, and dies without having shown gastric symptoms. At the post-mortem examination an arteriole is found open in the exulceration (Murchison).

Finally, the patient suffers from gastric troubles, with loss of flesh, and has also an epigastric tumour, which is thought to be cancerous. As the patient is syphilitic, specific treatment is administered, and a cure is effected (Dubuc).

This polymorphism of syphilis of the stomach proves that there are no signs and symptoms which allow us to make a positive diagnosis. We must, however, remember that, in a patient suffering from the gastric troubles which we have just discussed, syphilis must always be looked for. When it is evident that the patient has had syphilis, and even more so when it is possible to reconstruct the various stages of syphilis which has caused trouble for several years, we must at once prescribe specific treatment. It is the more necessary to arrive at a diagnosis because we must not send to the surgeon a patient suffering from stomach trouble which is rebellious to all ordinary medical means, but which is at once cured by specific treatment.

This treatment must consist in the combination of mercury and iodine, and I would add the former is more important than the latter. I give the preference to injections of an aqueous solution of biniodide of mercury, in doses of  $\frac{1}{6}$  grain per diem.

## XVII. DILATATION OF THE STOMACH.

**Pathogenesis.**—Dilatation is a morbid condition, met with in many affections of the stomach. It is sometimes mechanical, and results from a constriction of the pyloric orifice (cancer, cicatrices following on simple ulcer, spasm of the pylorus), but in this case it is rather a question of distension than dilatation. Sometimes it follows atony of the muscular fibres (chronic catarrh, neurosis, neurasthenia, tuberculosis, general exhaustion, and typhoid fever).

Dilatation is frequent in large eaters and heavy drinkers. According to



Bouchard, dilatation of the stomach is not simply a symptom of various pathological conditions, but a morbid entity, the stomach allowing itself to be distended because its resistance is unequal to the obstacle which it has to surmount. Although dyspepsia and dilatation of the stomach are always associated, the dilatation is the cause more often than the result of the dyspepsia.

**Pathological Anatomy.**—The dilated stomach does not always preserve its normal form (stomach *en bissac*) ; its capacity is such that it may contain as much as 10, 20, or 40 pints of fluid. The increase in size always takes place at the expense of the greater curvature, which is depressed.

The lesions of the muscular layer are very variable, and there may be hypertrophy or atrophy. Amyloid degeneration has been noticed. The mucous membrane generally shows chronic inflammatory changes. Amongst the elements contained in the stomach there is frequently a cryptogam, known under the name of *sarcina*.

**Symptoms.**—The appetite may be diminished or increased, and the thirst is intense. Constipation is the rule, while digestion is delayed, painful, and frequently accompanied by vomiting.

The vomited matter may amount to several pints daily. This enormous loss of fluid naturally diminishes the amount of urine (Kussmaul). The vomited matter is generally mucous, coloured, of foetid odour and bitter taste. The food in the vomit has been ingested, as a rule, two or three days previously. In a few exceptional cases true attacks of hæmatemesis have been noticed. Constipation is followed by attacks of profuse diarrhœa. The patient rarely complains of sharp pains.

The dilated stomach often forms a tumour in the epigastric region. Percussion, which should be carried out when the stomach is empty, reveals extensive hyperresonance, while gentle tapping in the epigastric region yields a **splashing** sound, which is more marked if the patient swallows a glass of water. The **succussion splash**, which is produced on shaking the patient, is similar in nature. Nodules are sometimes seen on the second joints of the fingers ; they are evidence of the rheumatic diathesis, so common in persons suffering from dilatation of the stomach (Bouchard). When the dilatation is of recent date and of moderate extent, we do not find the symptoms just enumerated. As the disease progresses, the dyspepsia, vomiting, and malnutrition cause loss of flesh and cachexia, so that it is often difficult to distinguish between simple dilatation and cancer of the stomach.

In some individuals we see a series of complications, amongst which I may mention hypochondria, vertigo, palpitation, cardiac intermittence, and angina pectoris. I would also mention cramp, contraction of the flexor muscles of the fingers, and epileptiform fits, complications comparable to

those of uræmia, and resulting, according to Bouchard, from the absorption of toxic substances produced by abnormal fermentation in the dilated stomach. Paralyzes, either alone or associated with convulsions, have also been noted.

According to Bouchard, primary dilatation of the stomach, with food stasis and consequent fermentation, is of considerable importance. In such a case it is not simply a question of an individual having a very dilated stomach, as in most cases the dilatation is only very slightly pronounced. Fermentation in the dilated stomach (flatulence) is usually due to diminution of the hydrochloric acid, which under normal conditions has an antiseptic action. In these patients gastric trouble, accompanied with fever, is common. In other words, the description and treatment of dyspepsia in general, and gastritis in particular, blend with dilatation of the stomach. In other cases there is gastric insufficiency (Ewald).

Dilatation of the stomach coincides, very often, with entero-colitis, and, especially, with muco-membranous entero-colitis. The existence of gastro-entero-typhlo-colitis, with distension especially marked in certain parts of the intestinal tract, has much impressed me. Many patients sent to Châtel-Guyon or to Plombières for intestinal treatment also derive benefit by the cure for gastric derangements.

As regards treatment, excellent results are obtained from lavage. The remedies advised for dyspepsia are suitable in dilatation of the stomach. The reader is therefore requested to refer to the chapter on Dyspepsia. Milk diet or dry diet should be prescribed, according to the particular case.

## XVIII. STENOSIS OF THE PYLORUS.

**Stenosis of the pylorus** is characterized by the complete or incomplete, transitory or permanent, impermeability of the pyloric orifice.

**Ætiology.**—The most frequent causes are cancer of the pylorus and ulcer. It is, almost always, a case of an old, callous, indurated ulcer, surrounded by a fibrous plebolith with thick peritoneal adhesions which give the lesion the consistency of a tumour. Sometimes, even, during an operation the surgeon, after grasping the stenosing mass, cannot decide as to the inflammatory or to the neoplastic nature of the tumour. More rarely, the stenosis is due to ulcerations caused by caustic liquid, by syphilitic or by tubercular sclerosis, by gastric polyadenomata, or by plastic, chronic inflammation, which is, after all, nothing but a neoplastic tissue. Let us, further, mention stenosis by compression (neighbouring tumours, peritoneal bands, pericholecystitis).

**Symptoms.**—The common evolution of pyloric stenosis is as follows:—



A patient who, for a long time, has presented the classical picture of ulcer notices that, little by little, the symptoms change in character. The pain and the fits of vomiting which appeared immediately after a meal seem to have lost all relation with eating and drinking. The acute and intermittent pains give way to dull and continued ones. The period between the fits of vomiting lengthens. The patient is able to pass several days without vomiting. He then vomits an abundance of foul-smelling fluid, in which the food ingested a few days previously is recognized. This last characteristic has a diagnostic value of the utmost importance.

Let us now proceed to the physical examination and observe the patient when lying on his back. At times, the stomach is seen to outline itself in the form of a globular, contracted protuberance which appears to creep from left to right, from the cardia towards the pylorus (peristaltic undulations). The hand, when placed over the stomach, perceives the hardening of the organ, which is fighting against the pyloric obstacle. Palpation and percussion, carried out with or without previous inflation, reveal the splashing sound which may be heard below the umbilicus, as far as the pubic symphysis, testifying to the enormous dilatation of the organ. In the case of certain patients, palpation reveals a tumour to the left of the median line, which, however, must not cause us to eliminate the hypothesis of a pyloric tumour. In case of doubt, insutflation will cause the tumour to pass back towards the right.

Catheterization of the stomach gives useful information. This method, when carried out in the morning, on an empty stomach, yields abundant fluid containing free hydrochloric acid, the acids of fermentation, pepsine, and food débris. Examination of the gastric juice after a test meal reveals, according to circumstances, hyperchlorhydria or hypochlorhydria. Finally, radiography and radioscopy assist in the diagnosis of gastric affections.

**Diagnosis.**—Aided by the ensemble of the signs we have just enumerated, the diagnosis of stenosis of the pylorus is relatively easy, but it is not always so, especially in cases of but slightly marked stenosis, the clinical signs of which are defaced, or are uncertain. Furthermore, pyloric stenosis having been diagnosed, it must not be confounded with sub-pyloric, or with mediogastric stenosis.

The gastric crises in tabes are often accompanied by fits of vomiting and gastric intolerance which, at times, simulate pyloric stenosis. In such a case, the characteristic signs of tabes must be sought for systematically. The gastric dilatation of patients suffering from hyperchlorhydria or from hypochlorhydria with fermentation is fairly easily distinguished from pyloric stenosis by the emptiness of the stomach in the morning when fasting, or, at least, by the absence of food débris in the residual liquid.

The presence of food débris is, however, only of value when noted several days consecutively (Lion). When this sign has, however, been duly observed, is it pathognomonic of pyloric stenosis? (Soupault, Bouverey and Devic, Mathieu). Gastrosuccorrhea may be the index of a secretory trouble. It may be caused by retention, by hypersecretion, or by gastric atonia. Now, from the practical point of view, this gastric atonia must not be confounded with pyloric stenosis, because surgical intervention has no influence whatever on atonia, whereas it has a beneficial effect on pyloric stenosis.

Radioscopy often facilitates the diagnosis. The patient, placed in front of the screen, swallows milk of bismuth with gum accacia, and the following radiograms show how the filling of a normal, of an atonic, or of a stenosed stomach takes place.

In the normal condition (Fig. 38), the stomach moulds itself on its contents and, below the air-chamber, A, behaves like a resisting tube, the walls of which separate throughout their entire length in a progressive manner, according to the quantity of bismuth introduced. In atony

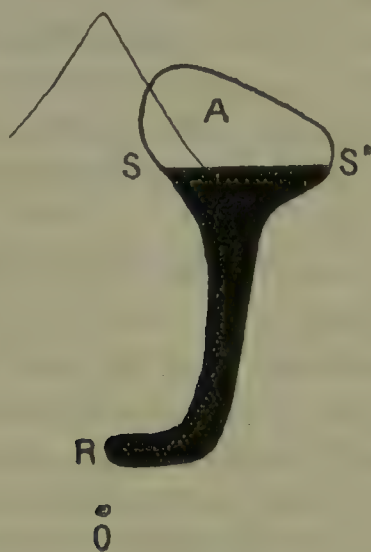


FIG 38.

O, umbilicus; R, pylorus; A, air-chamber; SS', level of bismuth.

(Fig. 39), the atonic walls of the stomach allow themselves to be distended from the commencement and, from the outset, the bismuth falls to the lowest point.

In stenosis of the pylorus (Fig. 40) the walls are more resistant, the peristaltic waves mould the mass of bismuth in a line with festooned edges.

Where is the seat of the stenosis? This is the most important point in the diagnosis, for it happens too often that the surgeon, forgetting the possibility of a sub-pyloric or of a medio-gastric stenosis, is led to intervene uselessly and in an incomplete manner. Sub-pyloric stenosis



closely resembles pyloric stenosis. Hayem bases the differential diagnosis on the presence of the bile in the gastric contents and on the extreme division of the food débris.

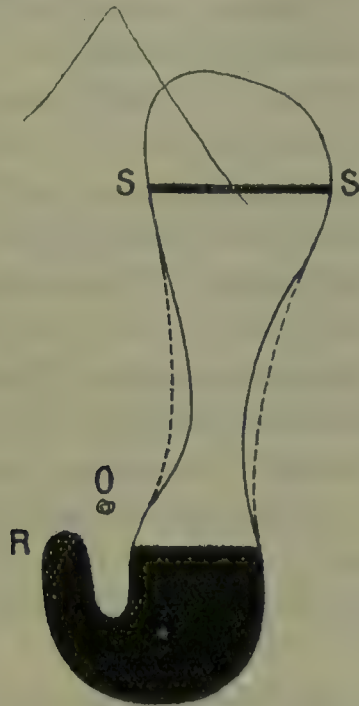


FIG. 39.—GASTRIC ATONY.

O, umbilicus; SS', level of bismuth; R, pylorus.

Mediogastric stenosis with an hour-glass stomach leads to still more frequent errors, for the capital signs of pyloric stenosis are all present, and all the more so as the obstacle is seated close to the pylorus. The



FIG. 40.—STENOSIS OF THE PYLORUS.

differential signs are furnished by catheterization, by insufflation, and by radioscopy.

Catheterization, in the case of hour-glass stomach, produces the following symptoms:—The water introduced into the stomach issues forth only in a very incomplete manner (Jacvorski). After lavage, the fluid comes out clear, and then suddenly is charged with food débris resulting from the evacuation of the pyloric pocket (Bouveret).

After the stomach is apparently empty, the splashing sound is still perceived.

Insufflation furnishes the following signs:—If the orifice of communication be sufficiently permeable, the two pockets swell and the stomach assumes the typical hourglass shape. If the cardiac pocket be very small, it is hidden under the false ribs, so that the bilobulation is no longer apparent. If the orifice of communication be impermeable, the cardiac pocket alone is distended and this gastric pocket, which, generally, remains quite to the left of the median line, points to the bilobulation of the stomach. The diagnosis is completed by radioscopy and by radiography. We shall discuss the semeiological value of each of these signs later. We shall merely remember the frequent concurrence of pyloric stenosis and hour-glass stomach.

To discover whether the pyloric stenosis results from an ulcer or from cancer is often a very difficult pathogenic diagnosis. Let us remember that the neoplastic masses present themselves on the screen, after filling of the stomach by bismuth, in the shape of absolutely colourless white sand.

**Prognosis.**—The prognosis of pyloric stenosis depends on an early and accurate diagnosis. It depends, also, in a great measure on the nature of the lesion which has provoked the stenosis. Left to itself, stenosis, unless it be arrested, condemns the patient to inanition, to infection, and to auto-infection, with vertigo, convulsions and crises of tetanus. The end may be precipitated by rupture of the stomach, which, according to circumstances, causes localized or general peritonitis.

**Treatment.**—Surgical intervention is necessary in every case of contracting stenosis. Opinions differ as regards cases of relatively indolent stenosis. We must never forget syphilitic stenosis. Then even, in case of doubt, we must treat it by injections of biniodide of mercury.

The operations that have been performed for pyloric stenosis are: Pylorotomy, pyloroplasty, gastrectomy, and gastro-enterostomy. Pyloroplasty has quite a limited application; it is suitable only in cases of non-malignant stricture, and consists in dividing the stricture, so that the pyloric passage is made free.

Pylorotomy or resection of the pylorus has been employed especially in cancer; the aim of the operation is to remove the whole of the diseased pylorus, the divided ends of the stomach and duodenum being united by



suture. The operation has not been attended with marked success; the cancer is apt to become diffuse, to spread to adjacent tissues, and to lead to early invasion of the lymph glands.

Gastrectomy implies the partial or complete removal of the stomach. In the case of malignant strictures of the pylorus, it is clearly the only operation that has a chance of affording permanent relief. Unfortunately, most cases of malignant stenosis of the pylorus are not seen until the condition has made such progress as to render the chances of a successful resection very slender. After the diseased portion has been removed, the stomach is united to the jejunum by lateral anastomosis.

At the present time, the operations most commonly practised are: posterior gastro-enterostomy (von Hacker), anastomosis by lateral union, or Roux's Y-shaped method of gastro-enterostomy. In England and America posterior gastro-enterostomy is the operation of election; it is usually performed according to the no-loop procedure, first advocated by the Mayos.

The two first methods have the inconvenience of leaving the patient with a nauseous taste, due to the reflux of the intestinal contents into the gastric cavity, which drawback is absent in Roux's operation, but, as Hofmann, Lion, and Moreau have demonstrated, the reflux of the bile is, perhaps, a safeguard against the recurrence of ulcers, which are always to be feared. In such cases, the secondary ulcer sometimes assumes the form of a peptic ulcer digesting the anastomosis and thus producing aseptic peritonitis, which is, habitually, insidious and, almost always, fatal (Gosset). This complication is, perhaps, less to be feared with von Hacker's than with Roux's operation. Such a complication is, happily, exceptional and, in the majority of cases, the results of the operation, even if considerably delayed, are favourable. If, for complex reasons, the gastric movements be often modified, the digestion remains good, thanks to the facility of the gastric evacuation, which is easily shown on the fluorescent screen. Parmentier and Dénéchau, however, insist on a secondary dyspeptic syndrome, which is said to yield to an appropriate dietetic regimen. These conclusions, so favourable in their entirety, do not, unfortunately, apply in stenosis due to cancer. In this case, gastro-enterostomy is merely a palliative operation which prolongs the life of the patient, lessens his sufferings and, for a short time, lets him believe that he is cured.

I had under my care a man suffering from cancerous stenosis of the pylorus. He was moribund. After the operation, nutrition was kept up so well that he gained 12 kilos in weight. He believed that he was cured.

## XIX. GASTRORRHAGIA—HÆMATEMESIS.

NOTE.—As I am anxious to avoid repetition, I cannot undertake in this section a complete description of gastrorrhagia and of hæmatemesis. I would ask the reader to refer to the sections on acute ulcerations of the stomach (pneumococcal erosions, appendicular erosions, exulceratio simplex), on chronic ulcers of the stomach (simple ulcer, syphilitic ulcer), and on cancer of the stomach. In these different sections gastrorrhagia and hæmatemesis are studied in great detail.

**Definition.**—The words **hæmatemesis** and **gastrorrhagia** must not be confounded. Gastrorrhagia is bleeding from the surface of the mucous membrane, or from the wall of the stomach, the blood being discharged subsequently into its cavity, whilst hæmatemesis simply denotes the vomiting of blood which comes from the stomach, or has been discharged into the stomach after coming from a neighbouring region. It is clear, therefore, that hæmatemesis and gastrorrhagia are not always associated. Either may occur alone, as in the following examples: An individual vomits blood, which has entered the stomach in consequence of epistaxis or profuse hæmoptysis: in this case there is hæmatemesis without gastrorrhagia. Another individual, suffering from cancer or ulcer of the stomach, has an attack of hæmorrhage from the stomach, but the bleeding is not followed by vomiting, and the blood passes from the stomach into the intestine, giving rise to **melæna**: this is an example of gastrorrhagia without hæmatemesis.

**Ætiology.**—Gastrorrhagia arises from many causes—to wit, lesions of the stomach, traumatism, contusion, chronic gastritis, cancer of the stomach, and especially simple ulcer and acute ulcerations, pneumococcal or appendicular erosions, and exulceratio simplex. In three of the preceding sections I have discussed the bleeding which accompanies hæmorrhagic necrosis of the mucosa, destruction of the mucous membrane and of the muscularis mucosæ, erosion of the arterioles under the muscularis, etc. Further reference is, therefore, superfluous.

Lesions obstructing the portal circulation cause stasis, with or without gastric erosions, sometimes followed by hæmorrhage. Atrophic cirrhosis of the liver causes varices of the stomach (Letulle), and especially of the œsophagus, which may rupture and lead to profuse hæmatemesis. This point will be discussed under Laënnec's Cirrhosis.

In the three cases quoted by Gailliard fulminant hæmatemesis was caused by the rupture of miliary aneurysms. Active congestion of the stomach accounts for the so-called nervous (hysteria) and supplementary (suppression of the menses, hæmorrhoids) gastrorrhagia. Attacks of gastrorrhagia which supervene in black variola, typhus, icterus gravis, yellow fever, etc., are due to changes in the blood and capillaries.

**Symptoms.**—Gastrorrhagia is not always preceded by prodromata.



Rigors, pallor, fainting-fits, which accompany profuse hæmorrhage from the stomach, are not prodromata, but symptoms associated with gastrorrhagia. They are the consequence thereof, and sometimes, when hæmatemesis is absent, they are the sole index of hæmorrhage from the stomach. The cases of **gastrorrhagia without hæmatemesis** are worthy of recognition, and often pass unnoticed, though they are more frequent than is generally believed. It may be the first symptom of ulcer or cancer of the stomach. "People in good health," says Trousseau, "are suddenly taken ill with a vague feeling of malaise; they grow pale, and suffer from syncope. A few hours later, on going to stool, they pass fæces as black as pitch, and for some time they remain weak, and suffer from loss of appetite, with pallor of the skin, and then health returns. These troubles may recur at longer or shorter intervals, and are often mistaken, not only by the patient, but also by the physician."

In this first class of cases, gastrorrhagia, whether accompanied or not by pallor, fainting-fits, or syncope, is not followed by hæmatemesis. These cases are fairly frequent. Although many people with cancer never vomit during the whole course of the disease, yet if their stools are carefully examined, pitchy fæces will be found (*melæna*), indicative of gastric hæmorrhage.

Gastrorrhagia is generally followed by hæmatemesis. The vomiting of blood shows itself in different forms.

The quantity of blood vomited may be large or small. Slight hæmatemesis may pass unnoticed. Vomiting of food is frequent in ulcer and cancer of the stomach. On careful examination of the vomit, collected in a basin, a blackish dust, like coffee-grounds, is seen on the surface of the liquid or on the sides of the vessel. This dust is due to slight hæmatemesis, and the microscope will confirm the diagnosis.

In severe hæmatemesis the blood is rarely red; most often the vomited blood is blackish, like soot dissolved in water. Blackish clots of the size of a nut, a small pear, or even larger, are mixed with the fluid. Under *Acute Ulceration of the Stomach* I have mentioned several cases in which the vomit contained some 3 pints of blood, either liquid or in clots. Sometimes the hæmatemesis is fulminant, and due to the opening of an arteriole, which causes death from hæmorrhage. Every intermediate degree is seen between the slight hæmatemesis, in which a blackish dust floats in the mucous or viscid fluid, and the fulminant hæmatemesis.

**Diagnosis.**—The diagnosis of gastrorrhagia rests upon the existence of hæmatemesis and *melæna*, but care must be exercised to make sure that the blood really comes from the stomach, as it is well known that the blood in profuse epistaxis may pass into the stomach, and be got rid of later in the vomit or the stools.

We must next diagnose the cause. Is the gastrorrhagia due to a lesion of the stomach, such as acute ulcerations, ulcer, or cancer? and even if the subject is in good health, is it not an early warning of cancer? Is it the result of a lesion in the liver (atrophic cirrhosis), or is it caused by gastric congestion (hysteria, supplementary hæmorrhage)? A reply can only be given to these various questions by carefully studying the past history of the patient and the symptoms preceding the gastrorrhagia. The diagnostic value of hæmatemesis has been discussed at length in the preceding sections, and especially under *Exulceratio Simplex*.

**Treatment.**—On the question of treatment I would refer the reader to the section on *Exulceratio Simplex*. Every patient suffering from gastrorrhagia must be put on starvation diet, and given large injections of artificial serum. Rectal feeding is necessary (nutrient enemata, peptones, lactose, eggs, etc.).

## XX. VISCERAL PTOSSES.

The abdominal viscera may undergo displacement from stretching of their suspensory ligaments. The interesting study of visceral ptoses was first undertaken by F. Glénard. The heart even is not exempt from ptosis (Rummo). As I shall devote a special section to Floating Kidney, I shall now mention only the ptoses of the stomach, intestines, liver, and spleen.

**Gastroptosis** is downward displacement of the stomach. It shows itself by abnormal prominences in front of the vertebral column (prominence of the pancreas, of the superior fold of the stomach, and of the stenosed transverse colon), and causes gastric succussion and downward displacement of the lesser curvature of the stomach, which is well marked after the insufflation of the organ. Gastroptosis must not be confounded with dilatation of the stomach, with which it has many symptoms (F. Glénard) in common.

**Enteroptosis** is prolapse of the intestine. It is characterized by relaxation of the abdominal walls and flattening of the epigastric region, where the pulsation of the aorta may be seen and felt. The patient experiences a feeling of relief when the abdominal wall is lifted upwards and backwards by a person standing behind him.

**Hepatoptosis** is downward displacement of the liver, which is felt below the false ribs, while the upper limit of the hepatic dullness is lowered.

**Splenoptosis** is downward displacement of the spleen. It includes the classical mobile spleen, which is always enlarged, as well as true splenoptosis, which is always accompanied by hepatoptosis, and sometimes by nephroptosis.

Visceral ptoses are rarely isolated; as a rule, several organs are affected.



The functional signs, which vary slightly, according to the organ affected, are chiefly of the neuropathic kind—asthenia, dyspepsia, twitching and sensations of weight and emptiness, to which vertigo, insomnia, headache, and nervous instability are added. The **ætiology** of visceral ptoses depends, according to Glénard, on **hepatism**, or the hepatic diathesis, which produces more or less marked functional weakness. Support of the organs by special belts forms the basis of treatment, though surgical intervention is necessary in obstinate cases. In hepatoptosis it has given splendid results (Glénard, Marchant).

## CHAPTER V

### DISEASES OF THE INTESTINE

#### I. ACUTE ENTERITIS

**Definition.**—**Enteritis** is inflammation of the mucous membrane of the intestine. When the stomach takes part in the inflammation, there is **gastro-enteritis**. The word “enteritis” refers to inflammation of the small intestine. If the large intestine is also inflamed, there is **entero-colitis**. Inflammation of a portion of the intestine is called, according to circumstances, **duodenitis** (duodenum), **typhlitis** (cæcum), or **rectitis** (rectum).

Before describing enteritis, it will be useful to see what we mean by the term employed. It is essential not to confound enteritis with one of its usual symptoms—**diarrhœa**—since these different morbid states lead to different therapeutic indications. And yet confusion is frequent. We are too prone to speak of acute or chronic enteritis when the case is one of simple non-inflammatory diarrhœa. Diarrhœa is often associated with intestinal infection, and from slight catarrh to grave enteritis it constitutes an important symptom. In other cases, however, it has nothing to do with enteritis, being of different origin. To this latter category belong: (1) **sudoral diarrhœa**, which is caused by a disturbance of the functions of the skin (sudden suppression of perspiration); (2) **nervous diarrhœa**, which arises from mental emotions, and constitutes one of the disorders of secretion in **tabes dorsalis** and **exophthalmic goitre**; (3) diarrhœa caused by **irritation**, consequent on abnormal excitement of the glandular adnexa (liver, pancreas), and on the ingestion of certain foods and drinks. These are all simple secretory troubles, and must be distinguished from acute enteritis.

**Ætiology.**—Acute enteritis is a disease of all ages, and in young children has a special importance, which I shall discuss in the next section. It is more frequent in warm weather, and is often brought on by a chill. Chill is well known as an exciting cause producing enteritis in some persons and bronchitis in others. If the primary cause of enteritis is carefully sought for, it will be seen that the disease may be of **infectious** or **toxic** origin. There is no need for me to speak here of certain specific microbes (typhoid fever, tuberculosis, cholera), which provoke specific catarrhs. I allude to those microbes which usually inhabit the intestine, and become pathogenic in certain conditions: such are the *Coli bacillus*, the *Bacterium aceti*, amœbæ, etc. Other microbes ingested with the food pass from the stomach into the intestine, but in order to do this the putrefying food must



find an insufficient antiseptic in the hydrochloric acid. The toxic substances capable of causing acute enteritis are in some cases elaborated by microbes, in others taken into the stomach, or, lastly, are manufactured by the patient, who is already ill (uric acid in gout, carbonate of ammonia in uræmia, changes in the bile).

Certain individuals are predisposed to enteritis, and the influence of the season produces epidemic enteritis.

**Symptoms.**—Enteritis is accompanied by intestinal fermentation, which causes the absorption of toxic products, with the phenomena of auto-infection, so carefully described by Bouchard.

Slight enteritis is not febrile, but the same cannot be said of severe enteritis. Colic and diarrhœa are the first symptoms, and the pain is especially severe at the umbilicus, whence it radiates. Colic sometimes occurs in fits, which may be very painful, accompanied by borborygmi, and followed by loose stools. The evacuations, which are more or less fluid, are formed at first of the contents of the intestine; later they become liquid, yellowish, and composed of serous fluid, mucus, and bile. The appetite is diminished or absent; thirst is great. The tongue is coated, the belly distended and painful. In slight cases these symptoms improve rapidly, and the disease ends in a few days; in severe cases the evacuations are frequent and copious, the enteritis is **cholericform**, the loss of strength is rapid, and the prognosis in children and old people is exceedingly grave.

In **gastro-enteritis**, gastric pains, vomiting, and nausea are added to the preceding symptoms. In **entero-colitis** the diarrhœa-like stools are at times blood-stained and slimy, and as the patient suffers from tenesmus, the enteritis is said to be **dysenteriform**.

**Pathological Anatomy.**—The mucous membrane of the intestines is swollen, red, and congested, especially around the solitary follicles and Peyer's patches. The solitary follicles are swollen near the end of the ileum (psorenteria), and at times small ulcers (follicular ulcers) develop.

**Treatment.**—In the adult we must prescribe a saline purgative, such as sulphate of soda, Pullna water, Birmenstorff water, etc., which is repeated if necessary. After free evacuation, opiates are given, either by the mouth or in enemata. The diet must be strict, and the beverages should consist of rice- or albumin-water, sweetened with syrup. If the colic is very severe, subcutaneous injections of morphia should be given, and opium fomentations applied to the abdomen. Salicylate of bismuth and lime-water in milk may also be employed.

**Duodenitis.**—Both anatomically and physiologically, the duodenum must clearly be differentiated from the small intestine. Its *rôle* assigns to it a special place in the intestinal tract. It is, therefore, natural that the diseases of, and the remedies for, the duodenum should have their share of

autonomy. These ideas, which several authors had enunciated, have been clearly formulated by René Gaultier. By means of the systematic examination of the fæces as a method of functional exploration, he has succeeded in giving a picture of the duodenal syndrome.

## II. INFANTILE GASTRO-ENTERITIS (INFANTILE CHOLERA).

In young children digestive disorders acquire additional gravity from the special importance of the functions of nutrition at this age. Super-acute gastro-enteritis in infants deserves mention, because of its frequency and gravity (H. de Rothschild). We shall also have to describe chronic dyspepsia of infants, which often paves the way for more acute complications. Finally, the study of athrepsia belongs to the digestive disorders of early infancy.

**Acute Infantile Gastro-Enteritis—Infantile Cholera.**—The most characteristic form of acute gastro-enteritis is that which Trousseau described under the name of “infantile cholera.” This affection is most common and severe during the summer. It attacks bottle-fed infants of four or five months of age, or older children, up to about a year or so, who have been dyspeptic since weaning. The child is taken ill with fever and restlessness, and cries constantly. The face becomes pale, the eyes have large circles around them, and prostration succeeds restlessness. The milk is vomited soon after ingestion, but this act is a regurgitation without effort and without nausea. Diarrhoea is present, and the stools are serous, holding in suspension foetid greenish particles, and mixed with gas, so as to form a green froth. The temperature rises to 103° or 104° F.; in some cases it remains high. The skin is dry, the tongue parched, the restlessness constant, and sleep is absent (febrile form). But in other cases (algid form) the initial rise soon gives way to a fall of temperature, which may be as low as 95° F. The little patient is pale or cyanosed, with a bistre tint under the eyes and around the lips. The child wastes with terrible rapidity, and becomes dried up, while the abdomen, at first ballooned and sensitive to pressure, becomes scaphoid. The skin of the abdomen and of the extremities hangs in folds, the eyes sink into their sockets, the scalp falls in at the fontanelles, the pulse grows weak, and the respiration becomes slow and barely perceptible. The patient looks like a corpse, and passes insensibly from life to death. In some cases convulsions, sclerema, and muscular rigidity are present in the last stage. This description is not applicable to all cases. Sometimes diarrhoea is absent (cholera sicca, Hutinel), and yet the toxoinfectious condition presents the same characteristics. In the less acute forms the diarrhoea is not so liquid, and resembles chopped spinach. The general condition fails less rapidly, and there are alternative periods of



improvement and aggravation. The disease may be prolonged for a week or a fortnight. With proper treatment, the child may regain health, and once the disease is checked recovery is relatively rapid. In the very slight forms we may see only vomiting, yellowish or greenish foetid diarrhoea, slight fever, and restlessness, and in a few days the danger has passed.

**Athrepsia.**—The onset of gastro-enteritis is not always so sudden as in the preceding forms. The digestive troubles (vomiting and diarrhoea) insensibly affect the general condition. The nutrition of these children has been defective since birth, and instead of developing normally, they waste away. The skin wrinkles, the cheeks get hollow, the face becomes senile, and the body does not develop. The little patient may, however, live for two or three months, although he weighs less than at birth. The temperature may be subnormal or raised. The child flickers out like a candle, being carried off by latent pulmonary complications or by convulsions. It is to this chronic gastro-intestinal cachexia of the new-born that Parrot has given the name of “athrepsia.”

**Chronic Dyspepsia of Nurslings.**—Chronic digestive troubles are less evident in older nurslings, because they only affect the general condition after some considerable time. This chronic dyspepsia is seen especially in children overfed with milk that is too rich in casein, or in those who have been prematurely weaned, or fed after weaning with too coarse foods. These children are too fat; their abdomens are too prominent, and may be soft (large, flabby belly) or resistant. The stools are scanty, hard, whitish, dry, and chalky (Marfan). Such infants show very early the symptoms of rickets. At the time of weaning they are especially liable to acute enteritis, and later their flabby belly favours hernia and visceral ptoses. If these children die, the lesions of gastro-enteritis, with lengthening of the intestine, are found post mortem.

**Pathogenesis.**—Acute or chronic digestive troubles in infants always come from improper feeding. The digestive canal of the new-born can only digest the mother's milk in a perfect manner. The breast should be given at most every two hours for very young infants, and every three hours from the third to the fourth month, so that the next meal should not encroach on the digestion of the preceding one. Under these conditions (the child being breast-fed), digestive troubles are exceptional. They may supervene, however, during dentition, upon sudden changes of temperature, or from illness of the wet-nurse, but they are slight and transient. It is in bottle-fed children that digestive troubles, and especially infantile cholera, arise in the great majority of cases.

The weekly returns in Paris show clearly the cause of this disease. We find that the infantile mortality from gastro-enteritis, which is very low during the cold months, reaches during the heat of summer its maximum,

which is then ten times the average. Acute gastro-enteritis in infants is mostly due to the multiplication of the microbes of fermentation in the milk and to the poisons which they excrete.

In very young children, even in perfect health, one bottle of fermented milk may produce an attack of enteritis; but older children can offer greater resistance, and the disease is not severe unless they are already suffering from chronic dyspepsia. In dyspeptic infants the least alimentary excess in summer may be fatal, and here, as in the new-born, the ferments poured into the digestive canal are the cause of the trouble.

**Bacteriology.**—The *Bacillus coli* is found in the stools in great numbers, sometimes in a state of pure culture and of exalted virulence. The *Bacillus coli* in infantile cholera does not belong to a special kind, and though the biochemical characters vary in each case, their agglutination by the serum does not allow of differentiation from other coli bacilli (Widal, Nobécourt). The chromogenic bacillus of Lesage seems to be a variety of the coli bacillus, gifted with the power of secreting a green pigment. It is, however, not constant in green diarrhoea of infants.

Certain microbes are often found associated with the coli bacillus, and play their part. The chromophil bacillus of Escherich, the streptococcus (Marfan and Marot), the pyocyaneus (Nobécourt), and the proteus (Ardouin), are amongst the number. Nobécourt has insisted on the gravity of this combined infection.

**Pathological Anatomy.**—Histology (Marfan and Bernard) explains the process of the disease. In the normal condition the microbes are only found on the surface of the intestine; in acute gastro-enteritis they penetrate into the lumen of the glands, and invade the deep layers, whence they can enter the organism. The distant lesions are explained by the general infection and intoxication, so that pulmonary congestion and broncho-pneumonia due to the coli bacillus, meningitis due to the same microbe, phlebitis of the abdominal veins, thrombosis of the cranial sinuses, and degeneration of the liver and kidneys, may be found on post-mortem examination.

**Prophylaxis.**—Careful breast-feeding insures for the child almost certain immunity from gastro-enteritis. It is, therefore, the duty of the physician to induce every mother to nurse her own offspring. Absence or insufficiency of milk is exceptional, and though the secretion of the milk must be waited for, a delay of forty-eight hours does not harm the infant. If the mother is unable to suckle, a wet-nurse must be engaged. It is only as a last resort that the bottle should be made use of, and then great precautions must be taken to avoid acute gastro-enteritis. In order to diminish the risk of gastro-enteritis in bottle-fed infants, rigorous asepsis must be observed in the administration of food. The greatest cleanliness must be



insisted on with regard to the bottle and to the nipple, and when the bottle is finished, no milk must be permitted to remain in it.

Which milk should be used? A brand of milk, sterilized in the autoclave at  $120^{\circ}\text{C}$ ., and preserved in sealed bottles, is on the market. If care is taken to open the bottle only at the time of pouring out the milk into the feeder, the infant is certain to get a drink free from microbes. Unfortunately, sterilization at high temperatures increases the drawbacks of cow's milk, which is naturally thicker than human milk. It is richer in casein and in salts, while its clot is firmer and more resistant to the digestive juices. Moreover, cow's milk, such as the factories produce, sterilized at high temperatures, is only suitable for strong babies of several months old.

To remedy these drawbacks, some firms prepare, under the name of "maternal" or "humanized" milk, a milk which, by centrifugalization, partial coagulation, sweetening, and skimming, is brought down to a composition something like mother's milk, and is then sterilized at  $120^{\circ}\text{C}$ . The drawback of all methods of sterilization lies in the partial change of the lactose into caramel, which gives a brownish colour and a strange taste to the milk.

This drawback disappears with the economical method of sterilization invented by Soxhlet and popularized by Budin. The milk is divided in as many bottles as may be required during the day or half-day. The bottles are then plunged for twenty minutes into a water-bath of boiling water. As milk only boils at  $101^{\circ}\text{C}$ ., this operation does not cause it to boil, and its composition is consequently not changed. On the other hand, all the pathological germs are killed. When it is desired to use a bottle, all that is necessary is to substitute a nipple for the rubber disc, and a bottle of food is thus obtained without decanting the milk.

For very young infants indeed, cow's milk may be diluted with a third to a fourth part of water before sterilizing the mixture, and this precaution is a safeguard against chronic dyspepsia (dyspepsia of pure cow's milk, Marfan).

The Soxhlet-Budin method is the best when we are certain of receiving fresh milk from a good dairy several times a day. In large towns these conditions are difficult to fulfil, and in spite of Pasteurization, which to-day is universally adopted in the milk industry, we may receive milk in which microbes have commenced to grow. The passage through the water-bath will certainly kill the microbes, but it will not destroy the toxins already poured into the milk, and gastro-enteritis will not be avoided. In large towns milk sterilized in the laboratory is to be recommended in summer, but humanized milk is to be preferred in the case of very young infants.

The use of milk as the sole food of the child may, without inconvenience, be continued until the age of one year. The weaning should be gradual,

and soups, milk, panadas, broths, or vegetable purées, are the first kinds of food to be given to an infant. Infants ought not to be weaned during very hot weather.

**Treatment.**—Cases of chronic dyspepsia in childhood usually recover on a return to rational feeding, but in acute enteritis treatment is more active. It is first of all necessary to cleanse the digestive canal of its toxic contents by a small dose of calomel, and next to withhold fermentable food. The child should be fed with boiled water, given as required. Mustard baths, recommended by Trousseau, injections of lukewarm water, and stimulation by rubbing, may be employed with advantage. Subcutaneous injections of artificial serum in doses of 50 to 100 grammes, repeated if need be several times a day, are a resource which sometimes leads to recovery even in desperate cases.

### III. APPENDICITIS—APPENDICULAR PERITONITIS—APPENDICULAR INTOXICATION—APPENDICÆMIA.

During the last few years I have devoted ten clinical lectures to appendicitis, in addition to my numerous contributions to the *Académie de Médecine*. These data I shall use in the present section.

**Anatomy.**—The appendix resembles an earthworm suspended from the cæcum (appendix vermiformis). In the foetus it occupies the apex of the ampulla of the cæcum, but in the adult its situation changes; as the ampulla is much dilated below, the appendix is attached higher up to the postero-internal part of the cæcum, an inch above the ileo-cæcal valve. According to an American surgeon (McBurney), this point of attachment corresponds on the abdomen with the centre of a line drawn from the antero-superior iliac spine to the umbilicus, and the pain in appendicitis is generally situated at this spot. The appendix is about 4 inches in length and  $\frac{1}{2}$  inch in breadth. It is often held in position, in the middle half of the right iliac fossa, by a fold of the peritoneum (meso-appendix), but the situation and direction of the appendix vary exceedingly. In the **descending** type the appendix crosses the psoas, and dips into the pelvis, a situation that explains the topography of the prerectal abscess, which tends to open into the rectum, vagina, or bladder. In the **ascending** and **posterior** type, which is frequent, the appendix runs upwards along the posterior face of the cæcum and colon, a situation which explains the topography of the retro-cæcal abscess in the iliac fossa and of abscesses situated high up behind the colon. This means that the starting-point of the appendicular lesions (with or without adhesions, which limit these lesions) may be below, above, internal, or external to the cæcum.

The appendix is sometimes provided at its opening into the cæcum with a valve, called Gerlach's valve, which prevents the entrance of fæcal matter into the appendicular canal. Though Clado and Lafforgue consider this valve exceedingly rare, I have been able to find it frequently.

The appendix has the same structure as the cæcum, and is composed of four layers: an external peritoneal coat; a muscular coat, with longitudinal and circular fibres; a submucous coat, composed of connective tissue and almost lacking in elastic fibres; and a mucous tunica, with cylindrical epithelium, adenoid stroma, closed follicles, and tubular glands. It must be noticed that the appendix grows smaller towards its tip,



where the muscular fibres and glands gradually disappear, and the cellular layer becomes more important.

The appendix is traversed throughout its entire length by a central canal, the lumen of which is very narrow and uneven, and does not measure more than from 3 to 4 millimetres in diameter.

**Discussion.**—During the last few years appendicitis has assumed an importance of the first order in medicine and surgery. Acute general or local peritonitis, sub-phrenic empyema, purulent and putrid infection of the pleura, gangrene of the lung, purulent infection of the liver, hæmorrhagic ulcerative gastritis, nephritis, perinephritic abscess, endocarditis, phlebitis, icterus and icterus gravis, albuminuria, anuria, uræmia, etc., are formidable complications due to toxi-infections of appendicular origin, so that it is customary to class them under appendicitis.

But from the outset let it be well understood that the appendix is the starting-point of these complications, which were formerly put down as typhlitis and perityphlitis.

Our knowledge of appendicitis is of fairly recent date, and until 1888 typhlitis reigned supreme. This stercoral typhlitis, as it was called, was usually ascribed to obstruction of the cæcum by fæcal matter. This fæcal obstruction caused an inflammation of the walls of the cæcum, called typhlitis, with its attendant symptoms of constipation, pain, swelling, induration in the right iliac fossa, and, in more severe cases, fever, nausea, and vomiting. This so-called typhlitis generally ended in resolution, but it might end in ulceration and perforation of the cæcum, causing perityphlitis, peritonitis, or iliac phlegmon. There was peritonitis if the lesions were seated on the anterior surface of the cæcum, and iliac phlegmon if the lesions were seated on the posterior surface, which was incorrectly believed to be lacking in peritoneum.

These ideas were held for a long time, but they were wrong, for stercoral typhlitis, in this sense, does not exist. In the first place, the posterior surface of the cæcum is not lacking in peritoneum. In 1887 Tuffier showed that the cæcum in the adult and in the fœtus is never in direct communication with the cellular tissue of the iliac fossa. The cæcum is surrounded by peritoneum, and “the hand can be passed round it in the same manner as it can be passed round the apex of the heart in the pericardium.”

On the other hand, American surgeons had begun to perform early laparotomy for symptoms formerly attributed to typhlitis, and they saw clearly that these symptoms had their origin, **not in the cæcum**, but in the appendix. The same observation was made hundreds of times by surgeons in England, Switzerland, Germany, and France, so that the precise idea of appendicitis was gradually substituted for the mistaken description of the old typhlitis. These ideas, which some authors were loath to admit, were sanctioned at the Surgical Congress of 1895, and, in the discussions at the Société de Chirurgie, Routier took care to state that in a large number of personal cases the appendix was the seat of the mischief, and not the cæcum. I have witnessed operations in more than 200 cases of appendicitis, and I am convinced that the various troubles formerly put down to typhlitis must always be put down to the appendix, and never to the cæcum.

This does not mean that the word “typhlitis” is to be removed from our nosology, but a clear understanding is necessary. The ulcerations of typhoid fever and of tubercular disease frequently attack the cæcum, but these specific lesions are beside the question which at present occupies our attention. Tuberculosis may be localized in the cæcum in a primary or chronic form, which is curable by operation, but this tubercular typhlitis which simulates cancer so closely has nothing in common with the typhlitis now under discussion.

A perforation of an unknown nature might involve the cæcum, like the ileum, but, once again, all these dissimilar cases, though seated in the cæcum, have nothing in common with the question before us, and it is well established that the troubles set down to typhlitis must now be put down to appendicitis.

Finally, it will be said that in undeniable cases typhlitis with ulceration, gangrene, or suppuration of the cæcum has been found. If, however, we examine them closely, we shall see that these lesions are consecutive to appendicitis, and, as the appendix, which is primarily diseased, runs along the posterior surface of the cæcum, the cæcal and retrocæcal lesions are secondary to the appendicitis. Gambetta's case was of this nature. Appendicitis must, therefore, be substituted for typhlitis.

These restrictions being established, it is hardly necessary to state that nobody has ever thought of denying typhlitis in so far as inflammation of the mucous membrane of the cæcum is concerned. The cæcum, like the colon or the ileum, shares in the infections of the intestinal mucous membrane. Typhlitis exists, just as do enteritis and colitis. Intestinal infections, classed together under the name of enteritis or of follicular, mucomembranous, or sabulous entero-colitis, are prone to affect the mucous membrane of the cæcum. I have given them the name of **entero-typhlo-colitis**, and yet no one has ever seen typhlitis or typhlo-colitis whether it is accompanied or not by constipation, membranes, or sand, produce gangrene, perforation of the cæcum, and peritonitis.

**Pathological Anatomy—Bacteriology.**—According to the particular case, the appendix may be purple in colour, lengthened, doubled in size, indurated, or in a condition of erection. We may find suppuration, fluctuation, gangrene, perforation, calculi, twists, or kinks, strangulation by bands, etc.

Its lumen is constricted or obliterated at its origin, or at some point of its length. The canal may have recovered its permeability at the time of operation. The obstructions of the canal favour the formation of closed cavities. The closed cavity may be small, and of almost the normal size and shape of the appendicular canal. In other cases it is larger, and gives to the appendix the form of a spindle, club, cherry, pear, etc. The closed cavity contains a small amount of muco-purulent, fæcal, or blood-stained fluid. It may be transformed into a pseudo-cyst, containing inoffensive fluid. The pathogenic microbes of appendicitis are the microbes of the intestinal flora, and the coli bacilli are most in evidence.

**Pathogenesis of Appendicitis.**—The pathogenesis of appendicitis has been influenced by the views held as to the causation of typhlitis. Constipation and diarrhoea have been given as causes, although it was not known which of these conditions was the more effective. Foreign bodies, such as fragments of bone, fish-bones, egg-shells, pins, needles, prune-stones, fruit-pips, etc., have been accused.

I may say at once that fragments of bones, prune-stones, date-stones, cherry-stones, etc., though quite capable of injuring the cæcum, are absolutely incapable of entering the appendicular canal. At most only very small bodies indeed, such as grape-stones, could do so, and even then, since appendicular concretions have been carefully studied, it has been found that these concretions, which may take the form of coffee-beans, date-stones, grains of corn, beans, and pips, are simply calculi formed *in situ*. Their origin and structure are now well known.



We must abandon, then, the old theory of a foreign body entering the appendix, injuring its walls, and causing peritonitis by perforation. I have tried, by collecting the anatomical, bacteriological, and experimental proofs, to simplify the pathogenesis of appendicitis, and find a formula applicable to every case. In my opinion the theory of appendicitis may be summed up as follows : **Appendicitis results from the transformation of a part of the appendicular canal into a closed cavity, which becomes a focus of infection and intoxication, due to the increased virulence of the imprisoned microbes.** Let us analyze first the different ways in which the appendicular canal may be converted into a closed cavity.

**Calculous Appendicitis.**—The presence of a calculus in the appendicular canal has given to this variety the name of **calculous appendicitis**. The calculus is found, if the infectious process has not destroyed it. If the appendix has become gangrenous, the calculus may pass into the peritoneum, where it is found at the time of operation or during the subsequent dressings. These calculi have been described by Rochaz, who gives a description and illustrations of sixty-five appendicular calculi. They are usually elongated and fusiform or cylindrical. As a rule, one calculus is found, but as many as three or four may be present. They then have several facets. The colour is usually brownish, but their consistency varies in nature—friable or dense and of stony hardness, while every intermediate degree may be seen. These calculi are formed of different elements. Berlioz has made a most minute analysis of several calculi, and the results of his researches agree absolutely with those obtained by other chemists (Volz, Butler, Pelet).

In these calculi we find brownish fæcal matter, soluble in ether and mineral salts, principally phosphates and carbonates of lime. We sometimes find salts of ammonia, traces of chlorides, sulphates, and, very rarely, cholesterin (Walt). The various organic and mineral elements are cemented by mucus from the glands in the appendix.

It will now be understood why the appendicular concretions are sometimes soft and friable, sometimes hard and, as it were, calcified. If the concretion is rich in organic matter, it is soft, or at least it is easily softened at the time of infection ; if the concretion is rich in calcareous salts, it may assume a stony consistency, like a true calculus. It is easy to find these concretions in the process of formation. It is only necessary to make a section of them to see that they are usually **stratified**. Around one or several nuclei there are eccentric layers, the stratification of which proves that the calculi grow slowly in the appendicular canal by the addition of organic and mineral layers.

By studying this process, and comparing the slow formation of appen-

dicular calculi with the analogous formation of biliary calculi, I was led to propose the name **appendicular lithiasis**, which seems to me to be very similar to urinary and biliary lithiasis.

What becomes, then, of the old theory (Talamon) that the calculi reach the appendix after being previously formed in the cæcum? According to Talamon, it is in the cæcum that the scybala are rolled and mashed.—"they are rounded there like pellets under the finger"—and after this pill-making process they pass from the cæcum into the appendix. This theory has been upset by the arguments of Rochaz: "Talamon supports his theory by the perfectly spherical form of the calculi; but we only met with this spherical form three times in sixty-five cases, and the usual shape of the calculi is cylindrical. How are the long stercoral sausages, filling the appendix from one end to the other, to be explained by Talamon's pellets? How could large concretions penetrate by an opening which at most has a diameter of only 5 millimetres, and which is, besides, more or less completely closed by a valve? On the contrary, the disposition of the calculi in concentric layers indicates a slow formation, which could only take place in a recess separated from the intestinal canal, and not in the cæcum, where the calculi would be carried away, after a short time, by the flow of matter."

I have nothing to add to the excellent reasoning of Rochaz. It is, indeed, clear that the so-called calculous appendicitis is associated with a process of lithiasis, which may be compared in some measure to biliary and urinary lithiasis.

**Non-Calculous Appendicitis.**—Appendicitis is not always of calculous origin. In most cases it runs its course without the smallest concretion being found in the canal. It is here a question of an infection of the mucous membrane, which American surgeons have called **catarrhal appendicitis**, for want of a better name. I propose to call it **obliterating appendicitis**. In such a case, perhaps from the swelling of the infected mucous membrane, the appendicular canal is obliterated at its orifice, or at some point in its length, exactly as the biliary canals are obliterated in so-called catarrhal icterus, or as the Eustachian tube is obliterated in a case of acute otitis. This acute process terminates in the formation of one or several subjacent cavities in the appendix, and causes all the complications.

Several causes of obliteration are sometimes found in the same appendix, and appendicitis may be at the same time calculous and obliterating. The calculi may be free in the closed cavity.

These non-calculous cases of appendicitis are just as serious as the calculous forms, and the symptoms and the complications are similar. They may likewise cause appendicular and peritoneal troubles, and give rise to



gangrene, or to perforation of the appendix. We must therefore reject Talamon's theory that a calculus (which frequently never existed) played an important rôle in the compression of the appendicular vessels.

I have just spoken of the acute obliterative process, but there is also a chronic form, which brings about fibroid constriction and obliteration of the appendicular canal at some point of its length (a stenosis which may be compared with stricture of the urethra). This process may be chronic from the outset, or may follow attacks of acute appendicitis, just as endocarditis is sometimes followed by mitral constriction. In a case quoted by Achard, where the lesions had caused perforation of the appendix and abscesses of the liver, the infective focus in the appendix was due to obliteration of the orifice of the canal by fibroid tissue. There was no trace of a calculus. Rendu has published a case of purulent appendicitis caused, not by calculi, but "by a sort of fibrous constriction, which separated the diseased appendix from the healthy cæcum." It was easy to see that the communication between the cæcum and the appendix was completely obliterated.

**Appendicitis caused by kinks and strangulation.**—In some cases the appendicitis is neither calculous nor obliterating, but results from kinking of the appendix, from strangulation by bands or adhesions, or by twisting of the appendix around its mesentery. In such cases the portion of the canal below the kink or strangulation is converted into a closed cavity.

The appendicitis thus caused is as serious as that due to calculus or obliteration, and the toxi-infection in the appendicular focus causes similar symptoms and results.

**The Closed Cavity.**—From the various processes just described, it follows that the canal may be obstructed either at its cæcal orifice or in its length by a calculus, by inflammatory swelling of the walls, by twisting, by kinking, or by strangulation of the appendix. The obliteration may be temporary or persistent. It matters little whether the obliteration is due to a calculus, strangulation of the appendix, swelling of the infected walls, or kinking of the tube. The essential fact is that **the portion of the appendicular canal below the obliteration is converted into a closed cavity.** The microbes, which in their normal state were inoffensive (as are all the microbes of the intestine **when free**), are now imprisoned, and therefore able to increase their virulence, as shown by Klecki's experiments, and to give rise to acute toxi-infection. From this moment appendicitis is present, and **if the microbes imprisoned in the appendicular focus are endowed with sufficient virulence**, abscess, perforation, or gangrene of the appendix may result, or else the microbes may pass through the walls of the appendix and reach the peritoneum, **without these walls**

showing the slightest perforation ; or, again, the toxines and microbes may reach the veins and infect the whole system. The patient is now liable to multiple complications, which are only too often dangerous.

The formation of a closed cavity is not always followed by complications, for they depend on the virulence of the imprisoned microbes, and on the toxicity of their products. This virulence may be insignificant, or may be annihilated by the phagocytes, in which case the appendicular lesions do not run their full course. A chronic process may obliterate the whole canal, causing a spontaneous and radical cure, which protects the patient from further trouble.

On the other hand, the infection of the walls may continue its course even after the initial obliteration has disappeared. This explains why, at the time of operation, we may find a canal which has recovered its permeability, the closed cavity being thus destroyed, after having been the *primum movens* of the infection, which has continued its course. This view of appendicitis due to a closed cavity was suggested to me by the beautiful experiments of Klecki.

**Experimental Research.**—In 1889 Clado presented to the Surgical Congress some remarkable clinical and experimental work on hernial infection. He found that the microbes from the strangulated intestine passed into the sac, without any perforation of the gut, as early as the day following strangulation. He followed the migration of the microbes through the unperforated coats of the bowel, and proved that the peritoneal cavity could, in its turn, be invaded. Finally, he noted the possibility of general infection.

Benneckén, Oker-Blom, and many others, have since repeated these experiments, and have proved that the coli bacillus, enclosed in a strangulated or invaginated coil of gut, may pass into the peritoneum either by penetration through the coats or by way of the lymphatics, and thus cause peritoneal infection.

The most important work on this subject, however, is that of Klecki, who, with strict aseptic precautions, occluded an intestinal coil in dogs by means of caoutchouc rings. After twenty-four to forty-eight hours the dogs were killed. The strangulated coil was not perforated, and yet had caused peritonitis. The results of these experiments are : the intestinal coil, converted experimentally into a closed cavity, contains swarms of the microbes usually found in the bowel, but their virulence is much increased. The microbes, in their new pathological state, can traverse the non-perforated intestinal wall, and cause peritonitis. The virulence of the microbes in the ligatured coil of gut is greater than in the peritoneum. "It is therefore not in the peritoneum that the solution of the question must be sought. The coli bacillus and the other microbes which give rise



to polyinfection reach the peritoneum through the pathological coil, in which they have already undergone biological changes, causing an increase in their own virulence, and also in that of their toxines."

As these experimental data appeared to me absolutely applicable to the pathogenesis of appendicitis, I proposed the theory of the closed cavity. Roger and Josué ligatured the appendix in a rabbit, taking care to spare the vessels. They killed the animal some time afterwards, and found that the part below the ligature was converted into a purulent cavity. They therefore concluded that it suffices to imprison the microbes in the appendix, "in order to transform the inoffensive microbes of the intestine into pathogenic agents."

De Rouville also has caused appendicitis experimentally in the rabbit by ligaturing the ileo-cæcal appendix at its base. He was thus able to reproduce the lesions caused by the closed cavity. His conclusions are as follows: "The first experiment showed the only too frequent course of appendicitis in man, ending in gangrene, perforation of the appendix, peri-appendicular abscess, and general peritonitis. The perforation was produced apart from any calculus or from any operative injury to the vessels. The second experiment showed that, even if infective lesions may, in certain cases, be for a long time limited to the appendix, the latter is none the less the focus of an extremely violent infection, and the possible starting-point of formidable complications, which nothing but early surgical intervention can avoid."

"These two cases testify in the same manner as the one recently reported by Roger and Josué, and, like the latter, support the theory of the closed cavity maintained by Dieulafoy."\*

Another experiment: De Rouville introduced into the appendix of a rabbit a fine stem of laminaria. Appendicitis did not appear until the swollen laminaria came into intimate contact with the walls blocking the opening, and converting the lower portion of the appendicular canal into a

\* The new theory of appendicitis, as maintained by myself, differs in many respects from the ideas enunciated by Talamon. According to Talamon, the calculus "enters the appendix suddenly by an untimely contraction of the cæcum, penetrates into it by rubbing, and encloses itself in the superior portion of the narrow canal." Rochaz, as we have seen above, has disproved this theory. According to Talamon, one of the consequences of the calculus thus engaged is "compression of the walls of the appendix and stoppage of the circulation of the vessels contained in the walls"; then "the inoffensive microbes, which are powerless against the healthy cells, triumph without difficulty over these cells when they are deprived of their blood-supply." The experiments of Rouville also disprove this hypothesis. Accordingly, the various experiments, and the numerous cases of appendicitis without calculus, condemn Talamon's theory, which, however, has one happy expression—"the closed vase"; but this expression was still-born, lost in the midst of inexact theories (see Delbet, *Arch. Génér. de Médecine*, 1897, p. 321).

closed cavity. Other foreign bodies, such as small grains of lead or pieces of glass, introduced into the appendix do not cause appendicitis, because they do not gradually swell up, causing obliteration of the canal, and converting it into a closed cavity. These experiments prove that the arrest of foreign bodies or of appendicular products does not suffice to provoke appendicitis. Appendicitis is only produced when the canal is **completely obstructed** at some spot.

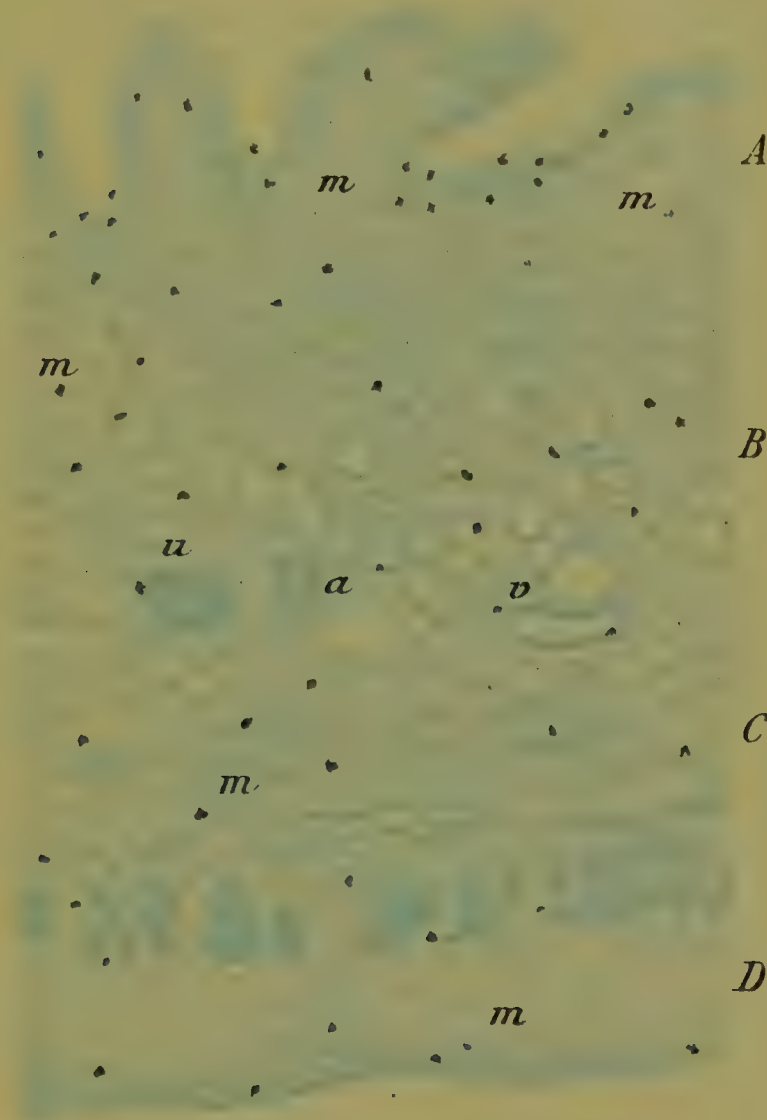
**Migration of Microbes.**—If it is in the closed cavity that the virulence of the microbes is increased, we must prove that their migration takes place through the walls of this closed cavity. In conjunction with Apert and Caussade, I have verified this point. We made use of an appendix which had been removed. The histological lesions and the bacteriological features are noted in Plate VI. The prominent features are as follows: **above** the obliteration, the wall has preserved its normal structure; no microbes are found in it: **on a level** with the obliteration, the lumen of the canal has completely disappeared, and the wall is largely replaced by fibrous tissue. Microbes are few in number: **below** the obliteration, in the closed cavity, the deep part of the mucous membrane still exists, but its superficial part is ulcerated. The base of the glands only is seen. The subjacent layer forms two-thirds of the thickness of the wall. It has a lymphoid structure, and is traversed by small veins, congested with blood, and by a few thickened arterioles. The lymphoid tissue invades the muscular layers, and separates the bundles, especially those of the circular layer. The continuity of the longitudinal layer is better preserved. The peritoneum is very much thickened, and the subperitoneal layer is traversed by numerous blood-vessels. The walls of the closed cavity are traversed by **numerous colonies of microbes**, occupying the lymphoid tissue below the mucous membrane, and especially the spaces of the reticulum. These microbic masses are in part engulfed by leucocytes, and are formed by agglomeration of the microbes, as if the coli bacillus and other microbes had formed colonies *in situ*. Similar colonies are found in the portions of lymphoid tissue separating the layer of smooth muscular fibres. They can be followed into the subperitoneal layer, and are spreading to the peritoneal cavity. The pathogenesis of certain cases of appendicular peritonitis is thus explained, **although the walls of the appendix are neither gangrenous nor perforated.**

The chief fact, which by itself alone would suffice to prove the pathogenic rôle of the closed cavity, is that the colonies, though so numerous in the walls of the closed cavity, where their virulence is increased, are **absent** in the walls of the appendix above the closed cavity. The infective process is, therefore, much the same in a closed cavity of the appendix or in a strangulated coil of bowel. In both cases the increase in virulence favours



MIGRATION OF MICROBES THROUGH A SEGMENT  
OF THE APPENDIX, WHICH HAS BEEN  
TRANSFORMED INTO A CLOSED CAVITY.

*Plate VI.*



— A section of the walls of the appendix on a level with the closed cavity.

A. — THE DEEP PART OF THE ULCERATED MUCOUS MEMBRANE.

B. — THE SUBMUCOUS LAYER, VERY MUCH THICKENED, FORMING TWO-THIRDS OF THE THICKNESS OF THE WALL.

It has a lymphoid structure, and is traversed by small veins (*v*) gorged with blood (*u*), and by arterioles with thickened walls (*a*).

C. — MUSCULAR LAYER, SEPARATED BY THE LYMPHOID TISSUE.

D. — SUBPERITONEAL LAYER.

*m*, Numerous colonies of microbes (*Bacillus coli*) traversing the appendicular walls from the mucous towards the serous membrane. To facilitate the description, the microbic elements have been diagrammatically enlarged.





the migration of the microbes through the walls. My researches as to appendicitis agree absolutely with those of Klecki as to the intestinal coil.

It is now easy to understand how the infection of the walls of the appendix at the site of the closed cavity takes place. We see the pathogenesis of certain cases of appendicular peritonitis, the peritoneum being infected, even when the walls of the appendix are neither gangrenous nor perforated.

And the proof that this is the **crux of the question** is that, while the obliteration may be due to kinking, twisting of the appendix, strangulation by a band, obliteration by calculi, or swelling of the walls, the result is always the same. **The infection starts from the closed cavity, and sets up the same train of symptoms and complications.**

**Increase of Virulence in the Closed Cavity.**—The following experiment (Hartmann and Minot) helps us to comprehend the degree of virulence which the microbes may acquire in the closed cavity :

Hartmann operated upon a patient for acute appendicitis, and found, on examining the specimen, that the canal was obliterated in its middle portion, the lower portion being converted into a closed cavity. Two sets of cultures were made on agar, one from the mucus taken from the free end of the canal and the other from the mucus of the closed cavity. Twenty-four hours later numerous colonies of the *Bacillus coli* had grown on both cultures. Two tubes of broth were treated separately with the colon bacilli taken from each of these cultures. On April 19 fifteen drops of these two broths were injected under the skin of the flank of two guinea-pigs. The guinea-pig inoculated with the broth containing the bacillus taken from the free ends of the appendicular canal remained in perfect health, whilst the fifteen drops of the broth containing the bacillus from the closed cavity caused considerable cedema and loss of flesh in the inoculated guinea-pig. On April 24 the experiments were repeated, the cultures being on this occasion four days old. A guinea-pig inoculated with twenty drops of the first broth suffered no ill-effects, whereas another guinea-pig, inoculated with twenty drops of the second broth, died in thirty-six hours, with an enormous phlegmon of the wall, and pus in the pleura, pericardium, and peritoneum.

I have repeated the experiments of Hartmann and Minot in my own laboratory with Caussade, and have obtained identical results.

For our experiments we took an acute obliterating appendicitis in which the closed cavity was absolutely shut off. We treated two broths, the one (No. 1) with liquid taken from the free part of the canal, the other (No. 2) with liquid taken from the closed cavity. These broths were placed in the oven. They contained quantities of colon bacilli. We chose twelve guinea-pigs of about the same weight, and divided them into two series of six each. The six guinea-pigs of each series were given, by injection under the skin of the abdomen, fifteen drops of No. 1 broth, in the one case, and in the other five drops of No. 2 broth. The twelve guinea-pigs were not, however, inoculated at the same time. They were inoculated two at a time on succeeding days, the last cultures being seven days old. The guinea-pigs inoculated with No. 1 broth showed at the point of inoculation a small indurated nodule, which was readily absorbed. They all remained in perfect health, whilst the guinea-pigs similarly inoculated with No. 2 broth showed gangrenous abscesses, with pus containing the *Bacillus coli*, and all died from the infection. These experiments are therefore conclusive.

**Manufacture of Toxines in the Closed Cavity.**—The preceding experiments, though conclusive from the point of view of infection, gave no precise information as to the **toxicity** of the products elaborated in the closed cavity.

As I was anxious to make some experiments concerning the **toxicity**, Caussade and I filtered the culture broths, and made inoculations with the filtered liquid, which contained toxines, but not bacilli. Six guinea-pigs were inoculated, three with the filtrate of No. 1 broth and three with the filtrate of No. 2 broth. Each guinea-pig received an injection of twenty drops of filtered broth: the two guinea-pigs inoculated on Monday received cultures three days old, those inoculated on Tuesday cultures four days old, and those inoculated on Wednesday cultures five days old. In no case did the inoculation produce either abscess or induration.

The three guinea-pigs inoculated with the filtrate of broth No. 1 remained alive and well, but of the three guinea-pigs inoculated with the filtrate of broth No. 2 two died on the fifth and sixth days of the inoculation. They did not die of infection, but of intoxication. The closed cavity is a focus of infection and of intoxication, and the conclusion is that I was correct in calling appendicitis a **toxi-infectious** disease. This explains a series of complications which will be studied under the **toxicity** of appendicitis.

**Ætiology.**—As we have discussed fully the rôle of the closed cavity in appendicitis, we can pass on to the ætiology. We must first consider the question of **heredity**. Roux, of Lausanne, rightly maintained that appendicitis is frequently hereditary. My own experience has shown me that appendicitis is often seen in members of the same family, both in collateral branches and in descendants. On a closer study of the question it has seemed to me that heredity was especially noticeable in gouty families, and in those suffering from urinary and biliary lithiasis, so that I would include many cases of appendicular lithiasis under the gouty diathesis. To quote examples:

One of my assistants sometimes has renal colic. His father was gouty and his brother died of appendicitis. A few years ago one of my colleagues died of diabetes and his daughter of appendicitis. For some time past I have been treating a family in which gout, obesity, and diabetes are present. In this family I know of three cases of appendicitis, one of which, in a child of ten years of age, was fatal. One of my best friends had attacks of renal colic. His son was attacked by appendicitis; he was operated on by Bouilly and recovered. I know a lady who, for the past fifteen years, has been subject to hepatic colic, and, at my request, her son was operated on by Routier for calculous appendicitis. At Trouville I saw, with Collet, a child of five years of age taken ill with appendicitis, who was operated on by Pozzi. The girl's mother had attacks of hepatic colic. In 1895 I was called to a lady of seventy-two, who was suddenly taken ill with calculous appendicitis. Routier operated at my request. Two months later it was the fortune of her granddaughter to be operated upon, and she also had calculous appendicitis.

Appendicular lithiasis is, therefore, a family and hereditary disease. It is sometimes of arthritic origin, and it must therefore rank with biliary and urinary lithiasis. I might even say that of the three kinds of lithiasis, the



appendicular form generally appears first, since **it is common in children**. It is the more to be feared because the complications consequent on renal or biliary calculi are not to be compared either in gravity or frequency with those resulting from appendicular lithiasis.

In other cases the heredity of appendicitis (calculous or non-calculous) is manifest apart from any gouty diathesis.

Since I drew attention to this question of **hereditary and family** appendicitis,\* cases have multiplied. Faisans communicated to the Société Médicale des Hôpitaux six cases of family appendicitis, in which two, three, and four persons of the same family were taken ill with appendicitis. At the same meeting Rendu communicated similar cases. La Société de Chirurgie has also found identical results (Brun, Berger, Tuffier, Jalaguier, Quénu).

It is possible that the malformations of the appendix may also be hereditary (Talamon, Pozzi), and ready to favour the formation of a closed cavity.

**Determining Causes.**—Appendicitis may occur during pregnancy. Metchnikoff has blamed intestinal worms and the trichocephalus. Some authors have assigned a part to entero-colitis, as if appendicitis were the result of entero-colitis, but this opinion seems to me erroneous. I shall discuss this question in Section IV. Influenza has been blamed (Faisans), and also measles, angina, etc. I cannot agree with this opinion. It is more correct to say that the primary cause of appendicitis often escapes our notice.

**Onset of Appendicitis.**—Every intermediate stage is seen between an insidious onset, which is almost afebrile and painless, and a sudden onset, with much fever and pain. As a general rule, prodromata rarely occur, and the disease usually starts suddenly in the midst of excellent health, so that on the previous day there is no suspicion of trouble.

Mild appendicitis runs the following course : The patient has a feeling of pain and fullness in the belly, especially in the right iliac fossa. Nausea or vomiting is generally present, but the fever is insignificant, and the patient fancies that he is suffering from indigestion. He takes a purgative and an enema, because his intestinal functions are not normal. A physician is called in. The patient speaks of constipation and nausea, and calls attention to the pain in the right iliac fossa. "Quite so," says the doctor. "I find some induration and tension." And if the physician in question still believe in typhlitis, he will diagnose stercoral typhlitis or cæcal obstruction, and prescribe "a purgative," or even leeches.

In other cases the pain rapidly becomes very severe in the right iliac

\* Dieulafoy, "Cours de Pathologie Interne de la Faculté de Paris" (*Semestre d'Hiver*, Novembre, 1895).

fossa, though it may radiate in various directions. The fever is about  $103^{\circ}\text{F}$ . or more. Vomiting, especially of bile, occurs. A doctor is called in, and if he is imbued with the old and false ideas which I have tried to fight against, he declares, after having examined his patient, that the case is one of "appendicular colic." By this he means that a calculus coming from the cæcum has got into the appendicular canal, and provokes pain, accompanied by nausea and vomiting.

I have spoken strongly about this fatal theory. Rochaz has proved that the calculi do not enter the narrow lumen of the appendix. The pain is not caused by the passage of the calculus, and "appendicular colic" in this sense does not exist. As this term perpetuates an error, it must be abandoned. It is as incorrect to speak of appendicular colic as it is erroneous to admit the old conception of typhlitis. To what, then, is the acute pain at the onset of appendicitis due? There can as yet be no question of perforation or of peritonitis. The pain, as well as abdominal distension, muscular resistance, nausea, constipation, and vomiting, result from the formation of the closed cavity, and from the toxi-infection which follows it. The course of events in the appendix is similar to that in the middle ear, which is converted into a closed cavity when the Eustachian tube is blocked. As soon as the tube is obliterated, the microbes which have entered the middle ear increase in virulence, and acute otitis comes on, with violent pains, which radiate in various directions. Otitis often terminates without perforation of the membrane or other complications if the Eustachian tube becomes permeable once more, either by natural or artificial means. If the obstacle is removed, the free outflow of the infective matter puts an end to the dangers of the closed cavity. If the obstruction persists, the microbes imprisoned in the cavity may increase in virulence to such a degree that complications may arise. The perforation of the membrane is analogous to that of the appendix. Phlebitis of the lateral sinus and jugular vein is analogous to appendicular phlebitis. Meningitis is analogous to appendicular peritonitis, while the abscesses in the cerebrum and cerebellum, and even remote abscesses in the opposite cerebral hemisphere or elsewhere, are analogous to the remote abscesses in the liver or spleen, which are secondary to appendicitis.

The complications are therefore absolutely analogous in acute otitis and in appendicitis. The closed cavity and the appendicular infection cause acute pains in the abdomen, which were formerly mistaken for colic caused by the passage of the calculi. They also cause the attacks of nausea and vomiting, as in strangulated hernia. It is the closed cavity and the appendicular toxi-infection which cause the early collapse, and add to the gravity of the disease, whilst they are also the origin of diffuse or encysted peritonitis, even when the appendix has not been perforated. Again, the



closed cavity and the appendicular infection are the points from which the microbes spread to the liver, spleen, and lungs, and these same two factors cause the degeneration of the cells of the liver (urobilinuria, icterus, icterus gravis) and kidney (albuminuria, anuria, uræmia), as well as the erosions in the stomach, with copious hæmatemesis, etc. Thus, from the very outset, and **prior even to the appearance of the peritoneal lesions**, the appendicular toxi-infection reveals its presence by some of the troubles just enumerated.

It is the **pain** which is our most certain guide, for, sharp or slight, it always has certain characteristics in appendicitis.

The pain rarely reaches its maximum at first, and thus differs from the sudden and terrible pain (peritoneal dagger-thrust) due to perforation of a duodenal or gastric ulcer. The pain in appendicitis occupies the middle of a line drawn from the umbilicus to the anterior superior iliac spine on the right side (McBurney's point). This region must be examined with the greatest care, and even when the pains are slight, or preceded by pains in the epigastric region or elsewhere, or even when they radiate to other points of the abdomen, it is possible by means of careful palpation to provoke or increase the pain in the appendicular area or in its vicinity. The underlying muscle is more rigid than at other points. This **muscular resistance** is an excellent sign, and at the same spot we may find another sign to which I attribute great value—viz., **hyperæsthesia** of the skin over the appendix. If the skin is stroked gently with the pulp of the finger, hyperæsthesia, which exists nowhere else over the abdomen, will be found, and this symptom is at times accompanied by reflex cramps in the underlying muscles.

**Summary.**—Pain at McBurney's point, muscular resistance, hyperæsthesia, nausea, vomiting, and fever, usually mark the onset of appendicitis.

**Course of Appendicitis.**—Will a slight or violent attack of appendicitis that is of one or two days' duration, remain limited to the appendix, or will it be followed by peritoneal infection and toxic complications? Let us examine these various eventualities. Sometimes a sharp or slight attack of appendicitis ends favourably, and after a few days of pain, with more or less frequent attacks of vomiting, the fever decreases, constipation ceases, and the right iliac fossa is less sensitive and distended, while the muscles become relaxed, and the patient recovers. What has happened? Either the microbes causing the trouble were endowed with but feeble resistance, or the enemy has been vanquished by phagocytosis; or else the obstruction of the appendix has given way rapidly, the closed cavity no longer exists, free circulation is re-established in the canal, and the appendicitis is cured, as acute otitis is cured when the Eustachian tube recovers its permeability in time.

In other cases the attack is more severe, and the pain in the right iliac fossa is more intense and persistent. The distension of the abdomen is more general, and the vomiting more frequent; but yet the condition gradually becomes normal. Convalescence, however, is slow, and the patient shows traces of the appendicitis in the iliac fossa for a long time. He suffers from constipation, and is afraid to shoot or ride, and if he decides later upon an operation during the quiescent stage, we find adhesions, peri-appendicular peritonitis, an enlarged appendix, or at times a small encysted abscess, or even an imminent perforation of the appendix. All surgeons have seen cases of this kind. I have noticed them myself several times. I examined the appendix of a boy upon whom Routier operated at my request in the Necker Hospital. He went to work, although he had a peri-appendicular abscess resulting from acute appendicitis.

We often see cases in which appendicitis ends very quickly in gangrene. The illness is ushered in by fever and pain. Ice is placed on the painful region, and injections of morphia are given, while hope is entertained of being able to limit the disease. In two or three days the appendix becomes gangrenous, and the patient dies from toxi-infection, either because he has not undergone an operation, or because the operation has been performed too late. I could cite many cases of this kind.

In other cases appendicitis seems to run a favourable course, and is allowed to "get cold." The partisans of "cooling" congratulate themselves, and it is sometimes whilst they are quietly contemplating this "cooling" that the catastrophe occurs. The recent communication of Legueu to the Société de Chirurgie proves this point:

Legueu showed the perforated appendix of a child of five years of age who succumbed suddenly whilst the disease was "cooling." He was treated medically in a service where early surgical intervention has been systematically banished. This "cooling" had been going on for five days, and his general condition was excellent when he was taken suddenly ill with vomiting; the pulse-rate rose, the stomach became distended, and the extremities grew cold. In a few hours he was dead, before there was time to operate. The post-mortem examination showed the perforated appendix lying in a deep-seated abscess, which was separated from the abdominal wall by several coils of bowel. The abscess had burst into the peritoneal cavity, causing acute peritonitis. It is probable, adds Legueu, as his sole commentary, that an operation, as soon as the diagnosis was made, would have saved the patient.

Hartmann has described an analogous case:

A woman suffering from acute appendicitis was placed under my care. The symptoms were improving, and it was thought that "cooling" might be safely awaited. After some days passed, the situation suddenly became so bad that surgical intervention was impossible. The post-mortem examination showed recent general peritonitis, and a large retrocæcal abscess containing the gangrenous and perforated appendix.

I must now mention the two chief forms of appendicitis—namely, appendicitis accompanied by peritonitis and toxic appendicitis.



**Appendicular Peritonitis.**—When appendicitis commences, we can never be sure of its course. The disease may remain limited to the appendix. In this case the peritoneum is not affected, and the patient recovers from his attack. The appendix, however, remains normal, but may become sooner or later the origin of fresh mischief. In other cases the process is not limited to the appendix, and the peritoneum is attacked, but only to a very slight extent. The peritonitis is then strictly limited. Adhesions are then formed between the appendix and the neighbouring parts, and may serve to limit the mischief in case of fresh attacks. Finally, we see many cases in which appendicitis ends in diffuse or partial peritonitis.

Peritonitis is said to be diffuse when it spreads without showing any tendency to become encysted. The intestinal coils are sometimes sticky and dull-looking; the exudate is scanty, russet in colour, and thin. It is then almost always a question of acute septic peritonitis, which is very rapid in its course. Sometimes the peritoneal fluid is abundant, and more or less purulent, while false membranes float in the effusion, and form bands between the intestinal coils. This purulent variety is chiefly seen in cases of some duration. Peritonitis may occur very early, and appear on the second day of appendicitis; but at other times it supervenes later, during the first or second week.

Erroneous ideas have been long prevalent concerning the pathogenesis of these forms of peritonitis, and I have tried to rectify these errors. They were formerly considered to be due to perforation, and two stages were described—the appendicular stage, with symptoms of appendicitis; and the peritoneal stage, with symptoms of peritonitis. Talamon says: “In appendicitis there are generally two distinct periods—the preparatory period before perforation, which I propose to call the period of appendicular colic; and the peritoneal phase, which follows the rupture of the appendix.”

The question was simplified when thus presented, but the misfortune is that events run quite a different course.

In the first place, perforation of the appendix is not necessary for the production of peritonitis. In many cases, no doubt, the appendix is affected by suppuration, gangrene, or perforation, and the residue of the appendicular focus discharges itself into the peritoneum. Often, however, no perforation is found in the appendix. We have all seen such cases, and the peritonitis is due to the passage of microbes through the walls.

Furthermore, while there are cases in which the symptoms of peritonitis arise with the suddenness of peritonitis from perforation, there are others—and in my opinion they are the more numerous—in which it is difficult to define the exact onset of peritonitis.

It would be wrong, therefore, to suppose that the appendicular and peritoneal stages are always distinct, and to say that peritonitis is always

ushered in by a symptom-complex which allows us to foresee the danger. It is often impossible to recognize at what moment the peritoneal symptoms appear, for they are often blended with the appendicular symptoms. Pain, vomiting of food and bile, changes in the temperature and the pulse, constipation and tympanites, may all be present, whether the peritoneum is affected or not. We watch for the onset of peritonitis when it is already present; we delay when we ought to act; and the patient succumbs because we have not operated.

In many cases the peritonitis is encysted, and may then occupy the most diverse situations in the abdomen. The direction of the normal appendix prior to the disease (descending, ascending, or retrocæcal type), and old or recent adhesions due to former attacks, explain the localization of the peritonitis and its tendency to become encysted, and to give rise to ileo-inguinal, retrocæcal, prerectal, or peri-umbilical abscesses, known as iliac phlegmon and perityphlitis by older writers.

The ileo-inguinal abscess constitutes the most common variety. The pus is found in the right iliac fossa, above Poupart's ligament. It is bounded by the antero-internal surface of the cæcum, and the coils of the small intestine, and corresponds on the belly to a line situated a little above the ligament, and internal to the antero-superior iliac spine.

The prerectal abscess is much less frequent than the preceding variety. In some cases the appendix reaches downwards and inwards in front of the rectum. In this case encysted peritonitis may occur between the rectum and the bladder in the male, and between the rectum and the vagina in the female. Digital examination of the vagina and rectum gives information as to the existence and situation of the purulent collection, which may open spontaneously into the rectum, bladder, or vagina.

A few cases of peri-umbilical abscess have been noted. If the appendix is displaced inwards in front of the cæcum, the adhesions favour the limitation of the collection above and internal to the iliac fossa, near the umbilicus.

It is of the utmost importance to bear in mind the retrocæcal abscess. In many cases the appendix in its normal state ascends behind the cæcum as far as the colon. It may even be fixed in this region by adhesions left by former attacks. In such a case, pus may collect behind or around the cæcum, and point in the costo-iliac space or in the lumbar region above the iliac crest. If the appendix has a mesentery, the abscess may develop in the subperitoneal cellular tissue, above the iliac fascia. In this region the peritoneal abscess readily becomes cellular, and the varieties may be confounded. Let me also draw attention to appendicular psoriasis.

The retrocæcal abscess, bounded in front by the cæcum and ascending colon, may spread in different directions, and cause the following varieties:



It often opens into the cæcum. I would again remark that many cases formerly considered as examples of typhlitis ending in perforation of the cæcum and in perityphlitis are really cæcal and pericæcal lesions resulting from a primary lesion in the appendix. This variety of retrocæcal abscess may also empty itself into the ascending colon. In other cases the retrocæcal abscess is directed towards the inferior surface of the diaphragm as a subphrenic abscess, and may cause perforation of the diaphragm, with empyema and vomica, as described under Appendicular Pleurisy. Finally, the retrocæcal abscess may spread into the iliac fossa, and open into the bladder, rectum, or vagina, or pass into the inguinal canal and scrotum.

I may note also perinephritic abscess. Brun has reported a case of fatal peritonitis from perforation of the abscess in the cavum Retzii. The abscess was secondary to perforating appendicitis.

These are the principal varieties of abdominal abscess due to appendicitis. Spontaneous rupture into the intestine is followed by recovery, if the diseased appendix has been destroyed; in the contrary case, a fresh attack of appendicitis may supervene (as in a case which I saw with Pozzi) a few months after the spontaneous opening of a retrocæcal abscess into the intestine. Do not let us forget, too, that an encysted abscess may subsequently open into the peritoneum, and cause general peritonitis.

General peritonitis is more formidable than the localized form. These varieties of peritonitis are due to the toxi-infection elaborated in the appendix, although it is impossible at present to assign a special rôle to any one microbe.

Appendicular peritonitis, especially in encysted cases, is often of a very foetid odour, even in the absence of gangrene; and yet we cannot say that it is putrid, because it has none of the characteristics of putridity, and gas does not form. It is by an abuse of language that these cases of peritonitis are labelled putrid when they are only foetid. The operator, on opening the peritoneal cavity, is struck by the evil odour of the liquid, and speaks of putrid peritonitis.

If peritonitis is to merit the term "putrid," the symptoms should be like those of putrid pleurisy. We ought, on opening the peritoneum, as on opening the pleura, to find a mixture of liquid and gas. Such is not the case. The abdominal wound, like the thoracic one, should be liable to invasion by gaseous phlegmon. This is not so. Cultures of the peritoneal fluid, like those of the pleural effusion, should be capable of producing gas; and inoculation of peritoneal pus, like inoculation of pleural pus in the guinea-pig, should cause a gaseous phlegmon; but this result is not seen.

I have tried in vain to find a case of putrid peritonitis, whereas cases of putrid pleurisy are counted by dozens. And when putrid pleurisy follows a pre-existing peritonitis, as in appendicitis, we find that the pleurisy is

frequently putrid, whereas the peritonitis is not. And yet both the peritonitis and the pleurisy have the same origin, and appear to be due to the same microbes. What, then, is the explanation?

Although this fact is paradoxical, it exists, nevertheless. I might even say that it is the rule. In the chapter on Appendicular Purulent Pleurisy we find pneumothorax, pyopneumothorax, and the issue of gas during the operation, whilst similar signs of putrefaction in the case of peritoneum are nowhere described.

I am not speaking here of subphrenic pyopneumothorax, which is almost always associated with the perforation of a neighbouring organ. I am not speaking, of course, of the case where gas passes into the peritoneum through a perforation of the intestine. This is peritoneal pneumatosis by perforation, comparable to pneumothorax by perforation. In both cases the presence of gas is caused by effraction, and this has nothing to do with the formation of gas in putrid fluids. This fact is so true that if an operator, on opening the peritoneal cavity for appendicitis, notices the presence of gas, he immediately looks for a perforation in the cæcum or elsewhere.

I am not sorry to make a parenthesis on this topic, and I have shown by the clearest proofs that the toxi-infectious focus is produced in the closed cavity of the appendix. I am now able to add a further proof in support of my views. The frequency of gangrene and perforation in the course of appendicitis is well known. If the focus were not produced in the closed cavity, or if it communicated freely with the cæcum, as has been incorrectly maintained, the intestinal gases would pass into the peritoneum, and would cause **pyopneumoperitonitis**, which is not the case.

I have still a few words to add on peritoneal infection of appendicular origin. In the long ascending track of infection which starts from the appendix, climbs into the peritoneal cavity, and ends in the pleura, I see three stages which differ according to the nature of the lesions. The chief lesion in the appendix is mortification; foetor is the chief symptom in the peritoneum, and putrefaction is the chief process in the pleura. And yet we find the same microbes in all three stages, thus showing how premature are conclusions as to the action of certain microbes in appendicular toxi-infection. We must consider not only the quality of the microbes, but also the soil in which they live.

It is to be remarked that the process of putrefaction does not take place in the peritoneum, but in the pleura, which is in close relation with the air-passages. We have, therefore, two large serous membranes—the peritoneum and the pleura—inoculated with similar micro-organisms in appendicitis. The former does not lend itself to putrefaction, though it is protected from all contact with the air-passages; while the latter lends itself marvellously, though everywhere it is in most intimate contact with the



air-passage. I am content to draw attention to this fact, which does not tally with our knowledge of anaerobes, which become more active in the absence of air. The reason has still to be discovered.

In order to finish the description of appendicular peritonitis, I have still to speak of secondary foci, which seem sometimes to have no anatomical connection with the initial focus. These remote foci (Tuffier) resemble those we sometimes meet with as a consequence of otitis in the cerebral hemisphere or cerebellum on the diseased, or even on the opposite, side. These abscesses are due to microbes, which are carried to a distance from the place of origin. Sometimes the secondary abscess is already present when laparotomy is performed for peritoneal symptoms, but at other times it only appears one, two, or three weeks after the cure of the appendicitis. These secondary foci are usually found in the most internal and inferior portion of the cæcal region, near the bladder. In a patient convalescent from appendicitis Tuffier noticed and opened a remote abscess in the external oblique muscle. Routier has reported four cases of secondary abscess, appearing nine, fourteen, sixteen, and twenty-five days after laparotomy. The complications usually arise in the following way: the patient has undergone the operation, and everything is going on well. Cure appears certain, when new and alarming symptoms appear. Sometimes the symptoms clearly indicate the formation of a fresh peritoneal abscess, which is readily diagnosed; at other times the symptoms simulate those of acute phthisis or typhoid fever, and the abscess develops behind the cæcum, transverse colon, or stomach, or in the left iliac fossa, etc.

Another important complication is **secondary perforation** of the intestine. In addition to the appendiculo-peritoneal focus, it is not rare to find (especially in patients who have been operated on somewhat late) violet or blackish gangrenous patches on the cæcum and colon. These lesions often pass unnoticed at the operation, and all danger seems passed, when gas and matter make their exit through the wound. Secondary perforation of the gut has then taken place.

**Infection of the Liver, Pleura, etc.**—To complete the description of secondary infections due to appendicitis, I have still to describe purulent infection of the liver and purulent and putrid infection of the pleura. In view of the great importance of this question, I have devoted three special sections to it under Disease of the Liver and of the Pleura.

**Toxic Appendicitis—Appendicæmia.**—I have so far described the **infective** lesions, but there is another factor with which we have not reckoned—viz., the toxines found in the appendicular focus, with all the complications. This focus is not only favourable to an increased virulence of the microbes, but serves also for the manufacture of toxines. Clinical study has confirmed the experiments of the laboratory, and has furnished

me with irrefutable proofs as regards the toxicity of appendicitis. Thus are now explained the grave and fatal complications, the cause and genesis of which were formerly unknown.

If I am not mistaken, it was from the rostrum of the Académie that the idea of toxic appendicitis was first well established.\* Since then I have never ceased to study the effects of the poison on the liver, stomach, kidneys, etc. Thence came the idea of toxic appendicitis, which pours its poison into the blood, and leads to a toxæmia which I have called **appendicæmia**.

We shall have in future to consider albuminuria, icterus, and hæmatemesis, which all belong to appendicitis.

**Kidneys.**—I commenced a systematic search for albuminuria in acute appendicitis, and found it present in a large number of cases even early in the disease. The toxine may affect the kidneys by the second day. Sometimes the amount of albumin is small; at other times it amounts to 10 or 15 grains. Generally (and I am speaking of successful cases) albuminuria which has been marked prior to the operation disappears after removal of the appendix.

A more serious point is that from the onset of appendicular nephritis granular casts may be noticed in the urine. The lesions in these varieties will be studied under Appendicular Kidney.

**Liver.**—In some patients with acute appendicitis of moderate intensity slight jaundice is noticed from the first. It must be carefully looked for, and is particularly noticeable in the conjunctivæ. The skin of the face is earthy and of a salmon tint. Cases of this appendicular jaundice will be found under Lesions of Liver, Stomach, and Kidney, due to Appendicitis.

This early toxic jaundice is rarely due to the presence of true bile pigments, but depends chiefly on the false pigments (urobilin) which are found in the urine on chemical and spectroscopic analysis. In appendicitis jaundice may appear as an isolated symptom or be associated with other toxic symptoms. It may even run the course of icterus gravis. I shall describe this toxic hepatitis under Diseases of the Liver.

**Stomach.**—The vomiting noticeable in appendicitis may be caused not only by the appendicitis or the concomitant peritonitis, but may also be due to toxic gastritis, accompanied in some cases by erosions of the stomach. In such cases blackish streaks will be seen in the vomited matter, indicating slight hæmatemesis. Only too often, however, repeated attacks of hæmatemesis occur, and are of exceedingly grave prognosis. I have called them **appendicular vomito negro**.

Albuminuria, icterus, oliguria, hæmorrhage, and hæmatemesis are not always present together. They are sometimes isolated, or at other times

\* Dieulafoy, "Toxicité de l'Appendicite" (*Académie de Médecine*, 1898, et *Clinique Médicale de l'Hôtel-Dieu*, 1899, 17<sup>me</sup> Leçon).



appear in succession or in combination. The prognosis is more or less grave, according to the particular complication.

**Nervous Troubles.**—The appendicular intoxication is revealed by nervous troubles simulating meningitis (epileptiform convulsions, coma), with or without icterus gravis, and with or without uræmia.

In one of Rénon's cases the patient, who was deeply poisoned, succumbed, with bulbar symptoms: acute dyspnoea, cyanosis of the face and extremities, asphyxia, and syncope. In consultation with Legry and Hartmann I saw a woman suffering from gangrenous appendicitis and peritonitis. On the evening after operation the patient felt relieved, and next day showed an improvement. On the third day there was slight jaundice, and on the fourth Hartmann found that the patient had a strange look. She was not clear in her replies, and by noon was unconscious, repeating the same words continually, and uttering inarticulate cries. I saw her at seven in the evening, when she lay motionless, with her eyes fixed, uttering a short, strident cry at intervals. The loss of consciousness was complete, the pupils were equal, and the respiration was slightly accelerated. Ocular paralysis, facial palsy, hemiplegia, and contractures were absent. These negative symptoms eliminated any idea of meningitis, and the patient succumbed the next day from cerebral intoxication. Marchant, to whom I spoke about the case, told me of a similar one in a young girl operated upon for appendicular peritonitis. The result of the operation was excellent when, two days later, slight jaundice of the conjunctivæ was noticed. The restlessness commenced in the evening with involuntary movements of the head. The patient uttered cries at intervals, lost consciousness, and succumbed during the following day, without fever or acceleration of the pulse. Death in this case was due to intoxication.

Such are the toxic nervous complications. They must not be confounded with cerebral complications of an infectious origin (abscess of the brain), for these remote infections are slower than the toxic complications.

**Summary.**—Clinical study and laboratory experiments agree in demonstrating the toxicity of appendicitis. The intoxication may be slight or intense and fatal. The poisoning is slight when it is confined to transient changes in the liver and kidneys, and when it does not show itself by icterus and albuminuria. The intoxication is then not very formidable, but in appendicitis we never know what may happen. The first sign of intoxication must sound the alarm, and we may fear the worst if icterus gravis, uræmia, copious hæmatemesis, or nervous complications supervene. Valmont's patient died on the third day with symptoms of icterus gravis. The patient whom I saw with Hartmann and Legry was much improved by the operation, but the appearance of icterus gravis was followed by symptoms of cerebral intoxication and death. G. Marchant's patient was carried off under similar conditions, and a patient of Routier with epileptiform symptoms died in coma. In the case of a patient whom I saw with Gros and Cazin the toxic phase commenced with general icterus, oliguria, and anuria, and ended with terrible attacks of hæmatemesis.

Appendicitis is essentially a toxi-infectious disease, and therefore not only to be feared because of the peritoneal or remote infections, but also

because it poisons its victims, in addition to infecting them. Sometimes, indeed, the intoxication is worse than the infection. In a word, one of the most redoubtable results of appendicitis is **appendicæmia**. I cannot, therefore, urge too strongly that the only rational treatment of appendicitis is early surgical intervention and removal of the toxi-infectious centre.

**The Treacherous Calm in Appendicitis.**—I desire to call attention to the apparent calm which occurs when the condition is most threatening. In a communication to the Académie de Médecine I have called it the **treacherous calm**. To quote a characteristic example :

On January 14, 1899, I was called by Pinard and Roques to see a young woman who was four months pregnant. The pregnancy had been normal, except for severe vomiting in the second and third months. While in excellent health she was taken ill with pains in the stomach, vomiting, and severe diarrhoea during the night of January 9. She had six stools during the night, and two more next day. The family at first feared a miscarriage, and sent for Pinard, who discovered that this was not the case. The uterus had no part in the pains on the right side of the abdomen. On January 10 the condition was unaltered. Pinard did not see the patient. She passed a bad night on January 11, and the pains in the right iliac region were intense. Roques was called in. On the 12th two attacks of vomiting and pain at McBurney's point. On the 13th vomiting, meteorism, and muscular resistance in the appendicular region. Temperature in the morning 101° F. ; in the evening 102° F., preceded by a slight rigor.

Such was the situation on the evening of the 13th. It was a severe case of appendicitis. The danger was increasing, and the time was ripe for operation. Next day, at half-past eight in the morning, I was called in, and was told that a visible change had taken place since the previous evening. The abdominal pains had disappeared, and the vomiting had ceased. The patient had slept well, and had twice passed water and gas. The temperature had fallen to 99.5° F. The patient insisted on the improvement during the night, and considered herself well. At first sight the mischief seemed to have stopped. This condition was in some degree comparable with that of a patient who overnight is in the throes of pneumonia, and who on the following day experiences the relief of the crisis.

The chart on p. 779 gives an exact idea of this treacherous calm. If we merely look at this chart, noting the maximum and the fall of the fever, we might think that the disease had come to an end. We shall see that nothing of the kind had happened. I first examined for pain at McBurney's point, but the pain which had been so well marked, had now disappeared. I pressed the abdomen in all directions, especially over the right iliac fossa, and I was not a little surprised to find it painless. There was no question of a change brought about by medical treatment (morphia, antipyrin, or the application of ice), as none of these remedies had been employed. The pain had therefore disappeared spontaneously.

One abdominal symptom of the utmost importance, however, persisted—namely, general abdominal tympanites.

What had happened since the previous evening, and what did this apparently complete truce mean? We all agreed that the patient was suffering from severe appendicitis.

We were of opinion that it was one of those transient calms which deserve recognition. We felt that the patient had **peritonitis**, basing our diagnosis on the general tympanites, the slightly drawn look, and the acceleration of the pulse, which stood at 104, in spite of the fall of temperature.

When we arrived at noon, the apparent improvement still continued. The tem-



perature had fallen still lower, to 98° F. Second found, as we had done, absence of pain, but general distension of the belly and acceleration of the pulse, while the eyes were slightly sunken. He advised immediate operation. Hardly was the peritoneum opened when a flood of turbid serous fluid of most foetid odour gushed out. Diffuse peritonitis was present, and the appendix was gangrenous. In spite of these complications, the mother recovered. The miscarriage took place on the twelfth day after the operation, the foetus having succumbed to the appendicular toxins.

I desire particularly to point out in this case the **treacherous calm** of appendicitis. In the very midst of the peritonitis the calm came on, the

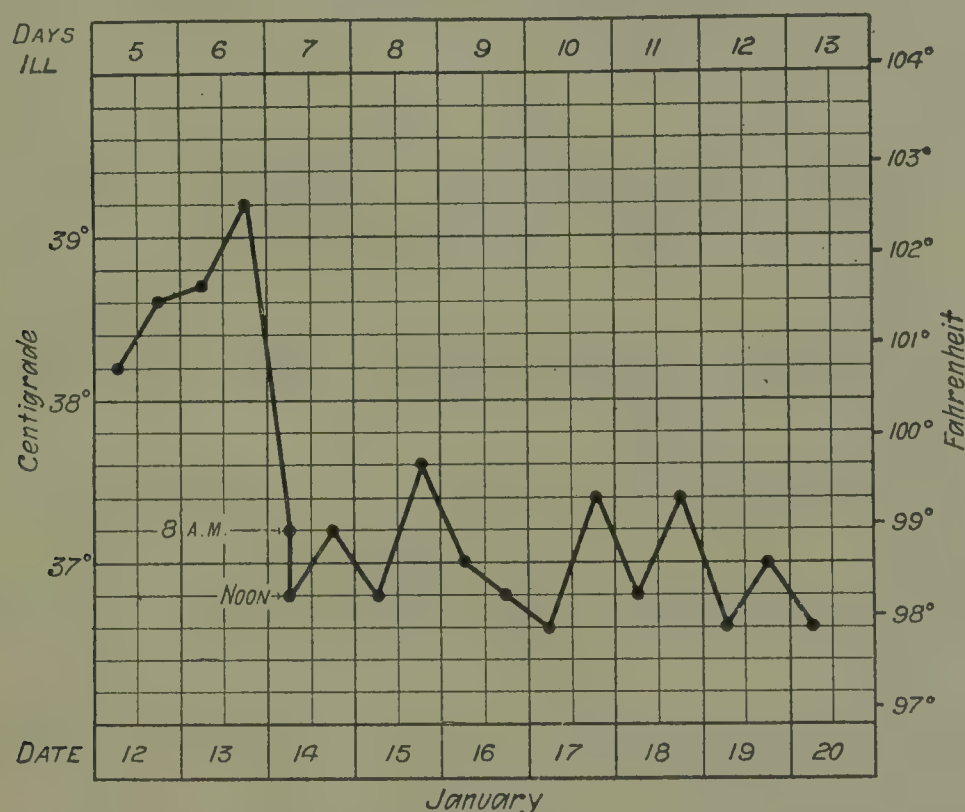


FIG. 41.—CHART SHOWING THE TREACHEROUS CALM IN APPENDICITIS.

fever fell, and the pains disappeared, and yet fatal mischief was commencing. The patient would have been dead in forty-eight hours but for the operation.

This treacherous calm is not uncommon. Several cases will be found in a clinical lecture on this subject.\* This apparent arrest or period of calm is frequent in appendicitis, and is not sufficiently recognized, because it has not been previously described. Whether this arrest of the disease is caused by treatment (bags of ice on the abdomen, opium pills, injections of morphia) or not, it is none the less true that the acute symptoms that signalize the first stage of appendicitis are sometimes followed by a well-being which looks like a real improvement in the condition. In such a case we postpone

\* "Les Accalmies Traïtresses de l'Appendicite" (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 12<sup>me</sup> Leçon).

the proposed operation in the false hope of gaining time. Complications appear quickly, and when we deem it desirable to intervene, we are too late.

Strange though the fact appears to be, we are forced to admit that in a fair number of cases the most severe peritoneal lesions coincide with an apparent arrest in the appendicular symptoms. It may perhaps be possible to find an explanation of such discordant facts. The remarkable experiments of Klecki on the pathogenesis of peritonitis of intestinal origin have led me to investigate the pathogenesis of the closed cavity in appendicitis. He has shown that the virulence of the coli bacillus reaches its maximum in the ligatured coil, which is thus converted into a closed cavity. The microbes then pass through the walls of the ligatured coil, reach the peritoneum, and cause peritonitis (a process identical to that which I have found in the closed cavity). The unexpected fact which results from Klecki's experiments is that the virulence of the bacilli taken from the peritoneal fluid is in most cases less than that of the bacilli in the loop of gut. In other words, "the passage of virulent microbes into the peritoneal cavity lessens their virulence."

Without wishing completely to assimilate clinical and experimental facts, it is impossible, nevertheless, not to be struck by their analogy. In the experimental case (conversion of an intestinal loop into a closed cavity), as in the clinical case (conversion of the appendix into a closed cavity), an intense exaltation of virulence and an acute focus of infection are produced. In the experimental, as in the clinical case, the pathogenic microbes become more virulent in the closed cavity, traverse the walls of the appendix, and cause diffuse, septic, or localized peritonitis. Why not admit, then, that the onset of peritoneal lesions may sometimes coincide with the temporary attenuation in virulence of the microbes which have come from the appendicular focus, thus causing the treacherous calm?

Theory is, however, of little importance. The essential clinical fact is that it is not possible, in the great majority of cases, to establish two distinct periods in the course of the appendicular and peritoneal symptoms, which are similar and subintransient. It is often difficult to know at what moment peritonitis shows itself. Indeed, the treacherous calm appears during the onset of peritonitis.

The course of events is as follows: A patient is suffering from appendicitis. Ice-bags are applied to the abdomen; opium or aperients are administered. Next day the disease follows its course. The pain at McBurney's point is acute, vomiting occurs, the pulse is quick, and the temperature rises to 101° F. Medical treatment is continued, injections of morphia are administered, and the patient is kept absolutely still on his back; and yet, in spite of medical treatment (which I consider is useless),



the situation becomes alarming—the pain is intense, the abdomen is distended, and the necessity for surgical intervention strikes the observer.

Then, in the midst of this indecision, an improvement takes place—the pain lulls, the temperature falls, the vomiting ceases, and the disease grants a truce. The medical treatment is given credit. But the truce is of short duration. Fresh symptoms soon appear, and the danger becomes pressing. The cloven hoof of peritonitis is stamped on the face of the patient. Surgical intervention is then accepted as a last resort; but, be the operation performed or not, it is already too late, and the patient will succumb. Many cases of appendicitis run this treacherous course, and end in death unless we operate in time.

In appendicitis we must distinguish between a real improvement and a treacherous calm. The former concerns all the symptoms at the same time, and there is no marked discord between them. The pulse-rate and the temperature fall, the tympanites decreases, the pains disappear, and the abdominal distension becomes less.

The treacherous calm is very different. The temperature falls, but the pulse remains small and quick (100 to 120 beats). The abdominal pains improve, either spontaneously or under the influence of ice and morphia, but the abdomen remains distended and tympanitic, and careful observation shows that the facies is peritoneal.

**Pregnancy and Appendicitis.**—The following example gives an idea of the relation between pregnancy and appendicitis :

On January 6, 1896, I saw a young woman, recently confined, who, within the last forty-eight hours, had been taken ill with pains in the abdomen and vomiting. Budin, who had delivered the mother, gave me full details. The pregnancy, the confinement, and the puerperium had all been normal. Eighteen days after the confinement, when everything was going well, the young woman complained of abdominal pains, which were slight at first, but soon became acute. The pains had been followed by nausea and vomiting. Constipation was absolute. The previous night had been bad, and the situation had grown worse. I found the features drawn, the eyes dark, and near the bed a basin containing greenish vomit. The fever was high; the pulse was small and quick. The case was undoubtedly one of peritonitis. The abdomen was uniformly distended and so tender that the patient begged me not to touch it. I wished, however, to be certain of **the seat and origin of the pain**, for in such cases diagnosis is impossible without a methodical examination. I palpated the abdomen with the greatest care, and when I came to the right iliac fossa, I found hyperæsthesia, pain on pressure, and muscular resistance so marked over the cæcum, that I felt sure of appendicitis during the puerperium. Budin shared my opinion.

Although it was but the third day of the disease, peritonitis was already present. Immediate operation was necessary, for the symptoms were alarming, and the prognosis was growing worse every hour. I asked Routier to operate. He did so at 10 p.m., and found diffuse peritonitis. The appendix was enlarged, of a purple colour, and surrounded with soft, purulent false membranes. It was removed, and I found that a calculus had given rise to a closed cavity, which was filled with pus. The disease did not at once yield, as the patient, who was poisoned by toxines, had fever for several

days, with rapid pulse, nausea, gastric intolerance, and slight jaundice of the conjunctivæ. The situation gradually improved, and the patient was cured at the end of three weeks. This case of puerperal appendicitis is, I believe, the first one of its kind published in France.\*

Since that date Pinard has published several cases, and has addressed a very important communication to the Académie de Médecine. His statistics show thirty-one cases of puerperal appendicitis operated on, and the period of pregnancy when the appendicitis occurred. They also show the extent of the peritoneal lesions, the nature of the operation, and the results as regards the mother and the foetus.

We have now to discuss the pathogenesis of puerperal appendicitis. In a general manner the pathogenesis of appendicitis is invariable, and we must always return to the *primum movens*—that is to say, to the conversion of a portion of the canal into a closed cavity, in which the toxic products causing all the complications are manufactured. We have to decide whether pregnancy has any direct influence on appendicitis. I can readily believe that lithiasis, which is one of the causes of appendicitis, may be set down to pregnancy, as may other forms of lithiasis, and especially biliary lithiasis. I do not say, of course, that every case of puerperal appendicitis is due to a calculus. We possess little information on this subject, for the examination of the appendicular lesion is generally curtailed or passed over in silence. Still, the two cases of puerperal appendicitis which I have seen, were both due to calculi.

However this be, as Pinard says, “the fact, which is well established, and which must henceforth claim attention, is: **appendicitis may complicate child-bearing in all its stages—during pregnancy, during labour, or during the puerperium.**” Thirty-one operated cases prove this assertion. Observations show that appendicitis may cause trouble during any stage of pregnancy, just as it may complicate the puerperium. Appendicitis generally interferes with the pregnancy, and threatens the life of the foetus as well as that of the mother. Nearly all authors have remarked how frequently a viable foetus has been expelled dead, or has died from some indeterminate cause, or presented signs of septicæmia during the few days of life. Our case may furnish an example of the fact. The culture made by Wallich gave pure cultures of the *Bacillus coli*, proving the infection *in utero*, the blood having been taken from the vessels of the umbilical cord.

A description of puerperal appendicitis is unnecessary, because there is no difference between it and the non-puerperal form. When appendicitis supervenes during pregnancy or after accouchement, it is always appendicitis with its many varieties, and with the symptoms which I have de-

\* Dieulafoy, “Manuel de Pathologie Interne,” 1897, t. iii.



scribed in detail. It begins without prodromata, whilst the patient is in a state of perfect health, with pain, localized in the right iliac fossa. This pain does not attain its greatest intensity at once, and no matter how acute the onset, the pain **gradually increases**. Careful interrogation of patients shows that the pain attains its maximum only after one or several hours.

Furthermore, the localization of the pain is of much help in diagnosis, and even if it spreads in various directions, the seat of election (pain, muscular resistance, and hyperæsthesia) occupies the middle of a line drawn from the umbilicus to the right antero-superior iliac spine. The prognosis in appendicitis is certainly aggravated by the puerperal condition. Not only the infection, but also the toxicity, is to be feared, and it must not be forgotten that in the pregnant woman the liver is in danger of becoming affected.

Here, as in every case of appendicitis, the only rational and efficacious treatment is surgical intervention. "I cannot but agree with this judgment," says Pinard. "Perhaps I would go further, and say that appendicitis during pregnancy must be treated surgically more rapidly than in any other case, because of the close relation of the infectious focus to the genital organs. For the moment I am not concerned with the question whether the infection spreads by continuity or by means of the blood-vessels. Whatever the means, infection is present, and two lives are endangered. This sums up the whole question. These conditions may not be set aside when the practitioner finds himself face to face with appendicitis in a young woman. Being mindful of the relation of appendicitis to the puerperal condition, it will be found wise to remove the infectious focus, though up to now we may have hesitated. This would be, in my opinion, good prophylaxis."

I have nothing to add and nothing to withdraw from the views I have so often expressed. Appendicitis, whether puerperal or not, must be operated on without delay. I know that there is some hesitation in opening the abdomen of a pregnant woman: the opposition of the friends is not always easy to overcome; but if exact examination shows us that the patient is suffering from appendicitis, she must be operated on at once. The foetus will, perhaps, succumb, but the mother will be saved. Cases will be quoted in which puerperal appendicitis was not operated upon, and yet neither the foetus nor the mother died. I grant this, but it is a poor argument in the face of our knowledge of the gravity of appendicitis, and especially of the puerperal form.

**Diagnosis.**—Appendicitis is the most important of all abdominal maladies. Its frequency, its various forms, and its gravity, must always keep us on the alert, so that we may diagnose it and act at once. There is a toxi-infectious focus, which must be removed as soon as it appears. To

temporize is to court disaster. We must try, therefore, to make a correct diagnosis.

While the diagnosis of appendicitis is very easy in some cases, there are others in which the reasoning cannot be too close. The treatment of appendicitis demands exact diagnosis, because we can then resort to opportune surgical intervention. Likewise, it is by a correct diagnosis that we can eliminate all idea of an operation in a patient whose symptoms merely simulate appendicitis.

In this discussion I shall base my arguments on the 200 cases of appendicitis which I have treated during the past eight years. As they were **all verified by operation**, the correctness of the diagnosis cannot be called in question.

In the first place, it will be well to establish the foundations on which the diagnosis must rest by discussing the signs and symptoms which mark the onset and the course of the disease. For the facility of description, the different varieties of appendicitis may be placed in three groups :

**Group I.**—Appendicitis of benign appearance. The onset is not very painful; the symptoms are classical, but not very severe; the fever is not high; vomiting is rare or absent; and constipation is the rule. Methodical examination of the abdomen shows that the pain, muscular resistance, and cutaneous hyperæsthesia are readily appreciable at McBurney's spot. This type of appendicitis formerly swelled the list of cases of so-called stercoral typhlitis, but we know to-day that the disease is appendicitis, not typhlitis, and the diagnosis has become easy; but the prognosis is impossible, because, in spite of its benign appearance and the absence of urgent symptoms, the attack may on the third or fourth day pass on to gangrene, peritonitis, or poisoning of the patient, who dies for want of early operation.

**Group II.**—The disease has an acute onset and severe course. The patient, who was in good health the night before, suddenly feels abdominal pain, which is not at first confined to the right iliac fossa, but may occupy the epigastric or umbilical regions, though it soon becomes most marked in the right iliac fossa, and rapidly reaches its maximum. "The pain is so severe as to make the patient cry out or writhe in agony." Nausea is constant, and vomiting of food or bile is sometimes early and frequent. Fever appears, and the pulse is quickened. On the first day the facies may be anxious and drawn. One examining the patient, the abdomen may be found painless in different regions, and especially in the epigastric region. If, however, the examination is methodical, we find the painful triad localized at McBurney's point: intense pain increased by pressure, muscular resistance, and cutaneous hyperæsthesia form the triad. If we watch the face of the patient, we see that the features contract, and that a look of agony is present when but slight pressure is made over the appendix. On the first or second



day the temperature may rise to 103° F. The urine is sometimes albuminous. This form of appendicitis certainly presents the least difficulties in diagnosis, because the severity and the clearness of the symptoms leave no room for doubt.

It must be admitted that this symptom-complex much resembles that of peritonitis. The peritoneum, however, may be absolutely intact, and appendicitis alone is capable of causing all these symptoms. The appendicular and peritoneal symptoms may be similar. It is often impossible to say when peritonitis complicates appendicitis, as I have previously pointed out.

**Group III.**—The disease may be called larval, as it assumes the **mask** of some other disease. The triad of appendicitis is not wanting if carefully sought for, but it is partially masked by other symptoms which alter the nature of the morbid picture. In one patient, for instance, the vomiting of food and of bile is so marked that it simulates ordinary indigestion. The physician is often called to a child some twenty-four hours after the so-called indigestion, when the fits of vomiting associated with the painful triad (which is to be found, if carefully looked for) were but the prelude of an appendicular infection. When the parents know the truth, they say: "We thought that it was a case of simple indigestion!"

If we are forewarned, we shall not be deceived by these appearances. In the case of a child or an adult who has been taken ill some hours before with so-called indigestion, let us always remember that the vomiting may be of appendicular origin. Let us think of appendicitis, and carefully explore McBurney's region, looking for pain, muscular resistance, and hyperæsthesia. Careful examination, which may be repeated **twice or three times during the day**, will give the true diagnosis, and show that the vomiting put down to indigestion is really due to appendicitis.

In some cases the disease is masked at its onset by the profuse diarrhœa which I have called "defensive." The intestine, by copious secretory action, apparently tries to get rid of the poison. This diarrhœa, wrongly taken for enteritis or for entero-colitis, has falsified the pathogenesis of appendicitis. The case may be thus stated: A person is taken ill with intestinal pain and diarrhœa. His first idea is that "his stomach is upset," and he treats himself with cataplasms and laudanum. He then sends for a doctor, who sometimes accepts the diagnosis of enteritis, relying on the information furnished by the patient. The cause of the enteritis is then sought for, and the mischief is attributed to indigestible food, iced drinks, or a chill. The supposed enteritis is treated with opium, bismuth, and cataplasms. In spite of the treatment, the situation does not improve: the abdominal pains become sharper, and constipation follows the diarrhœa, while the abdomen becomes distended, vomiting appears, and the patient

on the third or fourth day of his supposed enteritis is really suffering from well-developed appendicitis. We then recognize that the diagnosis of enteritis was incorrect, but we try to explain matters by saying that the appendicitis has been the outcome of entero-colitis. This is a mistake.

I have often seen appendicitis commence with diarrhoea, and I know of no better example of this masked form of appendicitis than one of the cases of which I have spoken in connection with the treacherous calm. We have become so accustomed to see constipation accompany appendicitis that we may not recognize appendicitis accompanied by diarrhoea. An error in diagnosis may then readily occur. If a patient is taken ill with abdominal pain and diarrhoea, we must investigate the cause and the seat of the pain. Methodical exploration of the appendicular region allows us to make our diagnosis.

Another form of masked appendicitis simulates **renal colic**. I had under my care the following remarkable case :

A man of twenty-eight had had attacks of pains for the past two years. The first attack occurred on March 30, 1896. His previous health was good, but he was seized with "cramp" in the right side of the belly. The pain was accompanied by vomiting and fever, and he was obliged to keep his bed for several days. His doctor diagnosed hepatic colic, and prescribed purgatives and milk diet. In December, 1896, he had a second attack of pain, which radiated to the right groin and testicle. The third attack took place in July, 1897, and was characterized by acute pain in the right side of the belly, fever, fits of vomiting, and radiation of the pain into the thigh and the right testicle. The diagnosis pointed to renal colic, and the patient was kept in bed for a week. Finally, the fourth and last attack occurred on April 20, 1898, and was in every respect like the preceding ones. The pain radiated to the umbilicus, the right thigh, and the right testicle. At times it caused **retraction of the testicle**, just as in renal colic. The patient had to keep his bed for eighteen days, and avoid all movement. The convalescence was unsatisfactory, and the abdominal pains did not completely disappear.

I examined him, and found pain localized over the cæcum. Hyperæsthesia and muscular contraction were also noticed, together with indefinite, deep-seated, induration. The triad in question (pain, muscular resistance, and hyperæsthesia over the appendix) was in favour of appendicitis. It was, however, difficult to explain the symptoms of renal colic in the two last attacks.

In spite of these symptoms, I discarded the diagnosis of renal colic and substituted that of appendicitis. My reasons for doing so were as follows. In renal colic the lumbar region on the affected side is always painful ; it was not so in this case. Furthermore, the pains of renal colic appear and cease suddenly. The pains, though atrocious for a few hours, disappear as if by magic as soon as the stone has passed into the bladder. In this patient the abdominal pain lasted eighteen days ; indeed, pain in the right iliac fossa was present when I examined him. And, again, the disease had left signs at McBurney's point which are characteristic of appendicitis, and not of renal colic. I therefore asked Marion to operate on the appendix.

The diagnosis was found to be correct, and the operation cleared up the symptoms which had been set down to renal colic. The appendix reached up behind the cæcum, and was surrounded by a mass of false membranes, which united the posterior surface of the cæcum to the ilio-psoas muscle. The adhesions were due to the attacks of appendicitis, which had been mistaken for renal colic.



The large adherent appendix was difficult to remove. It was obliterated at its base, and we found below the stricture a small, deep ulceration, which would soon have perforated. The tumour, adherent to the anterior surface of the psoas, was in intimate connection with the genito-crural nerve, which passes over this muscle. The stimulation of the branch of this nerve, which is distributed to the skin of the scrotum and the cremaster, explains the pain and the retraction of the testicle. The stimulation of the crural branch, which sends out cutaneous twigs to the superior and anterior part of the thigh, explains the pains in that region. The pathogenesis of the symptoms which in my patient had caused appendicitis to simulate renal colic was thus verified.

This is by no means an isolated case, and I have collected several in one of my clinical lectures.\*

Tuffier has published some examples of pseudo-nephritic symptoms in appendicitis. He mentions the case of a patient taken ill with vomiting, ballooning of the abdomen, and acute pain in the right side, radiating into the groin. The diagnosis of renal colic was made. As the situation grew worse, laparotomy was performed, and revealed appendicitis. Tuffier asks whether the association of pseudo-nephritic pains with appendicitis may not be attributed to the ureter being enveloped by adhesions. Vesical symptoms sometimes attract special attention in appendicitis. A few years ago I saw with Pinard a man of about fifty years of age who had an attack of appendicitis with anuria, and was sounded because he was convinced that he had a stone in the bladder.

Let us next consider appendicitis which simulates **hepatic colic**. In some cases of appendicitis the appendix ascends so high behind the cæcum and the ascending colon that the pain radiates up to the hypochondria. The patient complains of abdominal pains, and at the same time indicates the subhepatic region, so that we may hold the bile-ducts at fault.

Furthermore, some patients have jaundice, and if a patient has jaundice, vomiting, and pains in the hypochondriac region, the idea of hepatic colic naturally presents itself. On closer inspection, however, we find that in appendicitis, although the pain is felt as high as the hepatic region, there is always local pain at McBurney's point, **which is never the case** in hepatic colic. Again, the pains of hepatic colic frequently radiate into the shoulder. This never happens in appendicitis. The jaundice of hepatic colic is obstructive, and is generally characterized by clay-coloured stools, and true bile-pigments in the urine; whilst the toxic jaundice of appendicitis does not cause decoloration of the fæces, and is generally characterized by the presence of urobilin and brown pigment in the urine. Furthermore, in toxic appendicitis with jaundice the urine nearly always contains albumin. These different points should, therefore, prevent our mistaking appendicitis for hepatic colic.

We may now discuss the diagnosis of diseases which simulate appendicitis. I must insist particularly on entero-typhlo-colitis, which is often mistaken for appendicitis. Mucous, membranous, or calculous entero-typhlo-colitis always presents a symptom-complex, in which diffuse pain is the chief feature. The intestinal colic, which may or may not be accom-

\* "Diagnostic de l'Appendicite" (*Clinique Médicale de l'Hôtel-Dieu*, 1899, 13<sup>me</sup> leçon).

panied by fever and vomiting, is most marked over the ascending, transverse, and descending colons. When the cæcum is especially affected (typhlitis, or typhlo-colitis), the pain is very near the region of the pain due to appendicitis. While, however, the pain is most severe in the right iliac fossa, we have in favour of the diagnosis of entero-typhlo-colitis the diffusion of pain along the colon. The patient by a gesture may give a diagnostic hint. He speaks of a "painful bar," and indicates the transverse colon with his hand. In other cases he will complain of a "painful circle" in the abdomen, and at the same time trace by a significant gesture the diffusion of the pain along the three colons.

On examination, the right iliac fossa is often painful, but still you do not find the triad as clearly defined as in appendicitis. Besides, other painful points exist over the ascending colon, the hepatic flexure (in front and behind), or the transverse and the descending colons. Appendicitis does not show this feature.

In appendicitis the patient is almost always taken ill while in good health. Prodromata are rare, judging by the 200 cases which I have verified by operation. On the other hand, a patient with entero-colitis will say that some weeks or months previously he had pain in the left iliac fossa (descending colon), or in the subumbilical region (transverse colon). He will say that for months or years he has been subject to intestinal derangements, and that a chill, indigestible food, and fatigue give him colic and diarrhoea. He often suffers from constipation. He watches his stools carefully, and has noticed mucus, slimy matter, or "skins." Analysis of the fæces after an attack reveals the presence of sand. Nothing of this kind occurs in appendicitis, and there is only "an intestinal history" when the patient has already had several attacks of appendicitis.

It will, however, be urged that entero-colitis, or typhlo-colitis, may lead to appendicitis. I do not share this view. **Appendicitis is neither the consequence nor the outcome of entero-colitis.** This question will be discussed in detail under Entero-colitis. I may state here that if in an intestinal attack we find symptoms and signs of mucous, membranous, or calculous entero-colitis, there is practically no fear of appendicitis.

I cannot lay too great stress on the differential diagnosis between appendicitis and entero-typhlo-colitis. As appendicitis inspires widespread alarm, care must be taken not to see appendicitis everywhere, and to advise operation for appendicitis **which does not exist.** If I insist on this point with some complacency, it is because I have advised patients **against an operation** which had been advised. They were suffering from mucous, membranous, or calculous typhlo-colitis, and had not appendicitis.

**Cholecystitis** might simulate appendicitis, were it not that the pain occupies a different region. There are cases, however, in which appendicitis



is associated with cholecystitis. I have made a special communication on this point to the Académie de Médecine,\* and when I speak of the diseases of the gall-bladder, I shall discuss this association of cholecystitis and appendicitis.

The diagnosis between appendicitis and lead colic is not always easy. The pain in lead colic is sometimes very marked in the right iliac fossa—that is, in the region of the cæcum—and may be called saturnine typhlalgia. Do not let us forget that there may be typhlalgia, just as there may be enteralgia or colalgia. In the case of typhlalgia, spontaneous pain and pain due to pressure exist in much the same region as in appendicitis. The history, the blue line, the previous attacks of lead colic, and the absence of fever are, however, opposed to the idea of appendicitis. Furthermore, the pain in lead colic is diffused over the whole abdomen, which is tense and painful at other spots than the right iliac fossa, and pressure often relieves the pain.

I have previously quoted several cases of gastric or duodenal perforation mistaken for appendicitis. We may succeed in making a correct diagnosis by the following points: The suddenness, severity, and localization of the pain are in no wise comparable in the two cases. The pain following perforation of an ulcer is sudden and agonizing, and I have called it “the peritoneal dagger-thrust.” The pain of appendicitis is neither so sudden nor so acute, and may take several hours to reach its maximum. The localization of the painful triad is likewise different, being most marked in the right iliac fossa in the case of appendicitis. In the case of perforation of the stomach it is most severe in the epigastric region.

It is sometimes difficult to distinguish between appendicitis and primary pneumococcal peritonitis. I shall here mention the chief diagnostic points.

Primary pneumococcal peritonitis attacks young children at the very age when appendicitis is frequent. In both cases there is sudden and acute abdominal pain, with vomiting of food and bile. In both cases the fever may be severe or slight. Pneumococcal peritonitis, however, causes even at its onset severe foetid diarrhoea, whilst diarrhoea is exceptional at the commencement of appendicitis. Finally, though the pain of pneumococcal peritonitis often commences in the iliac fossa, the “painful triad” is in favour of appendicitis.

Inflammation of the right adnexa is sometimes difficult to diagnose from appendicitis. While the former disease is essentially pelvic, and the latter abdominal, the diagnosis is very complicated where previous inflammation of the tube and ovary has formed permanent adhesions with the appendix. A history of gonorrhoea or puerperal mischief, the onset and

\* Dieulafoy, “Association de l'Appendicite et de la Cholécystite, avec ou sans Péritonite” (*Académie de Médecine, Séance du 16 Juin, 1903*).

the course of the symptoms, and the exact localization of the pain must then be taken into consideration.

Some women at the approach of the menstrual periods suffer from congestion, ovarian pain, vomiting, and tympanites, with radiating pains and hyperæsthesia of the abdomen. The least touch causes sharp pain over the appendix. We find on closer inspection that pain is most marked, not at McBurney's point, but lower in the ovarian region. I once saw this happen in a young girl who was said to be suffering from appendicitis. Next day all the symptoms disappeared with the onset of the menses.

**Tubal pregnancy** shows some analogy with appendicitis when the lesion is on the right side. I saw the following case with Moizard and Nélaton :

A young woman had been taken suddenly ill with pain in the right iliac fossa, vomiting, and syncope. A few hours later the facies was drawn, the pulse was small and quickened, and the face **extremely pale**. These are not the symptoms of appendicitis. Excessive pallor, acceleration of the pulse, and a tendency to syncope are rather the result of hæmorrhage. Besides, in this case the subumbilical region of the abdomen was absolutely dull, which already indicated a large effusion. The diagnosis of intra-peritoneal hæmorrhage from rupture of the tube was made. She was two months pregnant. The operation verified the diagnosis and brought about recovery. About 3 pints of blood were sponged out.

**A floating right kidney** causes sometimes very severe pain in the right iliac fossa, with or without vomiting, and must not be mistaken for appendicitis. Careful exploration of the region will allow of a diagnosis being made.

I would remind the reader that **hysterical** patients may present the following symptom-complex : ballooning of the abdomen, pains in the iliac fossa, and vomiting. The condition somewhat resembles the picture of appendicitis, but in such cases we find stigmata of hysteria, with hysterogenic zones, and pain localized in the ovarian region, and not in that of the appendix.

**Chronic Appendicitis.**—I do not allude to tuberculosis, cancer, actinomycosis, etc., which may attack the walls of the appendix. They do not show the infective type, which alone deserves the name of appendicitis. I would divide chronic appendicitis into two groups—the one in which the mischief becomes chronic after one or more acute attacks, the other in which it is chronic from the first. The first group is the more common. Appendicitis which has thus become chronic, is sometimes the fault of the patient, who has refused a timely operation, and is therefore the victim of chronic mischief in the appendix.

This form is often the fault of physicians or surgeons, who put aside "the early operation," thus exposing the sufferer to very grave risks, and who prolong "medical treatment," which allows appendicitis ample time to become chronic.



In chronic appendicitis we always find lesions around the appendix—viz., false membranes and caseous or purulent material. As the chronic infection spreads beyond the appendix, it ends in the production of “irregular fibrous adhesions, which fix the appendix to the cæcum, ileum, mesentery, or peritoneum.” Patients complain of dragging pain in the right iliac fossa or at other spots. The pain is aroused by walking or other exercise, and may occur in acute attacks.

Chronic appendicitis at times affects the digestive organs. The patient complains of gastric or intestinal trouble, and becomes dyspeptic. We usually see constipation, with the passage of mucus or membranes in stools, but diarrhoea is also seen. We are then prone, through inaccurate observation, to suppose that chronic appendicitis is consecutive to mucous colitis. If, however, we arrange the history of events in chronological order, we find that the chronic appendicitis has caused these intestinal troubles, which roughly resemble muco-membranous colitis.

Appendicitis is more rarely chronic from the first. In this event we find calculi, ulcers in the mucosa, small abscesses in the wall of the appendix, and purulent fluid. These lesions run a slow course. Fever is absent, but dragging pain is present in the right iliac fossa or other regions. Marked exacerbations may occur, and closely simulate an acute attack of appendicitis. There is, however, no reason to suppose that these cases of chronic appendicitis are consecutive to colitis.

**Treatment of Appendicitis.**—At the Académie in 1896 I stated (and my statement still holds): “**There is no medical treatment for appendicitis.** Medical treatment simply causes loss of valuable time. It is obvious that the patient can be relieved by injections of morphia, application of ice-bags to the affected region, and other soothing measures; but do not let us be mistaken as to the efficacy of these means. Too often they lead us to believe in cure, when they only mask the symptoms. In view of this fictitious improvement, we speak of typhlitis, or appendicular colic, and prophesy recovery; but yet the patient dies for want of proper surgical intervention.”

The various descriptions of this medical treatment are a mass of contradictions. Some writers say: “Avoid enemata and purgatives, which excite peristalsis, and favour the spread of the disease. Give, rather, opium, which checks peristalsis and induces constipation.” Other writers say: “Give purgatives in appendicitis, because they favour intestinal antisepsis and lessen the infection in the intestine.”

These theories regarding purgatives, balladonna, or opium prove that the old views of the pathogenesis of appendicitis have still some weight. This arises from the fact that we are still fettered by wrong theories, which confound appendicitis, typhlitis, and enteritis, and deny to appendicitis its proper independence. I would ask of what use is it in appendicitis to

administer some castor-oil or magnesia? There exists in the appendix a focus **absolutely isolated from the rest of the intestine**, in which the toxi-infection is being elaborated. What benefit, then, can result from purgatives, belladonna, or opium? Physicians hold that they can cure appendicitis with purgatives or other medical measures. They exaggerate the importance of these measures, and they wrongly suppose that, in order to act on the appendix, they must act through the intestine! To reach the appendix we must open the abdomen.

The so-called medical treatment is both inefficient and inopportune, because, as soon as pain heralds the onset of appendicitis, the mischief is already done—the canal is obstructed, the focus is formed, the toxi-infection elaborated in the appendix is beginning its work, and no one can tell where this work will end. Colonies of virulent microbes traverse the walls of the closed cavity, the appendicular vessels become thrombosed, gangrene is imminent, the toxins are absorbed, and the poison is spread through the system, while the relative merits of belladonna and opium are being discussed! Moreover, we know the result of this medical treatment. Chauvet says that the death-rate has been 30 per cent., without counting the patients who did not succumb to the first attack of appendicitis, but recovered with a damaged appendix, and were therefore predisposed to fatal recurrence at an earlier or later date (Réclus). A death-rate of 30 per cent. is not equalled in the annals of the most deadly of our diseases, even though we go back to former epidemics of typhoid fever, scarlatina, or diphtheria.

As regards the cause of death in these cases, Chauvet writes: "The patients usually succumb from general suppurative peritonitis, sometimes from peritoneal septicæmia, or at other times from an infection of the entire system, starting from purulent or gangrenous sinuses that originate in the appendicular focus." **Since this focus is the cause of the disease and of death**, would not early surgical intervention be better than ice, purgatives, or opium?

Early surgical intervention is the only rational treatment of appendicitis, because it alone affords protection from immediate and remote complications, and because it alone forestalls relapses and the results seen in appendicitis which has not been operated on. We sometimes operate too late. We all know of such cases, and if we count them up we shall gain a more accurate idea of the value of medical treatment. I cannot see the advantage of temporizing. A patient has acute appendicitis, and the appendix is, perhaps, on the point of perforating or becoming gangrenous, and you temporize. A patient has peritonitis which is becoming diffuse, and you are satisfied to use purgatives, opium, or the ice-bag. My opponents will say: "We only advise operation in cases of appendicitis with peritoneal complications, in which it has been proved to us that medical treatment is



insufficient. I would, however, ask them: "By what signs and symptoms can you prophesy as to the course of appendicitis? Can you say that the case is not one of **toxic** appendicitis, which is more to be feared than the infective form?" When appendicitis has commenced, no one can say what terrible surprises may happen. I have repeatedly said that the violence of the fever and the rise in temperature have absolutely no precise signification. In many cases the temperature is but slightly raised, in spite of peritonitis. The intensity of the pain and the general symptoms are deceptive when we have to distinguish between appendicular and peritoneal lesions, because peritonitis is often insidious, and appears without hiccough, vomiting, or rise of temperature.

In my clinical lectures I have described apparently benign cases of appendicitis in which gangrene, perforation of the appendix, and peritonitis had set in within forty-eight hours.

In my first case the fever was insignificant, and the appendicitis appeared to be most benign; but yet in forty hours the appendix had perforated, and the peritonitis was fully developed. In my second case the appendicitis seemed so trifling at first that the patient thought it was indigestion, and yet in twenty-eight hours the appendix had perforated and the peritonitis was diffuse. In my fourth case the fever was slight and the symptoms were very trifling, but yet the operation, though performed twenty-eight hours after the onset of symptoms, proved the existence of diffuse peritonitis. The indication, or the contra-indication, for an operation cannot, therefore, be based on the severity or on the apparent benignity of the disease.

If the reader will refer to the sections on toxic infection of the liver, kidneys, and stomach in appendicitis, he will find numerous cases of patients who were either not operated upon or were operated upon too late. They succumbed, not to the infection, but to the intoxication, because the latter invades the system more quickly than the former.

In most cases of appendicitis it is impossible to foretell the course of events, and, *a fortiori*, it is impossible to push the finesse of diagnosis so far as to say that appendicitis is plastic, parietal, or otherwise. These classifications, which are faulty in pathological anatomy, are absolutely illusionary in clinical medicine. As regards treatment, appendicitis is indivisible. We can never say whether appendicitis may not be complicated with severe infection or toxæmia. On the second, third, or fourth day the appendix may perforate, and death may threaten. Medical treatment masks the symptoms, dulls the pain, and quickly leads the patient to his death.

As regards the surgical treatment of appendicitis, two camps have been formed. To the one camp belong those who advise the "cold" operation—that is, they do not operate until the appendicitis has "cooled"; the other camp comprises the partisans of the "hot" operation—that is, those who operate during the acute stages in order to suppress quickly the cause of the mischief.

It is evident that someone is at fault in proclaiming that the operation should be performed when "cold," or that it should be carried out when "hot."

The adherents of the "cold" operation publish statistics which have the appearance (and nothing but the appearance) of success. It is said that the "cold" operation gives remarkable successes, whilst the "hot" operation shows many failures.

Let me explain. The wonderful results of the "cold" operation are stated, and when these figures are compared with those of the "hot" operation, at first sight they appeal to our imagination. **This is only an optical delusion**, for these figures do not really state the case fairly. Let us place the matter in its real light, and consider the question of postponing operation in acute appendicitis until the disease has "cooled." In the first place, the success of the "cold" operation need cause no surprise. The "cold" operation is performed on a patient who is practically well. The acute phase has passed, the more or less poisoned system has issued victoriously from the strife, and when the peril is passed the "cold" operation collects the remains lying on the field of battle. Let us beware of ascribing to such belated surgical intervention the honours of the cure. The "cold" operator extinguishes a focus which is no longer on fire. Nevertheless, he performs a useful piece of work in safeguarding the future, but do not let us give to these statistics an importance which they do not possess.

These statistics ought to be all the more modest, because they should give the number of victims who have succumbed to acute appendicitis during the so-called "hot" stage. Many patients while awaiting the "cold" stage succumb in the very midst of the "hot" stage." My grievance against these statistics is that they only refer to the actual operation, and take no count of the deaths due to putting off the operation. In forty cases of acute appendicitis, ten, on an average, die of infection and intoxication whilst awaiting the "obligatory cooling." The statistics which have only the "cold" operation in view take care not to mention these deaths, which, however, may clearly be laid to the teaching deduced from these figures; and hence the said statistics falsify our conclusions and our judgment, as well as that of the public. For several years past I have carefully considered appendicitis from the medical and the surgical point of view. I have kept in touch with these questions, and the catastrophes I have seen have led me to protest against the unsound idea of "cooled-down" appendicitis.

The partisans of "cold" operations do not fail to point out the inferiority of the statistics concerning "hot" operations. Here again the statistics, as they are presented to us, are merely an **optical delusion**. Faulty compilation and wrong interpretation lead to error. As a matter of fact, in



these statistics of "hot" operations, writers have classed together operations performed at every period of the so-called "hot" stage, and do not take the trouble to tell us whether the operation was performed on the first day of the disease or at some later date. Exact details which would be of the greatest value are generally passed over in silence. We are informed of one thing only—viz., that the patient was operated on in the "hot" stage, and statistics drawn up *en bloc* are naturally erroneous, because they omit the most important thing—viz., the day on which the operation was performed. This point sums up the whole question. A case of appendicitis which would have been cured by timely operation may end in death if the operation is postponed to the third day or later. If the operation is done too late in the "hot" phase, when the patient is already hopelessly poisoned, failure will result. The failure is then wrongly attributed to the "hot" operation. These statistics *en bloc* concerning the failure of "hot" operations are therefore worthless, and we must be the more on our guard because by their apparent simplicity they falsify our judgment and they deceive the public.

On the other hand, if we analyze carefully cases of appendicitis operated upon at various periods of the so-called "hot" stage, we shall see that the ideal plan is to operate as soon as possible after the onset—not later than forty hours—because in almost every case the danger is at that time limited to the appendicular focus. By removing this focus we cut away the root of the disease.

I know that it is not always easy to intervene so quickly, but we as physicians and surgeons are responsible for postponing the operation until the appendicitis has "cooled," because we expose the patient to the risk of death by so doing.

I can, moreover, give exact details of more than 200 cases of appendicitis which have been operated upon by well-known surgeons. Cases of grave appendicitis operated on in the first forty-eight hours have **almost all recovered**. The few failures were cases of toxic appendicitis, with gangrene, high fever, great pain, and profuse vomiting. In such cases the operation at the end of the second day is almost too late.

Until lately neither the appendicular intoxication nor the rapidity of its course has been studied. The disease makes irremediable progress in a few hours. A patient who would have been saved by an operation before the end of the second day may succumb if he has been operated on a few hours later, because his organs have become poisoned by the toxins.

In severe cases of acute appendicitis operation on the third day may be successful, but such a result is not constant. The toxi-infection has already made headway, and is more to be feared than on the second day. Amongst those who recover, some are in great danger for a time.

As for cases of severe appendicitis operated upon on the fourth day, and, *a fortiori*, later, I can give no definite answer. There have been numerous successes, but the failures depend on the degree of the infection and poisoning which have had time to act **before** the operation. Too often the operation is performed too late.

These facts prove that we must strive to make a correct diagnosis. The patients or their friends sometimes tell us that the disease began twenty-four or forty-eight hours previously, because they make the onset of the disease coincide with the greatest pain. Methodical examination may rectify the error, showing us that a case of appendicitis said to be two is really three or four days old. These details are of extreme importance, for everything is of importance in a disease where even twelve hours or less can change the course of events.

These ideas must be universally taught. At different times men of the highest authority have proclaimed similar views, and have done much for their adoption. My colleague Lucas-Championnière recently made an interesting communication to the Académie de Médecine,\* and even more recently Legueu, Chaput, G. Marchant, Segond, Routier, Hartmann, Poirier, and Villard, have collected cases and communicated their views to the Société de Chirurgie. I trust their teaching will banish into oblivion the practice which exposed patients to the danger of death, because the operation was delayed until the appendicitis had "cooled down."

Our duty as physicians is to come to a decision, and to imbue the laity with our convictions. The surgeon is too often called in after we have temporized with futile medical treatment. It is not sufficient to operate: the operation must also be timely.

In my opinion, thirty-six hours should be the maximum delay in grave cases of acute appendicitis, and a delay of forty-eight hours should be the maximum in less severe cases. This formula, I believe, gives absolute security. By it the toxic focus will be removed before irreparable infection and poisoning have had time to occur.

A final word as to an attack of appendicitis which has not been operated on.

I cannot share the mental calm of those who abandon all fear when the patient has recovered from the attack of appendicitis. The fire often smoulders under the ashes, and the appendix is ready to cause relapse, perforation, or secondary infection.

*Hæret lateri lethalis arundo.*

There is one formula which I should like to engrave on the mind of those who may still be inclined to hesitate: You will never repent having operated

\* Académie de Médecine, séance du 15 Juillet, 1902, et *Journal de Médecine et de Chirurgie Pratiques*, 10 Août, 1902.



on a case of appendicitis, but you may often repent that you did not do so or that you operated too late. Given a correct diagnosis and a timely operation performed with proper skill, **patients ought not to die of appendicitis.**

**Summary.**—The evolution of the operative indications in appendicitis is the natural consequence of recent research which has elucidated the question.

If we look backwards, we see that at no very distant date the danger of appendicitis was supposed to consist in perforation and peritonitis. Peritonitis was looked upon as the only complication to be feared, and it sufficed, so it was thought, to watch its onset, in order to intervene successfully.

The question of appendicitis was soon found to be far more complicated. In the first place, the symptoms of appendicitis and of peritonitis often blend, so that it is almost impossible to say when the latter commences. It is, therefore, impossible to watch for its appearance. Furthermore, histological and bacteriological researches have shown that the pathogenic agents, after becoming more virulent in the closed cavity, pass through the walls of the unperforated appendix, and at times cause acute peritonitis. As this form has not the sudden onset of perforative peritonitis, its appearance may escape the notice of the cleverest physician. The idea, then, commenced to gain ground that in operative interference we must not consider the peritonitis, which is a secondary complication, but only the appendicular focus, which is the primary trouble.

More attention was paid to the suppurative infections of the liver, pleuræ, pericardium, lungs, etc. The pathogenesis of these remote infections was traced out, showing that they arose, not from the peritonitis, but from the appendicular focus whilst it was cooling down. It became obvious from the point of view of surgical success that we must early remove the appendicular focus.

Finally, the toxicity of appendicitis was proved, and it was shown that the intestinal microbes in the closed cavity may kill by the toxins which they produce. These toxins cause the early lesions in the kidneys, liver, stomach, etc., and are in a large degree responsible for the rapidly fatal complications of appendicitis. To this toxæmia of appendicular origin I have given the name of **appendicæmia**. Peritonitis has nothing to do with it.

Peritonitis has, therefore, lost its former importance. It is no longer the sole object considered by the operator, though it remains one of the most formidable complications in appendicitis. The predominant importance of the appendicular focus as being the origin of the infection and of the poison

is now recognized. The danger is not limited to the peritoneum. The focus in the appendix can produce appendicæmia, and peritonitis is of minor importance. Surgical intervention must systematically and promptly attack the *primum movens* of the trouble in the appendix.

We must never think solely of the peritonitis, thereby overlooking the rapidly fatal complications due to the appendicular toxins. Delay in operation till the appendicitis has "cooled down" may condemn the patient to death by giving the terrible toxi-infection time to spread; an evil that is too often irremediable.

#### IV. MUCOUS, MEMBRANOUS, AND SABULOUS ENTERO-TYPHLO-COLITIS—INTESTINAL LITHIASIS.

This section deals with mucous, membranous, and sabulous enterotyphlo-colitis, because all these forms may show similar symptoms, and may coexist or follow one another in the same individual. A description limited to muco-membranous entero-colitis and omitting the sabulous variety would be quite incomplete. "Enterocolitis" has always appeared to me an imperfect name, because it only includes the small intestine and the colon, and apparently disregards the cæcum, which plays a very important part. I have, therefore, substituted the name "enterotyphlo-colitis," in order to comprise the small intestine, the cæcum, and the large intestine, which all take their share in this disease. The colon, however, takes the chief share, whilst the small intestine plays but a minor part. Before describing enterotyphlo-colitis, I think that it will be useful to quote cases from my clinical lectures.\*

A young woman was suffering from severe attacks of abdominal pain, which was most marked in the right iliac region, and thus simulated appendicitis. This condition had begun a few months previously, and was characterized by attacks of colic, with the passage of mucus and skin-like membranes in the stools. The attack of colitis was followed by acute enteritis, and, later, by habitual constipation. Intestinal crises occurred from time to time, the pain being present both night and day. The pains were accompanied by tympanites; they lasted for several hours and spread over the whole abdomen, though they were sometimes more marked in the right iliac fossa. After examination, and for reasons which I shall detail later, I dismissed the idea of appendicitis, as I thought the case might be one of sabulous typhlo-colitis. I had the fæces examined. My opinion was confirmed, for abundant sand, as well as membranes, was present.

A little boy had for a year suffered from sharp attacks of pain along the descending colon. The parents had noticed that the attacks were followed by the appearance of slime and membranes in the stools. As I thought that sand might also be present, I had the fæces examined. They contained an enormous amount of sand. I saw several of the attacks. The pains were not general, but were confined chiefly to the left iliac

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\* *Clinique Médicale de l'Hôtel-Dieu*, 1897, 14<sup>me</sup> leçon.



fossa, in the region of the descending colon. The crises lasted from a few hours to a whole day, or even longer, the pain being as severe as in renal colic, and causing the patient to cry out.

In 1892 I saw a lady, aged about fifty, with abdominal pains, which came on without appreciable cause at indeterminate periods. The pains were accompanied by ballooning of the abdomen, nausea, and constipation. In 1895 the crises became more frequent and severe, lasting for days and simulating the intense suffering of hepatic colic. The pain was sometimes accompanied by a feeling of faintness. I thought of hepatic colic, appendicitis, and ulcer of the stomach or of the duodenum; but I soon rejected these ideas, because the localization of the pains and the other symptoms did not justify them. The pain was certainly not confined to any precise spot, and though it seemed to commence in the epigastric region, it soon invaded the hypochondria, the flanks, and the hypogastrium, extending up as high as the thorax. One day, after a violent crisis, the patient showed me some pieces of stone and a quantity of coarse sand which she had passed per rectum. It was, therefore, a clear case of intestinal lithiasis. I may add that this patient had for some years been subject to attacks of muco-membranous colitis, the stools often containing slime and membranes. The sand was composed of organic matter and a small quantity of lime salts; the larger fragments were made up of phosphate of lime and of organic matter in smaller proportion.

**Description.**—We find a chronic form of colitis, in which constipation is the chief symptom. The pains are at times very sharp, and appear a few hours after meals, either daily or at longer intervals. They are limited, for the most part, to the region of the transverse colon, giving rise to the sensation of a painful bar across the upper part of the belly. The constipation is so obstinate that it may continue for a fortnight or even longer. The patient complains of heaviness in the head. In order to overcome the constipation, he makes immoderate use of enemata and purgatives. At meals he selects “the most nourishing foods.” He has a constant desire to stool, and becomes hypochondriac and neurasthenic. During this obstinate constipation hard and dark-coloured matter, blood-stained mucus, and membranes are passed.

In another variety we find entero-colitis, accompanied by frequent and at times continuous diarrhoea. This form is rarely chronic at first. It is almost always preceded by acute or subacute attacks. Colic may not exist, and diarrhoea is the chief symptom. This diarrhoea, which may be accompanied by borborygmi, usually comes on after meals, and several stools are passed in the twenty-four hours. The character of the dejecta varies. They are often yellowish or greenish, and horribly foetid. In some cases bloody mucus and membranous ribbons are noticed. When the stools contain food which is incompletely digested, we speak of **lienteria** (*laxitas intestinalum*) (λεῖος, gliding; έντερον, intestine). There are periods of remission and of exacerbation, and even if the disease is only of short duration, it induces a loss of flesh which attracts attention, and causes us to think of intestinal tuberculosis.

Muco-membranous and sabulous entero-typhlo-colitis are common in children. With Hutinel and Valmont I saw a little girl of four and a half

years of age who had been suffering for a long time from muco-membranous colitis, the stools at times containing much intestinal sand. Analysis by Berlioz showed that it was composed of organic matter, with salts of lime and magnesia.

Subacute follicular enteritis is the most typical form of entero-colitis in children. It is fairly frequent between one year and five years of age, and may last for months or years. We often see acute attacks of intestinal infection, accompanied by alarming symptoms—infectious erythema, fever, vomiting of food, mucus, or bile, slimy or blood-stained stools, and sharp pains in the descending colon or cæcum.

The above symptom-complex is characteristic of entero-colitis. If the symptoms chiefly affect the region of the cæcum, a correct diagnosis may be difficult, and an operation may be advised for appendicitis, which does not exist.

The course of entero-typhlo-colitis varies. The disease may take the form of acute crises, which recur year after year, or may install itself slowly with diarrhoea or constipation, fever, and the passage of membranes and sand. Typhlo-colitis may be simply sabulous, though it is more frequently muco-membranous. Nevertheless, a patient who has passed mucus and membranes for a long time, may also have passed intestinal sand which has not been noticed. As a matter of fact, slime and membranes are more easily distinguished by their form and colour than sand, which is intimately mixed with the fæces.

The pains of typhlo-colitis have no definite seat. They may be most marked in the transverse colon as a painful bar, in the cæcum, at the hepatic flexure, or along the course of the descending colon. It is rare, however, for the pains to remain localized to one of these regions. They usually become general over a part of the abdomen. I have, too, seen patients who had lumbar pains somewhat like those of renal colic, and thoracic and precordial pains somewhat like those of angina pectoris. These facts had already been noticed by Potain in his masterly description of colitis.

The attack of entero-typhlo-colitis does not always begin suddenly. It is often preceded by malaise and ballooning of the abdomen. I may even say that tympanites (with or without eructations) is one of the most frequent and pronounced symptoms. The crisis which is preceded by these prodromata may last as long as thirty-six hours. These attacks of intestinal colic are frequently followed by the passage of firm motions, which contain slime, membranes, and sand.

The crises may be repeated for several days following at a fixed hour shortly after meals. They may recur several times a month or several times a year. One patient may have a series of crises lasting for some days



or weeks, and may then remain well for a long time. Another patient, on the contrary, may from time to time have a crisis lasting some hours, and recurring every month for many years.

Entero-typhlo-colitis is often associated with dilatation of the stomach, flatulence, and loss of appetite. In some instances the gastric symptoms precede the intestinal troubles. The patient is disposed to nervous symptoms (hypochondria, neurasthenia, melancholia), and to reflex troubles, which comprise dyspnoea, transient tricuspid insufficiency, precordial pain resembling angina pectoris, vertigo, and tremors, supervening during intestinal digestion.

Some patients stand entero-colitis without ill-effect and finally recover, but others are more seriously affected, losing flesh and appearing to have tuberculosis.

Many explanations are given of the intestinal infection just described. It is hereditary, and I have seen several cases of entero-colitis in the same family. It has been asserted that entero-colitis is more frequent in people who lead a sedentary life or give themselves up to overstudy. Muco-membranous colitis is often associated with uterine lesions, and is said to be due to an infection of the intestine from the genital organs. We shall see later the effect of arthritism.

The membranes and the sand deserve notice. The false membranes may be passed in fragments or rolled up in balls. They are whitish, firm, thick, and ribbon-like, so that the patient takes them for pieces of a tape-worm. The ribbons may be  $\frac{1}{2}$  inch broad, and 4 or 5 inches long. They are formed of mucus, degenerated epithelial cells, salts, and in exceptional cases cholesterin.

The sand appears in various forms. Most frequently it is a yellowish or brownish sand, mixed with matter, from which it must be separated by washing and sifting. Sometimes we find sand and pieces of gravel as large as a grape-pip or a hazel-nut. The chemical composition is always the same, and we find two elements present in variable proportions—stercoral matter and salts of lime and magnesia, with traces of silica and chlorides. Cholesterin, which is present in gall-stones, is not found. On the other hand, gall-stones never contain organic matter of stercoral origin.

It may, therefore, be said that sand, gravel, or calculi composed of stercoral matter and of lime and magnesia salts are of intestinal origin. Mixed calculi may, however, be found containing cholesterin, salts of lime, and stercoral matter. In this case the sand or the calculi have originated in the gall-bladder, and have taken up the stercoral matter in their passage through the intestine. I would, however, add that cases of mixed lithiasis (sand or gravel) must be exceedingly rare. I shall shortly speak of a patient in whom attacks of biliary and intestinal lithiasis appeared, together or in

succession, and yet the gall-stones and the intestinal gravel (analyzed by Berlioz) retained their own characters, never showing a mixed composition.

**Intestinal Lithiasis.**—Intestinal sand is of minor importance in enterotyphlo-colitis. I have always seen it associated with muco-membranous colitis. We might admit in such a case an infectious catarrh dependent on microbic agents, and giving rise to the intestinal sand or gravel (lithogenous catarrh). In the pathological history of these patients we may find typhoid fever, dysenteric catarrh, profuse diarrhœa, or obstinate constipation. The production of intestinal sand, therefore, would be simply an epiphenomenon, associated with the infectious lesions in the intestine. This view of the question is perfectly reasonable, and I believe that the presence of intestinal sand is part and parcel of chronic colitis or muco-membranous enterocolitis, without any other designation.

In other cases the facts may, I believe, be interpreted differently, intestinal lithiasis being looked upon as one of the most interesting manifestations of the gouty diathesis (Mongour, Fontet). The question has been asked, and I should like to add certain proofs that intestinal lithiasis is sometimes of gouty origin, just as much as renal gravel or tophi are.

Laboulbène reports the case of a man having hæmorrhoids, asthma, and renal colic as evidences of the gouty diathesis. He had been sent, when forty years of age, to Vichy for phosphatic gravel. When the gravel disappeared from the urine, he suffered from enteralgia, and passed intestinal sand, composed of organic matter and phosphates of lime and magnesia.

I saw with Charrier a lady of gouty stock who passed gall-stones and coarse intestinal sand. The calculi and sand were analyzed by Berlioz, and the interesting feature was that each preserved its own proper composition—cholesterin in the gall-stones, stercoral matter and salts of lime in the intestinal gravel. Her brother has several times passed renal calculi, and has been operated upon for vesical calculus. Their parents are gouty and suffer from hæmorrhoids.

I saw the following case with Rénon: A man of thirty-three, whose family was eminently arthritic (the father and mother having suffered from sick headache, eczema, and gall-stones, while the grandparents were rheumatic, stout, diabetic, and gouty), had been subject to sick headache since the age of five. A few years before, eczema appeared on the hands and face, and resisted all treatment. Three years ago hæmorrhoids appeared and the sick headaches disappeared. Some months ago he noticed slimy mucus in his stools. They were carefully examined, and about forty stones of the size of a grape-stone were found. This voiding of gravel was neither preceded nor accompanied by abdominal pains, which are so common in sabulous colitis. It is worthy of note that the patient was in excellent health at this time. On the other hand, the sick headaches and the eczema of the hands and of the labial commissures returned six weeks after the disappearance of the gravel.

These examples prove the close connection between enterocolitis, intestinal lithiasis, and the gouty diathesis. They show that gout, renal calculi, gall-stones, and intestinal gravel may appear in the same individual at different periods of life, or exist in the same family as hereditary manifestations. The passage of intestinal gravel coincides or alternates with the other



manifestations of arthritism. It may replace them, and, as I said in my communication to the Académie de Médecine,\* I propose to include intestinal lithiasis under the gouty diathesis. I do not say, of course, that every case of intestinal gravel is of gouty origin, for here, as elsewhere, it may be independent of the gouty diathesis. We see patients who are suffering from renal colic, though we are not able to find gout in their personal or hereditary antecedents. The same fact applies in intestinal lithiasis. It is, then, a purely local complication, almost always associated with muco-membranous colitis. At other times the intestinal gravel, with or without muco-membranous catarrh, has an essentially gouty origin.

From this point of view the phosphatic composition of intestinal gravel occasioned some doubt in the mind of Mathieu, who asked whether concretions, principally formed of salts of lime and magnesia, could be considered diathetic, like renal calculi, rich in urates, or, like gall-stones, rich in cholesterin. It seems to me that the composition of the gravel is only of secondary importance in the present discussion, because, firstly, clinical facts point to the diathetic and gouty nature of this gravel in certain patients; and, secondly, because gouty patients have often been attacked with renal colic, and have passed gravel composed both of urates and phosphates. I think it is quite clear that certain cases of entero-typhlo-colitis and of intestinal gravel are of diathetic origin.

**Diagnosis of Entero-Typhlo-Colitis.**—I shall not delay in diagnosing entero-typhlo-colitis and its painful crises from all the abdominal pains which may simulate it. We must, however, consider how to distinguish it from hepatic colic and from appendicitis. Many cases of so-called hepatic colic without icterus are nothing but entero-colitis. The study of these cases shows the source of the mistakes made. In both cases the pain may reach its maximum near the hepatic flexure of the transverse colon. In both cases food and bile may be vomited. The radiation of the pain, however, is not similar, being rather thoracic and scapular in hepatic colic, and abdominal in entero-typhlo-colitis. Tympanites is much more pronounced in entero-colitis than in hepatic colic. The only true way of making an exact diagnosis is to examine the stools for membranes, sand, or gravel. Chemical analysis will reveal their origin.

The diagnosis from **appendicitis** is given fully in the last section. We must now consider the relations said to have been noticed between entero-colitis and appendicitis.

**Appendicitis is not the Outcome of Entero-Colitis.**—We must here discuss a most important question in pathogenesis: Have enteritis and typhlo-colitis a direct influence in the causation of appendicitis? In short,

\* “La Lithiase Intestinale et la Gravelle de l’Intestin” (*Académie de Médecine*, séance du 9 Mars, 1897).

is appendicitis secondary or not to inflammations or infections of the rest of the intestine ?

In a communication to the Académie de Médecine on March 9, 1897, and in the discussion which followed, as also in my clinical lectures at the Hôtel-Dieu, there is one fact which I have tried to emphasize—viz., that patients who are suffering from membranous or calculous entero-typhlo-colitis rarely suffer from appendicitis, and that this proves that appendicitis is neither the consequence nor the outcome of entero-colitis. This rule, of course, is not absolute. Réclus and other authors have collected cases which prove that the same subject may at different times suffer from muco-membranous enteritis and appendicitis. The question, however, is not whether entero-colitis and appendicitis may sooner or later succeed one another in the same individual. We wish to know to what extent they can coexist or follow after one another. If people with entero-colitis were really threatened with appendicitis, entero-colitis, which hitherto has been considered as a benign disease, would then become a far more serious malady.

Comby has devoted a chapter to muco-membranous entero-colitis in the “*Traité des Maladies de l'Enfance*,” but he evidently lays no stress on the relations of appendicitis and enteritis, because the word “appendicitis” is not mentioned. Letcheff, in his thesis, reports twenty-four cases of muco-membranous entero-colitis collected in France and abroad, but he makes no mention of appendicitis. Potain, in a lecture on chronic muco-membranous colitis, makes no allusion to appendicitis, and yet he bases his description on 103 cases of colitis. He also said during the discussion at the Académie: “I agree entirely with Dieulafoy in considering appendicitis during the course of entero-colitis, not as the rule, but as a most rare exception.”

Bottentuit, publishing the results of his observations for some twenty years, saw at Plombières 460 patients suffering from muco-membranous entero-colitis. These cases are subdivided in the following manner: 250 cases in women, 150 in men, and 60 in children; and yet, amongst these 460 patients, not one, to his knowledge, was taken ill with appendicitis, though they were seen several years following.

Glénard had made the following statement: “In my last 100 cases of mucous or membranous colitis I have not seen a single example of appendicitis, and I have no recollection of having ever seen one previously.” Tanche, of Lille, has told me of three persons who suffered for ten years from attacks of muco-membranous entero-colitis, but yet “not one of the three had any sign of appendicitis.” Chabert, of Bagnères-de-Bigorre, has communicated to me his observations in thirty-six cases of patients suffering from muco-membranous colitis, and “not one of them showed the least symptom denoting infection of the appendix.”



Langenhagen, in his article on muco-membranous colitis, arrives at the following conclusions: "To sum up the debate, I cannot do better than give the conclusions of Dieulafoy, whose ideas have been confirmed by Potain, Duguet, Hutinel, Glénard, Berger, Hirtz, Hudelo, etc. Appendicitis—I do not mean pseudo-appendicitis (attacks of pain in the ileo-cæcal region), but true appendicitis, verified by operation—is quite exceptional in the course of entero-colitis. We must not, therefore, consider appendicitis as the consequence or the cause of entero-colitis until we are possessed of further information."

Ewald, of Berlin, is of the same opinion, and holds that there is no relation between appendicitis and muco-membranous colitis.

I quote here Pinard's opinion: "For more than twenty years I have observed many cases of membranous entero-colitis in women, and I find that appendicitis is exceedingly rare. I can, therefore, confirm my friend Dieulafoy's opinion."

For several years I have seen repeatedly, and followed most closely, 150 patients with divers forms of entero-typhlo-colitis. Only two were attacked with appendicitis after several years.

A summary of the above cases is of value, since it is based on some 800 to 900 cases. Many of the patients were seen several times and at several years' interval, and yet it is most difficult to find a case of appendicitis. Such is the clinical fact, and no matter how strange and paradoxical it may appear, it must be admitted. If appendicitis were really the consequence or the cause of entero-colitis, it would be very strange if it had not been observed several times in this large number of cases. As a matter of fact, if there is a disease favourable to enteritis and intestinal ulcerations, it is typhoid fever, where the lesions show a remarkable predilection for the ileo-cæcal region, which includes the appendix.

If appendicitis were often the consequence of entero-typhlitis, as has been maintained, it would surely occur under these conditions, and yet nothing is rarer than appendicitis in typhoid fever. The same remark applies to tubercular entero-typhlitis, as will be seen later.

As we have shown that entero-typhlo-colitis does not cause appendicitis, it will be interesting to ascertain in what proportion appendicitis **properly confirmed by operation** is preceded by entero-colitis. On consulting my personal statistics, based on 200 cases of appendicitis with operation, I can find only four patients who had previously had symptoms of entero-colitis.

Why, then, have some authors so readily admitted that entero-colitis may lead to appendicitis? This fact is due, in the first place, to the mistaken ideas on the pathogenesis of appendicitis; and, secondly, to inaccurate diagnosis, in which pseudo-appendicitis is wrongly taken for true appendicitis.

The discussion just undertaken yields a practical interest of the first

order, and after so many accumulated proofs I may be permitted to restate my dictum: "Appendicitis (**not pseudo-appendicitis**) is very exceptional in the course of entero-colitis." The conclusion is that people with muco-membranous or calculous entero-typhlo-colitis need not unduly fear appendicitis. The attacks of pain, due either to membranes or to gravel, may commence in the ileo-cæcal region (cæcum and ascending colon), the left iliac fossa (descending colon), or the epigastric region (transverse colon); but the physician will know that the case is entero-typhlo-colitis, which is a benign disease, and not appendicitis, which is a most formidable malady.

**Treatment.**—Much has been written on the treatment of entero-colitis, and I may state here the most salient points.

**Diet.**—Milk, milk foods, farinaceous foods, eggs, soups, vegetables *en purée*, green vegetables in small quantities, a little bread or meat; no wine, no pork, no game, no acid foods, no fatty foods.

**Treatment when Diarrhœa occurs.**—If milk disagrees, we may add lime-water, 8 ounces; hydrochlorate of cocaine,  $\frac{1}{2}$  grain; hydrochlorate of morphia,  $\frac{1}{6}$  grain. Five tablespoonfuls to be taken daily (one in each cup of milk). In some cases raw meat may be added to the milk diet or may replace it. Saline purgatives in very small doses—for example, 1 drachm of sulphate of soda every morning—give good results. Bismuth, prepared chalk, pure talc (Debove), opiates, astringents, and nitrate of silver, either in pills or enemata, have been prescribed. I have proved the efficacy of ipecacuanha in small doses, and prescribe four or five pills, each containing  $\frac{1}{2}$  grain of ipecacuanha and  $\frac{1}{12}$  grain of opium daily.

**Treatment in Obstinate Constipation.**—Castor-oil, 2 drachms, several times a week, 7 or 8 grains of rhubarb before each meal; cascarrine or Vichy laxative powder before going to bed; cream of tartar; sulphur and magnesia, small enemata of glycerine, oil, or soap.

Bonnier has obtained extraordinary success in cases of enterocolitis by cauterizing certain regions of the nasal mucosa.

**Treatment of Pain.**—Antipyrin, opium pills of  $\frac{1}{6}$  grain each, a teaspoonful of syrup of codeine, small injections of morphia, hot compresses to the abdomen.

**Local Treatment.**—Large enemata (2 or 3 pints) of water at 104° F. (Mathieu). Enemata of infusion of marsh-mallows; avoid medicated enemata; wear a belt to prevent enteroptosis (Glénard).

**Hydrotherapy.**—Cures at Pougues, Châtel-Guyon, and especially at Plombières.

## V. FALSE APPENDICITIS.

This section is the complement of the two preceding sections, and deals with a most important question in medicine and surgery, because an error in diagnosis may lead either to useless operation or to fatal inaction.



For some years past I have been at pains to prove the falsity of the teaching which holds that appendicitis is commonly the result of enterocolitis. I do not know how far this teaching is responsible for past mistakes, but I am struck by the fact that many persons suffering from muco-membranous or sabulous typhlo-colitis are operated upon for appendicitis.

I am a very warm partisan of surgical treatment in appendicitis. Every-day experience confirms the common-sense rule, which says: "Remove the toxic focus early, before it can cause fatal mischief."

When I found that many persons with typhlo-colitis were operated upon for appendicitis, and when I saw diagnostic and operative errors condoned on the plea of the histological examination, I raised the point for discussion at the Académie de Médecine.

I will first quote certain cases which are conclusive evidence:

1. I saw in consultation a young girl who had suffered for two years from muco-membranous typhlo-colitis. At times attacks of pain came on without any appreciable cause. The motions were loose, and contained mucus and membranes. During the later attacks the pain was most marked in the right iliac fossa, and appendicitis had therefore been diagnosed.

In consequence, an operation had been proposed, and been accepted by the patient, who hoped that surgery would accomplish what medicine had failed to do. I examined the patient, who had just had a sharp attack. The belly was supple, and the walls were flabby. Pressure caused sharp pains over the transverse colon and the hepatic flexure, but the pain was most marked in the right iliac fossa. This feature, however, did not convince me that the case was one of appendicitis, for past experience has shown me that marked pain in the right iliac fossa during an attack of muco-membranous typhlo-colitis is nearly always the result of typhlitis, and not appendicitis. My diagnosis, therefore, was muco-membranous typhlo-colitis, and I advised against an operation.

My colleagues took the opposite view, and decided upon an operation for appendicitis. The appendix was sent to me, and I examined it with Jolly. It was absolutely healthy; the canal and the walls were normal, and no lesion was discoverable on microscopic examination. For the last two years the patient has had attacks of muco-membranous typhlo-colitis. She was operated upon for non-existent appendicitis.

2. Some years ago I was called in to see a young man who had had muco-membranous typhlo-colitis for some time. The symptoms were: "Acute attacks of pain and passage of mucus and membranes. When I examined the abdomen, I found a typical scar in the right iliac fossa. I said at once: "You have been operated upon for appendicitis?" "Yes," he replied, "for appendicitis which I never had. My abdomen was opened by mistake, and the attacks of enterocolitis still continue."

3. Some two years ago I saw a woman, who described her case to me. For some years past she had had symptoms of muco-membranous typhlo-colitis. The last attack had not ended when I saw her. I found in the right iliac fossa a scar of unmistakable significance. "But," said I, "you have been operated upon for appendicitis." "Yes," she answered, "for appendicitis which I did not have, and I am just as great a sufferer as I was before the operation. I have the same abdominal pain, and still pass mucus and skins." It was an excellent opportunity to examine a painful cæcum, when the appendix had been removed ten months before. I recognized pain in the right iliac fossa, due to typhlitis, and comparable to the pain in many cases of appendicitis. I also saw clearly the error of those who, setting aside the other elements in the diagnosis, mistake typhlitis for appendicitis, and perform an unnecessary operation.

**Discussion.** — In certain cases of muco-membranous or sabulous typhlo-colitis, typhlitis is the chief factor, and the pain is most marked in the right iliac fossa; and then, because it is said that appendicitis often results from entero-colitis, our diagnosis may be led astray, and we operate for appendicitis which is not present. These diagnostic and operative errors have increased of late years to a surprising extent, especially since it has become customary to base the diagnosis of certain so-called cases of appendicitis upon a histological examination of the appendix (after its removal). Let me explain.

You are called to see a patient in a severe attack of muco-membranous entero-colitis. He knows the illness is not dangerous, although he suffers considerably. On palpation, you find the abdomen is very tender, especially over the course of the colon; when you press in the right iliac fossa, the patient cries out that the pain is more severe there than at other places. If you are a believer in the doctrine that appendicitis follows entero-colitis, you make a wrong diagnosis of appendicitis, and advise an operation. A surgeon is called in, and when he opens the abdomen, the operation gives the lie to your diagnosis. The appendix is absolutely healthy; typhlitis has been mistaken for appendicitis, and an unnecessary operation has been performed.

The microscope now comes in, and we hear of a new variety of appendicitis (non-existent, it is true), which I have called "microscopic appendicitis." If anyone wishes for information about this microscopic appendicitis, he need only consult the theses of our faculty, in which are published observations where the histological examination of the appendix has the unjustifiable pretension of substituting itself for any other diagnosis. This means that clinical methods, being powerless, yield to histology the right of deciding, after operation, whether appendicitis was present or not. When we read these observations, we find that the appendix was healthy—too healthy, indeed, for those who made the diagnosis. It is, then, necessary to bring in the histological examination to clear up matters which demand an explanation. Bands of fibrous tissue, hypertrophy of the closed follicles, and hæmorrhagic folliculitis, are the chief findings in many cases of "appendicitis," when the post-operative diagnosis is left to the microscope. These histological findings only have the significance that the observer desires to give to them. Conviction will follow the reading of Letulle's important work on this subject. If we take at random the appendices from several adults, it is quite a common thing to find fibrous tissue in process of formation or thickening of the lymphatic follicles, especially in the lower segment of the organ. These changes are quite commonplace in the tissue of the appendix, and we must banish the idea that these changes are appendicitis secondary to entero-colitis. Under the



term "folliculitis" ordinary hypertrophy of the closed follicles of the appendix has acquired too lasting an importance. I have often heard the following phrase: "Did the patient have appendicitis?" "Not exactly; but he had folliculitis." We know the meaning of such a statement, and we must banish this variety of follicular appendicitis into oblivion.

Orth, in a recent communication to the Berlin Medical Society, speaks thus of folliculitis: "It is always difficult to say in the case of a section of an appendix whether the lymphatic follicles have undergone pathological increase or whether the appendix in question is normal, but especially rich in lymphatic follicles"; and he has shown that in 9 or 10 out of every 100 appendices examined after removal for appendicitis the macroscopic and microscopic findings were absolutely normal. I hope in future that we shall not take refuge under a commonplace hypertrophy of the follicles when we have removed in error a healthy appendix.

There is another lesion which histological examination has given us as an integral part of the so-called appendicitis consecutive to entero-colitis. I mean hæmorrhagic folliculitis.

This interpretation is wrong: the follicular hæmorrhages looked upon as a lesion of appendicitis are really due to operative trauma. On this point Letulle says: "I have never found hæmorrhagic folliculitis, save in appendices removed by operation. The forcible ligation of the appendix, before its removal, explains, in my opinion, the circumscribed or diffuse effusions of blood in the reticulated or follicular tissue."

It is, of course, understood that I am not speaking of the hæmorrhages which sometimes accompany the gross inflammatory lesions of true appendicitis. Mention is here made simply of the follicular hæmorrhages which are found in the healthy appendix after removal, and which are wrongly supposed to be caused by appendicitis.

Fig. 42 leaves no doubt. It represents the traumatic hæmorrhage in the walls of a healthy appendix removed by operation.

Fig. 43 represents a longitudinal section of the meso-appendix above the ligature. The tissues are healthy; there is no trace of appendicitis; the ligature has produced the hæmorrhagic foci.

We know, therefore, that the follicular and extrafollicular hæmorrhages which are found (as Letulle has so clearly shown) in the healthy appendix after removal are the result, not of appendicitis, but of an operative procedure. Heubner, from an experimental study of this question, arrives at the same conclusions.

This so-called appendicitis, which has found shelter under the microscopic examination, is non-existent either anatomically or clinically. It cannot even find a place under the heading of "chronic appendicitis." It was this

form which gave the chief support to the theory that appendicitis is commonly the outcome of entero-colitis.

And if this variety is non-existent, I would ask those who believe in it to tell me on what symptoms they base their diagnosis. A patient is suffering from muco-membranous typhlo-colitis: what are the clinical indications for operation? I do not lay stress on the point. I have mentioned the numerous operative mistakes that have been made. Richelot, at the

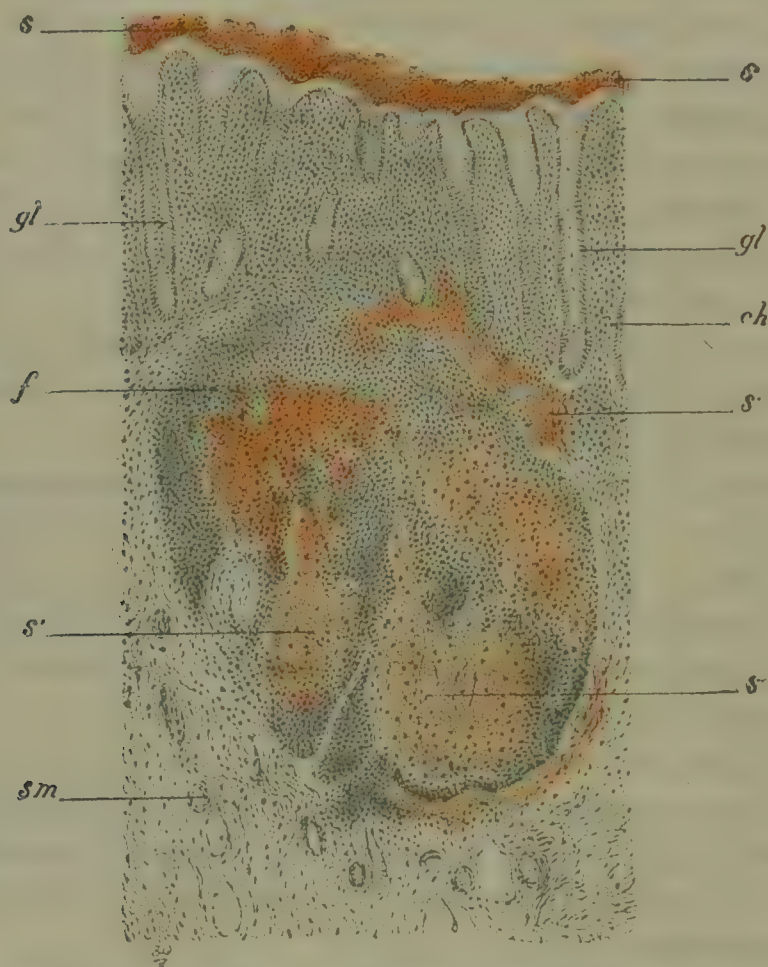


FIG. 42.—SECTION THROUGH A SUBMUCOUS FOLLICLE (LETULLE).

*sm*, Submucosa, quite free from any lesion; *gl*, tubular gland in the normal mucous membrane; *ch*, chorion of the mucous membrane (no inflammation); *f*, submucous follicle (the tissue is torn apart by effused blood *s'*; only red corpuscles are seen). Above and to the right *s''*, the hæmorrhage has extended beyond the capsule; *s*, red corpuscles on the surface of the normal mucous membrane.

Académie de Médecine, has thus expressed his opinion: "Shall I not admit with Dieulafoy that commonplace microscopic lesions may be found in the most innocent appendices, and that their discovery after section in an appendix unnecessarily removed is a poor excuse for the surgeon?"

Moreover, sound clinical investigation should have been able to curtail the faulty practice of operating on so many persons with entero-colitis under the assumption that they have appendicitis; it is sufficient to see



what happens after operations. We should have recognized that the operations effect neither cure nor change in the symptoms. Apart from certain neurasthenics who express relief, muco-membranous typhlo-colitis runs its course unaffected by removal of the appendix. My own inquiries on this point leave no doubt in my mind. In the cases mentioned above the symptoms have continued. In certain cases the improvement has been temporary, and the same attacks have recurred some weeks or months later. Wagner mentions the case of a child classed as a recovery from muco-membranous entero-colitis after the ablation of the appendix. Esmonet

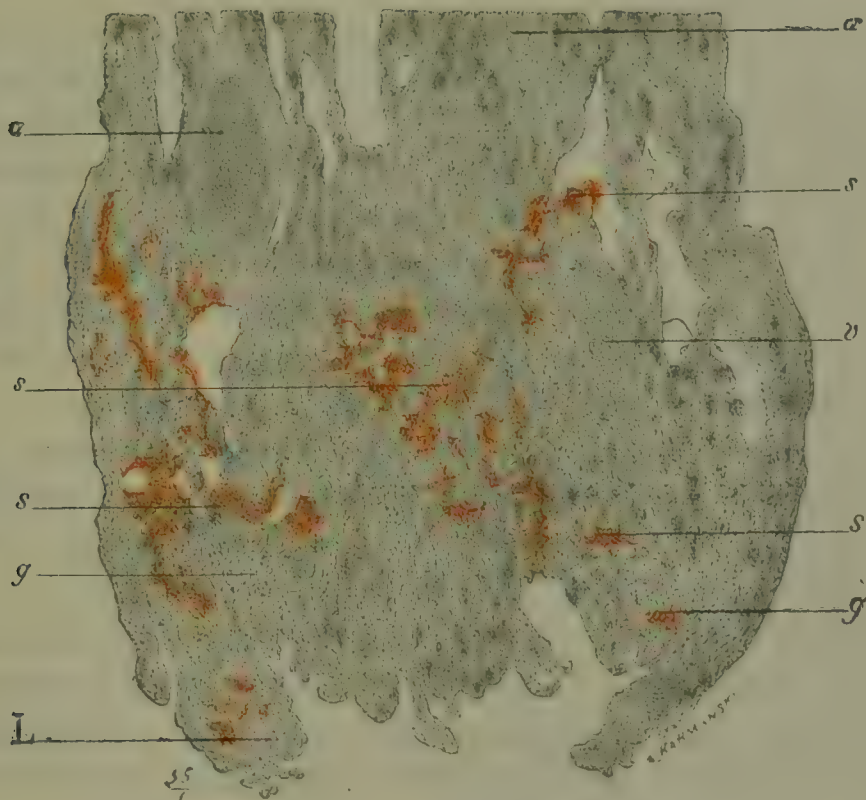


FIG. 43.—SECTION THROUGH THE MESO-APPENDIX (LETULLE).

*L*, limit of the ligature ; *a'*, *a'*, arteries distended by blood ; *v*, vein engorged with blood ; *g*, *g*, fat cells and loose connective-tissue of the epiploon free from any inflammatory lesion ; *s*, *s*, *s*, hæmorrhagic foci between the meshes of adipose tissue in the epiploon.

treated this child at Chôtel-Guyon for attacks similar to those which preceded the operation. Apert saw at the Tenon Hospital a woman suffering from mucous entero-colitis, with much pain. She had been operated on for appendicitis which she did not have, and the old symptoms reappeared. Deléage of Vichy had under his care a lady whose attacks of muco-membranous typhlo-colitis were not a whit better after removal of the appendix.

Bottentuit of Plombières has recently collected twenty-eight cases of persons suffering from muco-membranous or sabulous typhlo-colitis for a more or less lengthy period, and operated upon for appendicitis which they did not have. I have studied the details of these cases, and they closely

resemble one another : erroneous removal of the appendix ; the character of the attacks not changed by the operation ; pain and passage of mucus and membranes still persistent. The sufferers all came to Plombières in order to obtain relief by hydrotherapy.

A wrong diagnosis in a case of false appendicitis may have a vicious action upon the life of many a sufferer from muco-membranous typhlo-colitis. Appendicitis is mentioned, and an operation is suggested. The patient then becomes neurasthenic and fastidious in his choice of food ; he grows thin, and always keeps an eye on his right iliac fossæ ; constipation alarms him.

Sometimes he begs for an operation, at other times he dreads it ; his whole existence is affected. In certain instances he goes to a surgeon and asks to be relieved of his appendix. This is done, but he still has the same attacks of pain, and he finds out that the diagnosis of appendicitis was wrong.

If I speak thus, it is because I have studied this question carefully for many years. I have often prevented an operation which was fixed for the following day or for the following week. Years have passed, and time has shown that my diagnosis was correct.

In short, recent clinical and histological knowledge shows that many persons with typhlo-colitis undergo a futile operation for appendicitis. Correct diagnosis, then, is necessary to prevent error ; we must get rid of the idea that appendicitis is liable to result from entero-colitis.

Clinical study teaches us that in muco-membranous or sabulous enterotyphlo-colitis it is typhlitis which is responsible for the symptoms in the right iliac fossa, and not appendicitis. This view is confirmed by the published cases, and by the anatomical examination of many appendices removed owing to an error in diagnosis. If by chance true appendicitis does supervene, the diagnosis will, I admit, present difficulties, but these difficulties are not insurmountable. In such a case operation is imperative.

## VI. INTESTINAL TUBERCULOSIS—TUBERCULAR ENTERITIS.

**Ætiology.**—While tubercular enteritis may be primary, it is, as a rule, secondary to enteritis in chronic pulmonary phthisis. In some cases it results from auto-infection, the bacillus entering the digestive passages in sputum which has been swallowed. In other cases the bacillus enters in milk from tubercular cows. These infections enter through the mucous membrane of the intestine, but we should also mention infection through the deep tissues, the bacillus being carried by the lymph and bloodvessels, especially in the case of miliary tuberculosis. According to Tchistovitch, when infection takes place through the surface of the mucous membrane,



the bacilli traverse the epithelial layer, reach the walls of the intestine by means of the lymphatics, and spread in the submucous adenoid tissue around the bloodvessels. The transverse direction of these vessels explains the shape of certain ulcerations in which Koch's bacilli are especially numerous.

**Pathological Anatomy.**—On opening the intestine, two kinds of lesions are seen—ordinary inflammation and tubercular changes. The latter are seated, for the most part, in the terminal portion of the ileum and in the cæcum, but they are also found in other parts of the intestine. At the post-mortem examination the lesions appear as granulations and ulcerations, which vary in form and size. The latter are rounded when in the isolated follicles, but longitudinal or oval when seated in Peyer's patches. They are frequently transverse, and surround the intestine like a ring.

The tubercular lesions of the intestine are granulations and ulcers. The granulations arise in the walls of the vessels, in the connective tissue around the ends of the tubular glands, or in the connective tissue of the villi. Their presence induces an inflammatory process in the villi and glands.

The intestinal villi, infiltrated with round cells, unite at their base and form an excrescence, which looks like a tubercular nodule. The tubular glands become elongated and filled with cylindrical cells. The small tubercular mass becomes opaque and caseous, and commences to ulcerate. This is one method of ulceration, but we also find another process, in which the ulcerations originate in the tubercular inflammation of the closed follicles and Peyer's glands. At the commencement, the appearance of the affected follicles does not differ from simple psorenteria. The follicle is enlarged and infiltrated with lymphoid cells. Its centre soon becomes opaque and greyish, then soft, and finally converted into a small abscess, which rapidly ulcerates. "Often several of these altered follicles are found on a Peyer's patch or some other point of the mucous membrane, surrounded by diffuse inflammation of the submucous connective tissue, the villi and the glands, and united to form a prominent patch, which ulcerates." This is another method of ulceration in intestinal tuberculosis, but no matter whether the ulceration begins in the granulations or in tubercular inflammation, "its ulterior development and its consequences are the same."

The ulcers unite and often assume an annular form. Segments or complete rings, each about half an inch wide and some inches from one another, are then found on the mucous membrane. This annular disposition is probably due to the fact that the vessels themselves are disposed in rings around the intestine, their walls being infiltrated with tubercular granulations. The ulcerated Peyer's patches are not swollen, as in typhoid fever, but are only slightly prominent, and studded with crater-like ulcers. These two types—annular ulcers and ulcerations of Peyer's patches—may be isolated or present together in the same subject.

The edges and the floor are often studded with tubercular granulations, which are found in the different layers of the connective tissue of the intestinal walls, between the muscular fibres, and in the deep submucous tissue.

**The lymphatic system** participates largely in the process, and the lymphatic vessels which start from the ulcerated Peyer's patches are injected with tubercular matter. The areas on the peritoneal surface of the intestine which correspond to the ulcers of the mucous membrane, always show numerous tubercular granulations, standing out against the red ground of the inflamed serous membrane. The lymphatic vessels passing to the mesenteric glands are whitish, nodular, enlarged, and infiltrated with tubercular granulations.

Tubercular ulcers of the intestines sometimes cause stenosis, and in rare cases perforation. They are sometimes found in the cæcum in cases of hypertrophic tuberculosis. The ulceration may be so extensive in the large intestine that at first sight the lesions of tubercular colitis resemble those of dysentery.

Tubercular ulceration may also occur in the appendix, and generally coexists with similar lesions in the small intestine, the cæcum, and the colon. I have seen a case of this kind at the Hôtel-Dieu, and the lesions were very marked.\* The ulcers burrowed deeply into the walls of the appendix, but the lumen was quite free, and therefore no symptoms of appendicitis had been observed.

Why is tuberculosis of the appendix latent in spite of the marked lesions in the walls, whilst in appendicitis very small lesions can cause acute toxæmia? In tuberculosis (so long, at least, as the appendix remains permeable) there is no closed cavity, and, consequently, no increase in the virulence nor migration of the coli bacillus through the walls of the appendix. In the sections of the tubercular appendix, of which I have just spoken, the microbes did not pass beyond the superficial layer of the ulcer. They had not increased in virulence, because the lesions had not given rise to a closed cavity. In this case the intestinal ulcers had not caused peritoneal mischief; in sections of them, just as in those of the appendix, the microbes had not passed beyond the surface. Tubercular lesions of the appendix are common. Letulle in some recent statistics has noticed the frequency of lesions of the appendix in tubercular patients. One fact as to these patients struck us both—viz., the rarity of toxæmia, which, clinically speaking, alone deserves the name of appendicitis.

**Description.**—Enteritis is an almost constant manifestation of tuberculosis of the intestine. Tubercular enteritis is rarely found as an isolated

\* This case has been published by Apert (*Presse Médicale*, 1898, No. 102: "Tuberculose de l'Intestin et de l'Appendice; Lésions considérables de l'Appendice; pas de Cavité close; aucun Symptôme d'Appendicite.").



disease, because it is almost always associated with pulmonary phthisis. The severity of the symptoms depends on the extent of the lesions. Sometimes the enteritis is transitory, and is characterized by curable diarrhœa; more frequently the diarrhœa is profuse, obstinate, and accompanied by abdominal pains. Blood may be found in the stools, which have a blackish or brownish tint, similar to the black vomit of gastric carcinoma. This black diarrhœa may persist for several weeks without the slightest modification. It is foetid and as a rule appears only at an advanced stage of the disease. When the large intestine is ulcerated, dysenteric symptoms, with tenesmus, may be seen, as well as the usual symptoms of enteritis. In tubercular patients enteritis is a sign of evil augury. It often ushers in the cachectic period, leading to malnutrition, and hastening a fatal ending.

Hæmorrhage from the bowel is very rarely fatal, though in Chandèze's case the patient had several attacks of hæmorrhage, which were so profuse that he succumbed in twelve hours. The post-mortem examination revealed ulceration of the vessels. In Vallin's case intestinal hæmorrhage killed the patient in a few hours; in Honot's case the intestinal hæmorrhages followed in quick succession, and the patient succumbed in two days.

In **children** tubercular enteritis is always associated with lesions in the mesenteric glands (Parrot). This entero-mesenteric tuberculosis was long known under the name of **tabes mesenterica**. It is characterized by symptoms of enteritis, and also by distension of the abdomen, dilatation of the veins of the abdominal wall, and effusion into the peritoneum.

Perforation of the bowel with peritonitis is exceedingly rare. It would seem at first sight that tubercular ulcerations of the gut might cause perforation, but this is not the case.

In chronic ulcerative tuberculosis Koch's bacilli may pass into the tributaries of the portal vein and reach the liver, where they cause interstitial hepatitis. The lesion is periportal, and is accompanied by secondary cirrhosis and perilobular fatty infiltration.

**Diagnosis.**—In some cases of miliary tuberculosis, the intestinal troubles and the general symptoms resemble the clinical picture of enteric fever. In chronic tubercular enteritis the diagnosis is the more difficult, in that the case does not always look like one of tuberculosis. For months and years phases of improvement occur, and the intestinal troubles are ascribed to gastro-intestinal dyspepsia or to the arthritic diathesis. We must, however, be always on our guard. Cases of interminable diarrhœa or enteritis which seem to recover at Plombières or elsewhere, and then relapse, are most often the result of tuberculosis. In exceptional cases the tubercle bacillus has been found in the stools.

Tubercular enteritis is often rebellious to treatment. Subnitrate of bismuth in large doses, lime-water with morphia in very small doses, albumin-

water, and nitrate of silver in pills, are the means usually employed. A diet in which raw meat is the chief ingredient is generally associated with these medicines to advantage. Lactic acid (Hayem) and powdered talc (1 ounce a day) (Debove) have been recommended. The cure at Plombières is of great service.

## VII. HYPERTROPHIC TUBERCULOMA OF THE CÆCUM— DIAGNOSIS OF THE TUMOURS OF THE RIGHT ILIAC FOSSA.

This variety of tuberculosis commences in the cæcum or the ileo-cæcal angle, and runs a chronic course. It is often primary; it produces tumours which simulate lymphadenoma, and is often curable by operation.

This malady was described by Duguet in 1869. Surgeons have called attention to it (Terrier), and in the earliest operations they thought they were dealing with lymphosarcomata of the cæcum (Bassini, 1887; Bouilly, 1889). I may quote the works of Billroth (1891), Hartmann, Pilliet and Broca (1891), of Roux (1892), and the theses of Le Bayon (1892) and Benoît (1893).

I have collected a great number of cases in my clinical lectures,\* and I have proposed the name of **hypertrophic tuberculoma of the cæcum** for this disease.

Hartmann and Pilliet had described it under the name of "chronic tubercular typhlitis and cæcal tuberculosis." This name has the advantage of assigning the chief importance to the lesion of the cæcum. Other authors have described it under the name of "chronic ileo-cæcal tuberculosis," but this name is not exact, because it induces a belief that the lesion commences in the ileum, and then spreads to the cæcum, which is by no means always the case. Sometimes, indeed, the walls of the ileum are hypertrophied, and this fact might at first sight induce a belief in a tubercular lesion of this portion of the gut; but closer inspection shows that it is only a compensating hypertrophy caused by the stenosis of Bauhin's valve or of the cæcal cavity, and that this hypertrophy must not be confounded with a tubercular lesion. In short, hypertrophic tuberculosis does not commence, as a rule, in the ileum, but in the cæcum, close to the ileo-cæcal valve (Hartmann, Broca). The lesions are most pronounced, and the cæcal walls attain their greatest thickness at this point, so that the term **hypertrophic tuberculoma of the cæcum** seems to me to apply in the majority of cases.

The tuberculoma may remain limited to the cæcum without invading the colon, as in one of my cases. In most of the published cases, however, the cæcal tuberculoma spreads along the intestine, and finally reaches the ascending or even the transverse colon. In one of my patients, operated on by Legueu, the lesion began in the cæcum, invaded the ascending colon and part of the transverse colon. In Broca's case the lesion affected the colon, the mucous membrane being polypoid for about 3 inches. In Marion's case the lesion started in the cæcum, and spread into the ascending and transverse colons. In Bouilly's case the cæcal lesion had encroached on the ascending colon. In Roux's case the lesion started in the cæcum, and invaded the whole of the ascending and transverse colons. Hypertrophic tuberculoma does not, then, remain confined to the cæcum in the great majority of cases; it finally invades the colon. This distinction is important, as we may think that the operation should be limited to the cæcum, and then find a lesion which has invaded the ascending and the transverse colons, so that it is necessary to remove 10 or 12 inches of bowel.

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\* Dieulafoy, "Tuberculome Hypertrophique du Cæcum; Diagnostic des Tumeurs de la Fosse Iliaque Droite" (*Clinique Médicale de l'Hôtel-Dieu*, 1903, 14<sup>me</sup> et 15<sup>me</sup> leçons).



**Pathological Anatomy.**—Let us suppose a case in which the surgeon has just performed the operation. It is often tedious because the tumour is adherent to the organs in the iliac fossa, to the peritoneum, or to the anterior abdominal wall; chains of glands start from the tumour and reach towards the spine or spread out into the iliac fossa.

The tumour at first sight might be taken for a lymphosarcoma or cancer, on account of its external and also of its internal appearance. When we handled the tumour removed by Bouilly, we thought it a cancer of the cæcum. At the first microscopic examination Pilliet considered the mass to be a lymphosarcoma, and it was only later, on a second examination, that he recognized the case as hypertrophic tuberculoma. In a case reported by Chavannaz and Carrière they thought of cancer of the cæcum, even when they saw the tumour. The tubercular nature of the tumour was shown later on microscopic examination. Dr. Antipas recently sent me the cæcum of a patient whom he had cured by operation. According to his idea, it was a case of hypertrophic tuberculoma. We thought that it was a lymphosarcoma, but the microscopic examination made by Nattan-Larrier showed that it was really a tuberculoma with bacilli. It is probable that many cases formerly called cancer of the cæcum were really cases of hypertrophic tuberculoma.

The tumour is formed by the cæcum, which is much thickened, nodular, surrounded by caseous glands, and embedded in a thick mass of fibro-adipose tissue. The condition is, therefore, a true fibro-adipose perityphlitis, quite comparable to the fibro-adipose perinephritis which accompanies a tubercular kidney. This envelope was enormous in my specimens. It has been found in several cases, and Hartmann and Pilliet have given an excellent description of it in their work published in 1891.

The walls of the cæcum are sometimes enormous. They are lardaceous or fibroid, and creak under the scalpel. In my patients they were about 1 inch in thickness, while their thickness was half an inch in Marion's case and 2 inches in Gussenbauer's case. This hypertrophy is in part due to the fibroid tubercular change in the walls.

When the cæcum is opened, the internal surface projects, in the form of pillars and columns, as in one of my patients. Vegetations of a polypoid or papillomatous appearance may be seen, and resemble the tubercular vegetations found in the larynx. Ulcers are seen in places. The ileo-cæcal valve is sometimes ulcerated and destroyed, or at other times indurated and rigid, with a much constricted orifice. These lesions may cause stenosis of the intestine at the valve and in the cavity of the cæcum itself. This stenosis induces constipation and intestinal obstruction. In severe ileo-cæcal stenosis the last portion of the ileum is much dilated, and the ascending colon is contracted.

Microscopic examination shows that the thickening is due to a considerable infiltration of embryonic cells, which resembles sarcomatous tissue. This infiltration replaces the tunica mucosa, and invades the tunica muscularis, pushing aside the fibres. Tubercular granulations and large tubercles may be found in the cellular layer, but this process does not end in caseation, but in fibroid change, which makes the wall rigid and thick. Koch's bacilli were very numerous in the preparations from the cæcum of one of my patients.

Tuberculosis of the cæcum always produces enlargement of the glands. They are more or less numerous and enlarged, and indurated or caseous. They are usually found in the ileo-cæcal angle, but some may be met with in the mesentery, near the pancreas, in the supraclavicular region, and in the groin.

**Ileo-Cæcal Appendix.**—In these cases the walls of the appendix are almost always hypertrophied, and show tubercles, and yet the patient has not appendicitis in the clinical sense of the word. He has tubercular lesions in the appendix, but they do not give rise to the closed cavity, and consequently to the toxi-infectious symptoms which alone deserve the name of appendicitis.

I do not say that such a result is impossible, but I can find no mention of purulent peritonitis, remote abscesses, subphrenic empyema, purulent infection of the liver, putrid pleurisy, or toxic lesions in the kidneys, liver, and stomach, which are the appanage of appendicitis. An individual may, therefore, have tubercular lesions in the cæcum, and the appendix may be involved in the tubercular mass, but the terrible effects of appendicitis do not occur.

What is true of hypertrophic tuberculoma is equally true of ulcerative ileo-cæcal tuberculosis. I remember a phthisical patient who suffered from ulcerative tuberculosis of the intestine. The appendix was much affected by tubercular lesions, but they were parietal, and had not caused the formation of a closed cavity. Furthermore, the patient had never had any symptoms of appendicitis, and the histological and bacteriological examinations confirmed the absence of any toxi-infectious focus in the appendix. In other words, tuberculosis of the appendix and appendicitis are two very different things, and it would be wrong to include them in one classification. Tuberculosis of the walls of the appendix is fairly common, whilst tubercular appendicitis is exceedingly rare. This is also the opinion of Letulle, whose authority in this matter is great. As a general rule, the lesions of the appendix (tuberculosis, actinomycosis, cancer) which remain limited to the walls of the appendix do not cause appendicitis. These lesions may also be present in the tissues around the appendix, but they are incapable of poisoning the system, like the focus of appendicitis, which is



closed, and contains organisms. It is certain that patients suffering from hypertrophic tuberculoma of the cæcum **do not succumb to appendicitis**, and the prognosis is at least free from this complication.

**Clinical Cases.**—Two years ago I admitted into the Hôtel-Dieu a woman, of thirty-nine years of age, who had suffered for eighteen months from chronic diarrhœa and acute abdominal pains, especially in the right iliac region. From the commencement of the disease she had passed six or eight stools daily. The diarrhœa was not always preceded by colic, and had no special characters, as blood, mucus, and membranes were not present.

The pains were exceedingly acute and continuous or paroxysmal, but we noticed no special time of onset, which might have helped us to localize the intestinal lesion. They were most marked in the right iliac fossa.

She did not look ill, in spite of the duration of the disease. Nevertheless, she had lost flesh for the past two months, and the diarrhœa had been incessant. Treatment had given no relief.

What disease had caused so much pain and diarrhœa for eighteen months? There was no fever; the uterus and its adnexa were normal. Examination of the abdomen disclosed a tumour of the size and shape of a pear in the right iliac fossa. The tumour, which was painful on pressure, was fairly mobile and indurated, but not nodular. Its lower portion was wider, and reached nearly to Poupart's ligament, and its upper more narrow portion reached up in the direction of the ascending colon. Internally it did not reach the middle line. These signs and this localization being given, we could think only of the cæcum. It was still necessary to know its nature, because many tumours occur in the right iliac fossa.

I diagnosed hypertrophic tuberculoma of the cæcum, and not cancer, because the wasting was only of two months' duration. Furthermore, the sero-diagnosis of tuberculosis was positive, and confirmed the clinical diagnosis. The lungs were sound, and the case was, therefore, primary tuberculoma of the cæcum. Under these conditions, operation seemed to me to be imperative, and I requested Legueu to perform it.

An incision 6 inches long was made at the outer border of the rectus muscle. When the peritoneum was opened, the indurated portion of the intestine was exposed. Examination showed that the chief trouble was in the cæcum, which formed a large tumour. Legueu also found that the induration of the intestinal wall extended as far as the hepatic flexure. The ileum was healthy.

The diseased intestine was removed, and the two ends of healthy bowel (ileum and transverse colon) were joined by end-to-end anastomosis.

Some enlarged glands were removed from the mesentery. A catgut suture was applied to the free edge of the mesentery, so as to unite the two layers. The operation was concluded by the suture of the abdominal wall and the insertion of a single drainage-tube.

The patient had a motion on the third day. We commenced to feed her with milk and broth. The drainage-tube was removed, and from the eighth day onwards the progress was rapid.

Five weeks after the operation she was fat and well. Some 10 inches of intestine were resected, thus removing a tubercular lesion of eighteen months' duration, which would have had a fatal ending. At the present time her appetite is excellent, and her digestion is normal. The abdominal pain and the diarrhœa have completely disappeared.

Eight months later she came back to see us at the Hôtel-Dieu. She had gained about 20 pounds in weight, and had not felt the least malaise since the operation—a proof that the lesion had been taken away *in toto*. Although the cæcum, ileo-cæcal

valve, ascending colon, and a portion of the transverse colon were removed, her digestion was as regular as with a normal intestine.

Immediately after the operation we examined the specimen. The portion resected by Leguen comprised the end of the ileum, the cæcum, the ascending colon, the hepatic flexure, and a part of the transverse colon—in all, about 10 inches of gut. The following figure shows the intestinal lesion in detail.

The ileum was quite normal.

The cæcum formed a large tumour, the size of which has been increased by an adherent fibro-fatty sheath (*f*). Its nodular surface gave at first sight the impression of a sarcoma. Caseous glands were found at various points.

After opening the specimen we found the following details :

The walls of the cæcum were hard and fibroid, being 1 inch thick at the bottom (*e*), and  $\frac{2}{5}$  inch a little higher. The walls of the ascending colon were  $\frac{1}{5}$  inch thick, and those of the hepatic flexure (*b*) were  $\frac{1}{8}$  inch. The lesion began at the bottom of the cæcum, and became less marked as it extended towards the colon. The cæcal cavity was constricted by the thickening of the walls. Concentric hypertrophy was present.

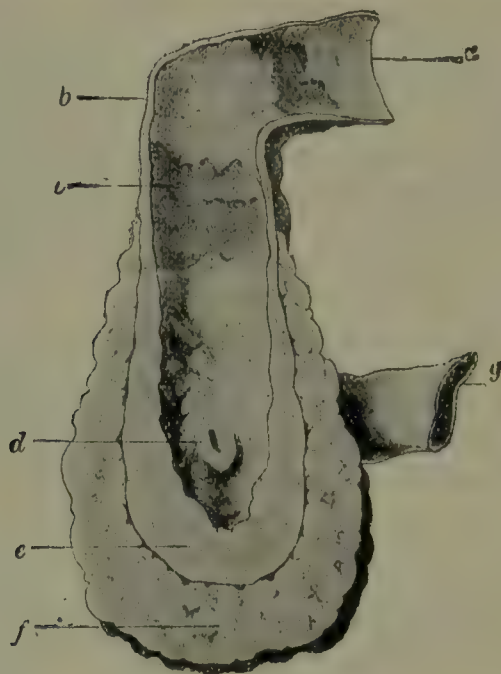


FIG. 44.

The internal surface of the cæcum was irregularly corrugated. The ileo-cæcal valve (*d*) was indurated, open, and constricted. Two superficial ulcers were found—one (*c*) in the ascending colon, and the other in the transverse colon. The appendix was slightly indurated and hypertrophied.

The histological examination by Nattan-Larrier showed the following details :

The hypertrophy of the walls of the cæcum was confined principally to the sub-peritoneal cellulo-fibrous layer and to the submucous layer, where the tubercular lesions attained their maximum.

The epithelium of the surface was preserved everywhere without a trace of ulceration. The glands of the mucous membrane were very much hypertrophied. In the submucous layer marked infiltration of leucocytes and a considerable number of small isolated or agglomerated tubercles were present. The muscular layer was separated by abundant oedema, and embryonic infiltration was found at certain points. The serous layer was very rich in adipose tissue, and patches of very dense fibrous tissue and some small tubercles were also found. Films on slides revealed the presence of Koch's



bacilli. The histological examination of the appendix showed non-tubercular hypertrophy of the walls, with obliteration of the canal throughout its whole length.

Another patient suffered from continual diarrhoea, and could not take food. He had lost 50 pounds in weight in three years. In the right iliac fossa he had a tumour which was easily felt, because the walls of the abdomen were very thin. Examination of the region did not cause pain. The tumour was of ligneous consistency, and as big as a large orange. It seemed to be adherent to the pelvis. It was 2 inches from the linea alba, and 3 inches from the false ribs. Below, it did not reach Poupart's ligament. The inguinal glands were hard, but not painful.

The tumour was evidently in the cæcum, but how were we to know whether it was cancerous or tubercular? Induration of the tumour, glands in the groin, loss of flesh, and cachexia were not sufficient to establish the differential diagnosis. The course of the disease, however, was important. The patient had been ill for three years. At this period an abundant bloody diarrhoea came on, and had never disappeared. It came on immediately after the ingestion of food. Diarrhoea, abdominal pains, anorexia, loss of flesh, and cachexia might be present in tuberculosis or in cancer of the cæcum. I nevertheless put aside the idea of cancer, on account of the long duration of the disease. Besides, we had another argument in favour of tuberculosis—the patient was suffering from pulmonary tuberculosis. The lung trouble was not the primary disease, because the patient had been examined several times at the Hôtel-Dieu, and the lungs had always been found healthy. Two years and a half before he was treated for intestinal troubles, but we found nothing the matter with his lungs. As the disease became worse, he returned for advice a year later, and we again found intestinal symptoms, but no indication of phthisis. I therefore diagnosed hypertrophic tuberculoma of the cæcum, with secondary disease in the lungs.

If this man had come to us earlier, before the onset of the lung trouble and of the cachexia, I should not have hesitated as to operation. In his actual condition there could be no thought of surgical intervention. We tried in vain to feed the patient up, but he succumbed six weeks after admission.

The results of the post-mortem examination were as follows: It was a case of hypertrophic tuberculoma of the cæcum. There was a nodular tumour of the size of an orange, which at first sight simulated a lymphosarcoma. The cæcal tumour was enlarged by a fibro-fatty covering. When this envelope was removed, the walls of the cæcum (*b*) were greatly hypertrophied, fibroid, and lardaceous, as will be seen from Fig. 45. The lesion was absolutely confined to the cæcum. The ileum (*d*) and the colon (*a*) were not affected. The delimitation of the lesion was as clear internally as externally. A large ulcer (*c*) had destroyed the valve, and occupied the bottom of the cæcal cavity. Elsewhere the internal surface of the cæcum was closely set with folds, forming bridles, pillars, and columns. Glands were not numerous. The appendix, though thickened and embedded in the fatty tissue around the cæcum, had preserved its normal calibre. In the histological preparations Koch's bacilli were found in abundance.

Bouilly's case (the first case operated on in France): A woman had been suffering from gastro-intestinal troubles for five years. An immovable painful tumour of the size of an orange was found in the right iliac fossa, and was supposed to be an ileo-cæcal cancer. The tumour was removed. On examining the specimen, thickening of the walls was found; the ileo-cæcal valve was unrecognizable, and the mucous membrane of the cæcum was covered with vegetations, which projected into the cavity. The case was, therefore, a tuberculoma. The appendix was large, but the canal was patent. Glands were present at the junction of the ileum and of the cæcum. The patient was in good health four years after this operation.

One of Billroth's cases refers to a child of ten years of age who had suffered from intestinal troubles for two years. A tumour of the size of an apple, which was tender

on pressure, mobile in all directions, and of a ligneous consistency, was felt in the caecal region. In order to clear up the diagnosis, an injection of tuberculin was given, and caused a reaction of  $104^{\circ}$  F. The case, therefore, was held to be tubercular. An operation was performed. The tumour was 4 inches in length; the ileo-caecal valve was much constricted; the mucous membrane of the caecum was covered with polypoid vegetations, surrounded by a callous zone. Microscopic examination showed an infiltration of small cells, grouped in tubercles and giant cells. The operation was followed by recovery.

**Description.**—The onset is generally slow and insidious. The patient complains of abdominal pains, with alternate diarrhoea and constipation. The pains, which are at times very sharp, may affect the form of colic, and be most severe in the right iliac fossa. The diarrhoea is sometimes intermittent, or at other times as obstinate as in ordinary tubercular enteritis. Blood is rarely present, contrary to what is noticed in ordinary tuberculosis of the intestine. During this first period the patient loses but little flesh.

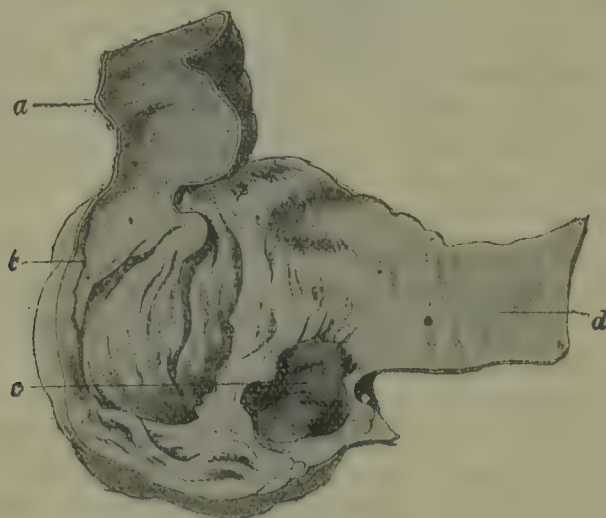


FIG. 45.

The stationary stage, during which the lesions become definite, varies from a few months to several years. During this period the symptoms are almost invariable, and comprise intestinal troubles, pains predominating in the right iliac fossa, diarrhoea, constipation, vomiting, and loss of flesh. The examination should be preceded by an aperient. Exploration of the abdomen reveals hyperæsthesia in the right iliac fossa, where an induration or a tumour, varying in size from a nut to an orange, is found. The induration sometimes appears diffuse, uneven, and nodular; at other times we find a tumour fairly circumscribed, mobile in every direction or only in a transverse direction, or immobile, on account of adhesions in the iliac fossa and to the abdominal wall. One or more fistulous tracks may open on the skin of the iliac region. What does the general examination of the patient teach us? In a woman vaginal touch reveals the integrity of the pelvic organs. The lungs are generally healthy, and pulmonary tuberculosis has



been noticed only **five times in thirty cases** (Benoît), and even then the signs were fairly discrete. Fever is, as a rule, absent, or only supervenes in acute complications. Appendicitis is practically never seen. The gravity of the prognosis does not arise solely from the tuberculoma, but is especially dependent on primary or secondary pulmonary tuberculosis.

**Diagnosis.**—The diagnosis of hypertrophic tuberculoma of the cæcum comprises the diagnosis of tumours of the right iliac fossa. During the first phase of the disease, when there is no tumour, the diagnosis is extremely difficult, because it rests on two symptoms alone—pain and diarrhoea, which are common to several lesions in this region. Later, when a tumour has formed in the right iliac region, the diagnosis is not less difficult, because tumours of every kind may occupy this region. Nevertheless, let us discuss tumours arising *in situ*, without troubling ourselves about those which have come from neighbouring regions.

**1. Glandular Lesions of the Right Iliac Fossa.**—I saw a young girl who had suffered from intestinal troubles for several months past. She complained of abdominal pains, principally in the right iliac fossa. At various times the crises, which were accompanied by vomiting, had awakened the idea of appendicitis. Diarrhoea was frequent, and, indeed, formed the chief symptom in the interval between the crises. Melæna had never been noticed. Digestion was painful, and alimentation imperfect. She had no fever, but she often kept her bed, because any movement brought on the abdominal pains.

On exploring the abdomen I found a tumour of the size of a nut in the right iliac fossa. This tumour was indurated, mobile, and very painful, and from its situation seemed to form part of the cæcum. The girl's father had died from tuberculosis, and though she showed no sign of disease in the lung, she looked like a consumptive. I had grave doubts about the diagnosis. I thought of the possibility of hypertrophic tuberculoma of the cæcum, and advised the family to call in Gérard Marchant.

He gave the following report at the Société de Chirurgie: "When the peritoneum was opened and the cæcum laid bare, I was especially struck by the existence of two juxtaposed masses of glands, one of which was larger than the other, on the cæcum, near its internal surface and close to its junction with the small intestine—that is to say, where normal anatomy shows glands. One of these glands showed a yellowish point near its surface. No adhesions were present, and I was able to enucleate these glands without disturbing the coats of the cæcum. Though the appendix appeared healthy, it was resected."

The glands and the appendix were examined. The appendix showed no lesion. The glands were caseous. The operation was followed by rapid improvement, the pains and the diarrhoea disappeared, and five years later I saw the patient, in perfect health.

I quote two more cases of **precæcal adenitis**, reported by Gérard Marchant: On November 14, 1899, he operated on a young girl who suffered every two or three months from sharp pain in the right iliac fossa, without vomiting. The pain, as a rule, subsided after a few hours' rest. The first severe crisis occurred in July, 1899, with high fever, vomiting which lasted four days, and violent pains at the usual spot. This crisis lasted for a fortnight, and yielded to ice, opium, and starvation diet. The patient, however, always felt pain in the right iliac fossa. The second crisis occurred during the night of November 2—nausea, lipothymia, and extreme distress, but no fever. Ten days later Marchant noticed a well-marked swelling in the cæcal region. He thought it was

a case of appendicitis, but he was surprised at the operation to find the appendix normal. On the anterior surface of the cæcum, however, there was a large suppurating gland, which was so adherent to the cæcum that extirpation had to be abandoned. The gland was treated by scraping and cauterization. The septic focus was then isolated from the general peritoneal cavity by suture of the cæcum to the edge of the peritoneum. The patient had a sister suffering from coxalgia, and a brother who was phthisical. The operation resulted in cure. Histological examination of the appendix showed follicular inflammation of the walls, but there was no appendicitis.

In another case reported by Gérard Marchant appendicitis had been diagnosed. There were persistent pains over the cæcum and rapid loss of flesh.

The operation showed the integrity of the appendix, which was, however, resected; but the operator found a quantity of small, hyperplastic, indurated glands, which were not limited to the cæcum, but were also present in the meso-appendix, the great omentum, and the right layer of the mesentery. None of these glands were touched. The abdomen was sutured, and the patient got better under the influence of general treatment.

In his commentary on these cases Marchant thinks justly that precæcal adenitis may be consequent on pre-existing tubercular or infectious lesions of the cæcum, or of the ileo-cæcal valve. I think that precæcal adenitis may become the chief lesion, the cæcal or ileo-cæcal lesion remaining *in statu quo*. The history of cervical or mediastinal tubercular adenitis furnishes us with similar examples. An apparently insignificant tubercular lesion in an organ may cause infection in the adjacent or remote lymphatic glands. Adenoid growths and tonsillar lesions, which I have already described under Masked Tuberculosis of the Tonsils,\* may lead to enlargement of the glands in the neck, whilst the primary lesion becomes stationary, and passes almost unnoticed.

Similar considerations seem to me to be applicable to cæcal tuberculosis. In studying the classical form of hypertrophic tuberculoma of the cæcum, we have seen that it is always accompanied by adenitis, which may spread far, though its seat of election is the anterior surface of the cæcum and the ileo-cæcal junction. Now, side by side with the classical form which is revealed by the symptoms enumerated in this chapter, we may ask whether a masked form of cæcal tuberculosis may not occur, and declare itself, not by the classical symptoms, but by pain and caseation or suppuration of the glands in the right iliac fossa, attacking especially those on the anterior surface of the cæcum and the ileo-cæcal junction.

The enlarged glands, which are more or less appreciable on palpation, according as they form a tumour or not, occupy the cæco-appendicular region, in which chronic lesions of the appendix and the cæcum are also found. Besides, these precæcal cases borrow symptoms from the neighbouring organs (peritoneum and intestine), and thus cause difficulty in diagnosis. They are accompanied by continuous or paroxysmal pains,

\* Dieulafoy, "Tuberculose Larvée des Trois Amygdales" (*Académie de Médecine*, séance du 30 Avril, 1895).



which somewhat resemble the pains of chronic appendicitis. They cause diarrhœa, which diverts the attention to the intestine, and they form a painful tumour in the right iliac fossa, just as hypertrophic tuberculoma of the cæcum does, so that we find three kinds of lesions—viz., cæcal tuberculoma, chronic appendicitis, and precæcal adenopathy. They occupy the same region, and have common symptoms. Differential diagnosis between these three lesions is, then, extremely difficult before the operation. The differential diagnosis is not of prime importance, because in all three cases surgical intervention is imperative.

**2. Hypertrophic Tuberculosis of the Ileum.**—We must next consider the diagnosis between hypertrophic tuberculoma of the cæcum and hypertrophic tuberculosis of the small intestine in the right iliac region. Bernay classes tubercular stenoses of the small intestine in three groups, according as the constriction is fibrous, cicatricial, or hypertrophic. The fibrous and cicatricial constrictions are almost always found in the upper three-quarters of the small intestine, whereas the hypertrophic constriction is principally met with in the lower fourth. This last form is much less common, since it only existed eight times in seventy cases of tubercular constriction. Even then it does not always extend to the cæcum.

A very clear case has been published by Tuffier.

A woman who was neither consumptive nor syphilitic had suffered from colic for about two years. The attacks of colic made their appearance four or five hours after meals, and ended in liquid stools without a trace of melæna. Although her appetite was good, she ate little, for fear of the intestinal pains, and she had lost flesh considerably. On exploring the abdomen a tumour of the size of a small tangerine orange was found in the right iliac fossa. The tumour was painless, mobile, and of a renitent consistency. The diagnosis of tubercular stenosis of the small intestine was made, and Tuffier operated. It turned out to be a case of hypertrophic tuberculosis of the small intestine. Thirteen inches of the intestine were resected, and in a few weeks later the patient had recovered. On examining the specimen it was found that the tuberculosis was at once ulcerative, hypertrophic, and constricting. The stenosis was the result of fungoid enteritis. The histological preparations showed considerable hypertrophy of the different layers of the intestine, but principally of the submucous one. In the deep layers of the submucosa large tubercular follicles with giant cells and tubercle bacilli were found.

In other words, **hypertrophic tuberculosis of the ileum** may give rise to a tumour resembling hypertrophic tuberculoma of the cæcum.

**3. Chronic Appendicitis.**—Let us now consider tumours of the right iliac fossa consequent on previous appendicitis. I do not speak here of the iliac abscesses which occur in suppurative appendicitis, but I allude to fibroid masses of slow and progressive growth and to tumours of a neoplastic aspect, which form a chapter in chronic appendicitis as yet but little studied.

In its extra-appendicular migration the infection may remain limited to the right iliac fossa, and does not always cause the formation of pus (suppurative perityphlitis). It sometimes gives rise to an inflammatory tumour, or produces false membranes and adhesions which have the look and the consistency of fibroid and lardaceous tissue. This process may reach the mesentery, and extend beyond. "Fibrous, hard, resistant, and irregularly distributed bands fix the appendix to the cæcum, ileum, mesentery, or iliac peritoneum." Thick adhesions may bend the termination of the small intestine at its junction with the cæcum, and compress the ileo-cæcal valve. The cæcum, embedded in the inflammatory tissue, is at times unrecognizable, the portion of the large intestine being replaced by a mass of fibrous tissue, in which ileum and ascending colon are matted together. On palpation of the right iliac fossa, a pericæcal or ileo-cæcal tumour may be felt.

This extra-appendicular process is slow in its evolution, and the symptoms closely resemble those of hypertrophic tuberculoma of the cæcum. The patient complains of heaviness, twitchings, and pains in the right iliac fossa. These pains may only amount to a feeling of numbness, or may occur in acute crises analogous to those of appendicitis. They then can only be due to bridles and to adhesions. Here, as in fibrous pericholecystitis, the adhesions may give rise to attacks of severe pain. The patient sometimes complains of dyspepsia, anorexia, or vomiting. Diarrhoea is frequent; constipation may be obstinate, and accompanied by symptoms of intestinal occlusion. The patient grows thinner. I have also seen reflex cardiac troubles and palpitation, which may obscure the diagnosis, by directing the attention to the heart, while the trouble really arises from the appendicular lesion.

When we examine the right iliac fossa, we may find an induration or a painful tumour, which on removal presents at first sight the appearance of a neoplasm. This tumour occupies the situation of cæcal or pericæcal tumours. If the onset of the disease has been clearly characterized by one or more attacks of appendicitis, chronic lesions of the appendix will be thought of, and the diagnosis made. If this guide is absent, we can only make a provisional diagnosis of cæcal tuberculoma or cancer of the cæcum; but in any case we advise an operation. The surgeon may find a shapeless mass, in which the ileum and the ascending colon are matted together; kinking of the ileum at its entrance into the cæcum, constriction of the ileo-cæcal valve, hypertrophic infiltration of the intestinal walls, caseous magma, purulent collections, adhesions, enlarged glands, or a neoplastic tumour, which all result from previous appendicitis. The operation is often successful. The above description shows how chronic lesions consequent on appendicitis give rise to a tumour in the right iliac fossa.



**4. So-called Inflammatory Tumours.**—Besides the tumours due to hypertrophic tuberculoma of the cæcum and ileum, to pericæcal adenitis, and to sequelæ of appendicitis, there are other tumours of the right iliac fossa—much rarer, it is true—the cause of which escapes us. We find neither tuberculosis nor appendicitis, and for want of a better name the ileo-cæcal tumour is called “purely inflammatory.” In this acceptation of the term, Marchant and Demoulin quote the cases of Hartmann, Boiffin, and Julliard; but some of these cases leave a doubt as to their “purely inflammatory” origin. Thus, in Boiffin’s case there was no examination of the tumour. It therefore cannot be said that it was not a case of tuberculoma. In Julliard’s case “the appendix was deformed and almost unrecognizable,” which leads us to suppose that the so-called inflammatory tumour had its origin in an infection of the appendix. Marchant has cited a case of these so-called inflammatory tumours. A lateral tumour of the cæcum was present, and was of the size of a nut. The appendix was healthy. The histological examination proved that this was neither a neoplasm, tuberculosis, nor lymphadenoma, but simply a so-called inflammatory thickening of the submucous coat.

Schwartz has communicated the following case :

A young woman was suffering from a tumour in the right iliac fossa. This tumour, which had been painful for a long time, was hard and relatively mobile. An operation was performed, and a tumour of the size of the fist was removed. It was formed by the cæcum and the terminal portion of the ileum. On incising the specimen, the resistance of the tissues was marked. The cæcum was hypertrophied to such an extent that its walls measured 1 inch. The ileo-cæcal valve was thickened, but not constricted. The mucous membrane was normal, and showed no trace of ulceration. This tumour was, so it appears, neither tubercular nor cancerous. It had not an appendicular origin. In what category should it be placed? The histological examination by Cornil revealed considerable hypertrophy of the musculature of the intestine.

**5. Actinomycosis.**—Actinomycosis of the appendix and cæcum may cause a tumour in the right iliac fossa. I had recently the opportunity of seeing a case with Widal and Segond.

The patient for a year past had had intermittent attacks which resembled recurrent appendicitis. They began with pain, tympanites, and obstinate constipation. The pains, though general over the whole abdomen, had been most severe in the right iliac region. In spite of the intensity of the pain, there had been neither vomiting nor fever. These attacks lasted for some days, and ended without leaving any trace of pain in the cæco-appendicular region. In the interval between these acute episodes the health had been good.

Though the attacks somewhat resembled appendicitis, they differed, nevertheless, in several points. Appendicitis is not ushered in, as a rule, by tympanites, and, in addition, we never see a severe attack of appendicitis which is not accompanied by fever and vomiting. Finally, very severe attacks of appendicitis leave behind for some time a painful feeling in the right iliac fossa. The patient showed an absence of all these symptoms, and I was, therefore, unwilling to admit appendicitis. I had thought of typhlo-colitis, but this diagnosis had to be abandoned, for in these cases the

attacks are followed by the expulsion of mucus, membranes, or intestinal sand. Several hypotheses were, therefore, possible, and preæcal adenitis, tuberculosis, or cancer was suggested. I admit that I did not think of actinomycosis.

A tumour, however, appeared in the ileo-cæcal region. The tumour, which was hard, elongated, mobile, and tender on pressure, shed no light on the diagnosis, and it might have been a case of cancer. The general condition remained good. Surgical intervention became inevitable. Second operated, and removed an enormous appendix, surrounded by membranes and adhesions, though no traces of suppuration or peritonitis were found. The walls of the appendix were very thick, hard, and lardaceous. The mucous membrane was healthy, and the lesion did not deserve the name of appendicitis in the true sense of the word. Microscopic examination showed actinomycosis of the appendix.

Actinomycosis of the appendix has been accurately described in recent years, notably in the treatise of Poncet and Bérard. During the first phase, which lasts from one to two years, the disease closely resembles recurrent appendicitis. The patient suffers from continuous or paroxysmal pains, which are most marked in the right iliac fossa. There are attacks of gastro-intestinal tympanites and constipation, or more frequently diarrhœa, with blood-stained stools and tenesmus. Later, in the cæco-appendicular region a ligneous swelling or an induration, giving the sensation of a deep fibroma, with ill-defined edges, is felt. This condition may remain stationary for months, until the actinomycosis comes in contact with the peritoneum and the abdominal wall, which it infiltrates like a breast-plate, when softening, ulceration, and fistulæ supervene.

It must be admitted that the diagnosis is most difficult in the first phase of the disease. There are only two cardinal symptoms to serve as guides—viz., the localization of the pain and, later, the appearance of the tumour. Chronic disease of the appendix, early cæcal tuberculoma, or some other lesion, is then thought of, but nothing as yet points to the existence of actinomycosis. In a case of chronic diarrhœa the parasite may be found in the stools.

**6. Cancer of the Cæcum.**—We must make the diagnosis of tuberculoma of the cæcum from ileo-cæcal cancer. The patient is more or less cachectic, and has been suffering from abdominal pains and diarrhœa for a long time. A small, irregular, mobile tumour is felt in the right iliac fossa. Has the patient cancer or chronic tuberculosis? Upon what signs are we to base our diagnosis? Obstinate constipation and intestinal obstruction may exist in both cases. Diarrhœa, with or without intestinal hæmorrhage, is also common in each condition. Nevertheless, melæna is very rare in hypertrophic tuberculoma, and more frequent in cancer. Spontaneous or provoked pain does not furnish adequate information. The dimensions, form, and mobility or immobility of the tumour tell us nothing positive. Indurated and enlarged glands in the groin and in the supraclavicular hollow may be present in both cases. The appearance of phlegmasia alba dolens may also



be seen in either condition. The search for pulmonary tuberculosis is only of secondary interest, for five times out of six chronic tuberculosis of the cæcum is primary and local, supervening in patients who are free from any other tubercular lesion. In favour of tuberculosis there is, then, only the long duration of the disease, which is more compatible with the hypothesis of tuberculoma, and the recognition of the bacillus in the fæces. Billroth once made a correct diagnosis by inducing a reaction of 104° F. with tuberculin, but I would rather have recourse to sero-diagnosis.

7. Finally, we may see a patient who has one or more discharging fistulæ in the iliac region. Whence comes the pus, and what is the nature of the lesion? Have we to do with a bone lesion or not? Even if the tubercle bacillus be found in the pus, and even if the tubercular nature of the lesion be proved by experimental means or by the reaction of tuberculin, we should know only one thing—viz., that we have to deal with a tubercular lesion. The passage of fæcal matter through the fistulæ would remove all doubts, and prove that the cæcum is the origin of the lesions.

**Termination.**—Chronic tuberculosis of the cæcum may end in different ways. It may recover without operation by a curative fibrous process, analogous to the curative fibroid process in pulmonary tuberculosis. In some cases the constriction of the ileo-cæcal valve and of the cæcum attains such a degree that the patient succumbs to intestinal obstruction. Iliac phlegmon and fistulæ, burrowing into the ischio-rectal fossa, the edge of the anus, or the right groin, are possible complications.

**Ætiology.**—Chronic tuberculosis of the cæcum, as I have already remarked, is most often primary. It is a local tuberculosis such as we find in the joints or bones. All the hypotheses made to explain primary tuberculosis of the digestive passages may here find their place. It is probable that the ileo-cæcal region, by its natural constriction, offers a favourable asylum for the tubercle bacillus.

**Treatment.**—The prognosis of hypertrophic tuberculoma of the cæcum has been completely modified during the past few years. A patient suffering from cæcal tuberculosis was formerly considered to be stricken with a fatal disease. At present the mischief can often be cured by operation. Cure, however, can only be obtained on certain conditions. It is necessary to operate as early as possible, before the subject is cachectic, and as soon as the disease is recognized or even suspected. The coexistence of pulmonary tuberculosis makes the prognosis far worse, because this complication is a counter-indication against operation. Do not let us forget, however, that pulmonary tuberculosis is rarely the source of tuberculosis of the cæcum. It is sometimes the consequence thereof, which is another reason for not delaying surgical intervention too long. Timely operation gives numerous successes.

## VIII. ANO-RECTAL TUBERCULOSIS.

**Lupus of the Anal Region.**—Lupus, which is relatively frequent in the face, is extremely rare at the anus. It is generally associated with lupus of the genital organs in women. In two cases lupus developed at the external orifice of a fistula. One of these cases, communicated by Besnier, is described as follows: The lupus consisted of an extensive cicatricial surface, fibroid at the centre, papillomatous at the circumference, and bordered with ulcerated tubercles, which formed large festoons around it. The diagnosis of lupus could not be doubted: chronic cutaneous affection evolving *in situ* for the last eighteen years around an anal fistula; fibroid cicatrization of the points first attacked, and peripheral extension by slowly growing crops of tubercles.

Neither syphiloderma nor epithelioma runs this course, nor have they the slowness of this process. One objective point alone might have pointed to syphilis, and that was the polycyclical border; but this formation is met with in some cases of lupus.

**Warty Tuberculosis of the Anus.**—It is probable that the warty form of tuberculosis was formerly confounded with epithelioma and papilloma. It has been recognized since the work of Hartmann, Routier, and Toupet. The lesion commences around the anus, and encroaches on the buttock in the shape of a patch studded with non-ulcerative mammillæ and crusts. Between the mammillæ, eroded oozing furrows are seen. The inguinal glands are sometimes healthy, sometimes enlarged. Histological examination reveals giant cells in the midst of the cell mass, and bacteriological examination proves the presence of the tubercle bacillus. Except by bacteriological examination or inoculation of guinea-pigs, it is scarcely possible to differentiate warty tuberculosis from papilloma or epithelioma.

**Anal Tubercular Ulcerations.**—Hartmann has collected about thirty cases. After several months of itching, oozing, and pains on defæcation, a superficial sore appears around the anus, and forms an ulcerated scrofuloma *en cuirasse*. The ulcer is confined to the edge of the anus, and does not encroach on the buttocks, but it often extends inside the anal canal as far as the lower extremity of the rectum. The ulceration bleeds slightly; its edges are often polycyclical, sometimes undermined, sometimes clean-cut. The floor of the ulcer is anfractuous, granulating, and often studded with yellow granules. The ulcer secretes a muco-purulent liquid, and is sometimes covered with a crust. Prominent tubercles may be seen around the ulcerated surface.

On examining the anus with a speculum, lesions are seen on the mucous



membrane analogous to those of the skin. Digital examination reveals the limits of the disease. The inguinal glands are almost always enlarged. These ulcerations are the more painful because the pain is repeatedly excited by the passage of fæcal matter, walking, and rubbing. In some cases, however, they are almost painless.

Tubercular ulceration of the anus has an exceedingly slow course, and shows no tendency to cicatrization. It coincides sometimes with tubercular ulcerations of the other parts of the intestine. The search for bacilli, in the secretions of the ulcerated surface is the most certain means of diagnosis.

**Abscess and Anal Fistula.**—Fistula of the anus of tubercular origin always follows on an abscess, and is not the result of burrowing ulceration. In twelve abscesses of the anal region Hartmann and Lieffring found the tubercle bacillus, with other microbes, in seven cases.

The subcutaneous tubercular abscess opens externally on the skin, or internally in the ano-rectal canal, but it may open both on the skin and into the intestine. If the track formed by the abscess becomes persistent, a fistula results. A fistula is a canalicular ulcer which shows no spontaneous tendency to heal. It commences when the process of repair ceases. A complete fistula supposes a passage with two orifices—the one cutaneous, the other intestinal. The fistula is said to be a blind external one if there is only a cutaneous opening, while it is said to be a blind internal one if there is only an intestinal opening.

The **symptoms** of fistula are preceded by those of abscess. In the external fistula the disease only causes pruritus, oozing, suppuration, and soiling of the underclothing. In the case of a complete fistula soiling of the linen by fæcal matter, fæcal odour, and tenesmus, which is sometimes painful, complicate the situation. At certain times the patient has attacks of inflammation. As soon as there is any obstruction to the exit of the infective liquid, and as soon as the fistulous passage is transformed into a closed cavity, the microbes increase in virulence (*coli bacillus*, *streptococcus*). These “revivals” of the fistula cause fresh abscesses, with fever, pain in the anal region, etc.

In tubercular patients anal fistula exists in the proportion of 12 per cent. (Cripps), 14 per cent. (Allingham), 16 per cent. (Geffrart), 50 per cent. (Hartmann), and 15 per cent. (personal observations). The abscess and the fistula which results generally develop during the course of pulmonary tuberculosis or at an advanced stage of the disease. In some cases, however, the abscess seems to precede the disease in the lung, and it is by no means rare to find people, who have been coughing for years, under the impression that they are suffering from simple emphysema with bronchial catarrh, when the appearance of a tubercular abscess of the anus reveals

the true nature of the bronchitis. In a tubercular family it is not rare to find one of the members—perhaps one of the children—with a tubercular abscess of the anus as the only sign of tuberculosis.

An important question presents itself in regard to these fistulæ: Can they be operated upon without harm to the patient? Generally speaking, anal fistula in tubercular patients has been looked upon as a sort of conduit which must be let alone, for fear of causing recrudescence in the pulmonary disease. This assertion is exaggerated, but yet, before we come to a decision and operate on the fistula, we must take careful stock of the patient's condition. The operation may be performed unless the pulmonary lesions are too far advanced. I am of opinion that in a patient with advanced phthisis we ought to proscribe an operation; in all other cases it should be performed, for I have never seen any serious inconvenience result.

#### IX. SIMPLE ULCER OF THE DUODENUM—PERFORATION— SUPERACUTE PERITONITIS.

**Pathological Anatomy.**—Simple ulcer of the duodenum closely resembles ulcer of the stomach and œsophagus. It usually affects the first portion of the duodenum, the anterior more often than the posterior surface, and at times encroaches on the pylorus. When there are several ulcers, they become fused, and give an irregular form to the ulceration. It is by no means rare to find both ulcer of the duodenum and of the stomach. The pathogenesis, course, cicatrization, and perforation of the duodenal ulcer, as well as the pathogenesis of hæmorrhage and peritonitis, are in all points comparable with the same processes in gastric ulcer. Adhesions and fistulæ may be found between the duodenum and the neighbouring organs.

**Symptoms.**—Simple ulcer of the duodenum generally arises in an almost latent fashion, like certain ulcers of the stomach. It is even more often latent than ulcer of the stomach. Pain, vomiting, melæna, and hæmatemesis are the usual symptoms.

The pain has much the same characteristics as have been mentioned under Ulcer of the Stomach, but it has a slightly different seat. The xiphoid and spinal points are often absent, and the pain appears two to three hours after meals in the pyloric region below the inferior edge of the liver.

The intestinal hæmorrhages may be fulminant in the case of erosion of a large vessel, such as the gastro-epiploic artery (Broussais), the pancreatico-duodenal artery (Knecht), the aorta (Stich), or the portal vein (Rayer). In ordinary cases melæna recurs at more or less close intervals, with or without pain, and is accompanied by hæmatemesis if the blood has



regurgitated into the stomach. Pallor of the face and of the body, and tendency to fainting or syncope, result from these attacks of hæmorrhage.

**Perforation of the Ulcer.**—Ulcer of the duodenum very often ends in perforation, and in 262 ulcers 181 perforations have been counted. The perforation caused general peritonitis 125 times. In other cases the peritonitis was limited by adhesions, or else the perforation had no evil result, because the liver or the pancreas formed a tampon.

Partial peritonitis and encysted abscesses due to adhesions are far less common in the case of perforating ulcer of the stomach. They may open at the umbilicus or in an intercostal space.

**Perforation of the duodenal ulcer**, with superacute peritonitis, is a terrible complication. The reader will obtain some idea of it from the following case, taken from one of my clinical lectures on perforation of simple ulcer of the duodenum :

A patient was brought into the Hôtel-Dieu, and lay groaning. He was immediately put to bed, and Charrier diagnosed acute peritonitis. The patient, with drawn face and hollow eyes, was very restless. His respiration was jerky and interrupted from pain. The abdomen was very distended, excessively painful, and resonant, except in the hypogastrium and the flanks.

He had been in good health until two days before his admission. His digestive functions had always been normal, when he was suddenly seized with very acute pain which he compared to a stab. Beausse was at once called, and found the patient writhing in agony.

Beausse immediately administered an injection of morphia, which gave the patient some relief, and he was then carried home. It was now possible to examine him. The abdomen was retracted and hard. Palpation was very painful, particularly in the right hypochondrium, whence the pain radiated towards the pit of the stomach and the spine. Deep palpation was not possible, because the abdominal walls were as hard as a board. As the pain was not marked at McBurney's point, the diagnosis of appendicitis was discarded. On the next day, Sunday, the abdomen was still painful and retracted, but the patient was much easier, as the temperature did not exceed 99° F.

During the day he passed a motion, and seemed better in the evening. The pains were less sharp, and the abdomen was still retracted. On Monday morning, however, he vomited greenish fluid, the eyes became hollow, the nose pinched, and the abdomen, which was previously retracted, now became distended. He was then brought to the Hôtel-Dieu.

The patient was taken into my ward, suffering from acute peritonitis, the pain, as at its onset, being most acute under the ribs on the right side, between the hypochondrium and epigastrium. Although the prognosis was almost hopeless, and the peritonitis was general, surgical intervention was necessary as a last resource. The operation was performed by Cazin within an hour of his admission into hospital. The peritonitis was general, and the pelvis was full of pus, but there was no appendicitis. As the operation had already lasted some time, and as the patient's condition was most serious, it was very wisely decided not to continue the exploration. The patient succumbed a few hours later.

Next morning, on arriving at the Hôtel-Dieu, I heard the details of the case, and I am sure nothing more could have been done. When a patient is attacked with acute peritonitis, he must be given the only chance of salvation, which lies in operation. It is true that we are not always successful, but I can quote desperate cases in which

surgical intervention has been a complete success. After hearing the report of the case, I gave my opinion as to the cause of the peritonitis, and I felt justified in affirming that he had died from the perforation of a latent ulcer of the duodenum or stomach.

Post-mortem examination: General peritonitis. Methodical examination of the intestine revealed the cause of the peritonitis. On the posterior surface of the duodenum, 1 inch below the pylorus, we found a simple ulcer, which had perforated at its centre.

On the intestinal surface the ulcer had a funnel-like shape, and its base measured  $\frac{2}{5}$  inch in diameter. The walls were steep, and formed two steps. At the bottom of the ulcer there was a perforation  $\frac{1}{8}$  inch in diameter, with circular, indurated, thick outlines formed by the rounded edges. On the serous surface the perforation was so clean that it seemed to have been punched out of healthy tissue. The remainder of the duodenum was absolutely normal. On the anterior surface of the stomach the radiating cicatrix of an old healed ulcer was present.

To sum up, the patient succumbed in less than three days from superacute peritonitis, due to perforation of a duodenal ulcer which had been absolutely latent.

**Analysis of the Symptoms.**—The first point to be noticed is that in the very great majority of cases the ulcer is latent. In all the cases mentioned in my clinical lecture the duodenal ulcer had been absolutely latent. During its course it caused neither pain nor hæmorrhage. It revealed itself, suddenly, by perforation in healthy persons, who had never felt any symptom pointing to the presence of such a terrible lesion. It is by perforation, then, that duodenal ulcer generally announces itself. The perforation causes the sudden agonizing pain which I have named the **peritoneal dagger-thrust**. All the cases in my clinical lectures came on in this way.

My patient, when he was about to mount guard at the police-station, was suddenly seized with agonizing pain in the subhepatic region. Nothing could have been more sudden. Dutil's patient was taken unawares at seven o'clock in the evening with such intense abdominal pains that he fell groaning to the ground. He succumbed in eighteen hours, and at the post-mortem examination a perforating duodenal ulcer was found. Vermorel and Marie's patient was also in a state of perfect health when he experienced, without the slightest warning, terrible pain due to perforation. He died in twenty-seven hours, and at the post-mortem examination a perforating ulcer of the duodenum was found. Brouardel's patient had gone to bed the evening before in excellent health, when he was awakened suddenly at three o'clock in the morning with most acute pain between the liver and the stomach. He died in twenty-eight hours, and at the autopsy a perforating ulcer was found.

Another important point is the state of the abdomen after the perforation during the first stage of the peritonitis. We are generally too much imbued with the idea that peritonitis due to perforation is followed by meteorism. This is not always the case, and it is wrong to exclude peritonitis because the abdomen is hard, flat, and retracted. We may see patients suffering from duodenal perforation in whom the peritonitis at first causes such contraction of the abdominal muscles from reflex action that the stomach is flat, hard, and retracted. The stomach was hard and flat in the cases of Letulle, Banzet, and Lardennois. The stomach was likewise



retracted and as hard as a board when Beausse was called in to the patient whose history I have just given. This point is important. Nevertheless, the two conditions may follow one another in the same case. A patient whose stomach is hard and flat in the early stage of peritonitis may present a tympanitic and distended abdomen some hours later.

The discussion which I entered into with regard to the diagnosis of perforation of a gastric ulcer is absolutely applicable to the diagnosis of duodenal ulcer. The suddenness, the intensity of the pain, and the **peritoneal dagger-thrust** are as much the appanage of perforation of a duodenal ulcer as of an ulcer of the stomach. This symptom, when well established, is sufficient to eliminate hepatic colic, intestinal occlusion, appendicitis, and other morbid states with peritoneal symptoms, which never begin with such violent and sudden pains.

The pain of hepatic colic and of duodenal perforation commences in the same neighbourhood, but still the pain of hepatic colic (even when it is severe) is not so sudden and severe as the pain from perforation of the duodenum. Furthermore, a man suffering from hepatic colic, in spite of the pain and vomiting, has never at the end of an hour or two the peritoneal look of one who has a duodenal perforation with early superacute peritonitis. The features are drawn, the complexion is of an earthy pallor, and the abdomen is hard and retracted from the defensive contraction of the abdominal muscles, or tympanitic from the distension of the paralyzed coils of the bowel and the passage of gas into the peritoneal cavity. The least touch causes the most acute pains. The pulse is small and rapid, and perhaps the patient already has hiccough. Nothing of this kind is seen in hepatic colic. I am not speaking of the icteric tinge and of the presence of bile in the urine, for these signs, common in hepatic colic, do not occur early; they supervene when the diagnosis should have already been made.

The knowledge furnished by the temperature is not of much diagnostic value. We are too liable to think that hepatic colic is apyretic, and that peritonitis due to perforation is febrile. This is quite true in some cases, but the converse is also true. Peritonitis may be seen without a rise in temperature, and, on the other hand, hepatic colic may be accompanied by violent rigors and a temperature of 102° or 103° F. I shall describe later this hepatalgic fever, to make use of Charcot's expression, which is a satellite fever in the migration of biliary calculi, and must not be confounded with bilio-septic fever. I refer the reader to the section on Appendicitis for the diagnosis between appendicitis and perforating ulcer of the stomach.

In a patient suffering from tympanites, with pain, vomiting, and stoppage of fæcal matter or gas, peritonitis from perforation has sometimes been taken for intestinal occlusion. This error, I think, should be avoided, because occlusion never commences with the **peritoneal dagger-thrust**. The

symptoms are gradual and progressive, and a period of constipation has preceded the symptoms of occlusion by several days. Nothing of the kind occurs in perforation of the duodenum.

It is almost impossible to say whether the ulcer is seated in the duodenum or the stomach; it can only be localized in the stomach if the perforation has been preceded by the classical signs of ulcer, and even then there may have been two ulcers—one in the stomach, which has cicatrized, and the other in the duodenum, which has perforated. This topographical diagnosis is only of minor importance, since the same operative indications are applicable in both cases.

The suddenness and the violence of the pain, the intensity and generalization of the peritonitis in perforation of a duodenal or gastric ulcer, depend on the dimensions of the perforation and on the rigidity of the tissues which form the wall. If the hole is large and gaping, the indurated edges have no tendency to close the void, as flabby tissues might do. Chyme, microbes, and toxines are rapidly discharged into the peritoneum in considerable quantity. In this respect the duodenal perforation seems to me more to be feared than that of the stomach, for the toxi-infectious matter of digestion may pass into the peritoneum in the form of chyme from the stomach and in the form of fæcal matter from the intestine by antiperistaltic movements. The peritoneum suffers such an attack that the peritonitis becomes general in less than twenty hours, and death may be very rapid.

What a difference from the peritonitis following the small perforations in typhoid fever, which may be so small that at the post-mortem examination it is often necessary to distend the intestine with water in order to discover the breach of surface!

**Treatment.** — Surgical intervention is the only treatment in super-acute peritonitis consequent on perforation of a duodenal ulcer. In nine cases published in the *Medical News*\* the operation was followed each time by death; but it is only fair to say that all these patients were operated on too late. The case of cure published by Landerer and Glückmann refers to a man who was operated on twelve hours after perforation. The conclusion is that in duodenal perforation, as in gastric perforation, it is necessary to make a correct diagnosis, and operate at once.

I have just spoken of cases in which the duodenal ulcer perforates; in some cases it cicatrizes, like ulcer of the stomach. The cicatrix may, however, cause constriction of the pylorus, followed by dilatation of the stomach, and occlusion of the bile-duct, with chronic icterus or thrombosis of the portal vein.

\* The *Medical News*, 1895, p. 460.



## X. CANCER OF THE INTESTINE.

Cancer of the intestine ranks next to cancer of the stomach, liver, and breast. It has this special feature—that it is fairly common in adults.

**Pathological Anatomy.**—The various parts of the intestine invaded by cancer are, in order of frequency, the rectum, sigmoid flexure, colon, cæcum, and small intestine, or, in other words, cancer becomes less common as we approach the upper portion of the intestine. Cancer of the bowel may be primary or secondary to cancer of the stomach or uterus. The following varieties are found: cylindrical epithelioma, encephaloid and scirrhus. It may arise in the glandular epithelium of the mucous membrane or in the submucous layer; it shows itself in the shape of a patch, nodule, or ring. The patches sometimes convert a segment of the intestine into a rigid tube, and the rings constrict the lumen, so that a goose-quill can scarcely be passed through them. The internal surface of the cancer is either fungous, ulcerated and bleeding, or hard and scirrhus. The intestine above the constricted point generally shows a dilatation which may be large; the muscular layers are much thickened in some cases. Extension to the peritoneum and mesenteric glands is a frequent complication, and is generally associated with serous or hæmorrhagic effusion into the peritoneum. Perforations of the intestine and communications with the neighbouring organs are rarer complications.

**Symptoms.**—Intestinal cancer generally runs the following course: The onset is insidious, and for several weeks or months the patient experiences only more or less sharp pains, which may be fixed or movable. The digestive functions are interfered with. Constipation is the rule, but the evacuation of a large quantity of more or less liquid matter often alternates with constipation. If the growth occupies the lower portion of the large intestine, and causes appreciable constriction, the motions are ribbon-like. **Melæna** is a frequent symptom, and the blood voided in the stools keeps its red colour if the lesion occupies the lower part of the intestine, or if the hæmorrhage is abundant and rapidly rejected. The blood is tarry if it has remained in the intestine in contact with the excreta. The abdomen is often ballooned, and this condition is unfavourable to the search for a cancerous tumour. In malignant stricture we may see attacks of pain in which the intestinal coils stand out in relief and are visible beneath the abdominal walls.

The growth is not always accessible to abdominal palpation. If the patient has grown thin, or if the abdomen is retracted, the tumour can be made out, and is generally found to be mobile; if the stomach is distended, or if peritoneal effusion is present, the tumour may be difficult to find.

The general symptoms, as in every case of cancer, are progressive anæmia and gradual loss of flesh. The pale colour of the skin is followed by the yellow tint of cancer, the distaste for food increases daily, emaciation becomes extreme, and cachexia, with œdema and dropsy, ushers in the end. Death supervenes from cachexia (dropsy, thrush, or diarrhœa), or in consequence of some complication (peritonitis, intestinal obstruction, perforation, or hæmorrhage).

Such is the general course of intestinal cancer, but the exceptions are numerous. The development of cancer is latent in some cases, and symptoms of obstruction suddenly appear during apparent good health. The sudden onset of the complications leads us to think of some other lesion. Laparotomy reveals a cancerous stricture of the intestine. In other cases before the cancer of the intestine has caused general symptoms, chronic peritonitis with effusion shows itself, and we naturally think of ascites due to disease of the peritoneum or liver. Abdominal puncture, however, yields blood-stained fluid. Palpation, made easier by the withdrawal of the liquid, shows the condition of the peritoneum and of the intestine (cancerous peritonitis), although the diagnosis was impossible prior to the evacuation of the liquid.

**Cancer of the rectum** belongs rather to surgery than to medicine, and if some mistakes are made, it is because digital examination of the rectum is too often neglected. It is only by such an examination that we can ascertain the existence and the site of rectal cancer.

**Diagnosis.**—The diagnosis of intestinal cancer is easy when its course is classical. The abdominal pains, the alternate constipation and diarrhœa, the presence of a tumour, and the invading cachexia, leave no room for error. The diagnosis is more difficult when the lesion is masked by complications (cancerous peritonitis, ascites) which accompany or hide the primary mischief. This diagnosis will be made with reference to **chronic tubercular peritonitis**. The diagnosis may be difficult with stenosis of the jejunum. Rénon reports a curious case of double stenosis of the jejunum with Koenig's syndrome, simple ulcer above each stenosis and perforation at the level of one of the ulcers.

The **treatment** of intestinal cancer is surgical.

## XI. INTESTINAL OCCLUSION.

**Definition.**—The names *ileus*, *volvulus*, *iliac passion*, or *miserere colic*, were employed to describe a morbid condition characterized by absolute constipation, ballooning of the stomach, uncontrollable vomiting, and severe pains. This disease was for a long time considered to result from spasm of the intestine. Later it received the name of **intestinal strangulation**. This name is wrong, since it only states a part of the question, and therefore the name **intestinal occlusion** (O. Massan) is to be preferred, as it has the advantage of being applicable to all cases of obliteration of the intestine.



**Ætiology.**—The various causes of intestinal occlusion are as follows (Jaccoud):

1. **Occlusion by Constriction.**—The constriction of the intestine may be due to compression by a neighbouring tumour, such as a cystic or cancerous tumour of the peritoneum, ovaries, or mesenteric glands, uterine fibroids, and aneurism of the abdominal aorta. The constriction may be due to changes in the intestinal walls (cancer or polypus), or to cicatrices caused by the ulcerations of dysentery and tuberculosis. Syphilis usually produces stricture of the rectum. Cancerous strictures of the small intestine are very rare. In 72 out of 108 cases the trouble was seated in the sigmoid flexure and rectum (Bulteau). Occlusion consequent on spasmodic stricture of the intestine has become rare since the other causes of occlusion have been better known. It does, however, exist, and I have seen two cases.

2. **Occlusion by Strangulation.**—This form is frequent. It is analogous to the strangulation of hernia. The intestine becomes engaged in an abnormal orifice of the mesentery or omentum, or in the foramen of Winslow. At other times the strangulation of the intestine is brought about by a band stretching from one intestinal coil to another; from the bowel to the abdominal wall, the mesentery, or the uterus; from the omentum to the peritoneum; from the appendix to an intestinal coil; or from an intestinal diverticulum to the wall of the abdomen. Meckel's diverticulum, which is adjacent to the termination of the small intestine, and is considered to be the remnant of the omphalo-mesenteric duct, may attain such a length that it forms knots around the intestine. In these different varieties the strangulation almost always occurs at the end of the ileum. In 151 cases collected by Bulteau the strangulation was seated 133 times in the small intestine, and only 18 times in the large intestine.

3. **Occlusion by Volvulus.**—Volvulus results from twisting of the intestine. The laxity of the mesentery favours twisting of the cæcum and of the sigmoid flexure. The small intestine is strangulated round the mesentery, which serves as an axis. Sometimes there is simply bending, and the intestinal coil forms a kink.

4. **Occlusion by Invagination.**—By invagination is understood the entrance of one portion of the intestine into another portion. In most cases the upper portion enters the lower portion. The canal of the invaginated segment is constricted, and the new wall is formed of the three layers of intestine. The mesentery, which has followed the invaginated portion, is found between the middle and the internal layer. If the invagination is not followed by strangulation, it may persist in the chronic state, and the intestinal occlusion is incomplete. The complications of strangulation which accompany invagination are due to the action of the invaginating segment,

which acts like a ring as regards the invaginated segment, and it is probable that the mesentery which pulls on the invaginated cylinder has some share in the phenomenon of occlusion.

The invaginated portion is compressed by the ensheathing portion, and becomes congested, swollen, and inflamed. The serous layers become adherent, and the inflammatory process causes one of the following terminations: (1) The inflammation and the acute symptoms may disappear, and give place to a chronic invagination, which narrows the lumen of the intestine, and gives rise to further troubles. (2) The invaginated cylinder becomes gangrenous, and is passed per rectum, whilst the adhesions of the walls prevent perforation, and render recovery possible. (3) Partial sub-acute peritonitis may occur around the intestinal segment which is the seat of the invagination; perforation may cause fatal peritonitis.

With regard to the seat of the invagination, out of a total of 763 cases 392 have been found to be ileo-cæcal; 220 were invaginations of the small intestine, and 151 of the large intestine. In **children** invagination is practically the sole cause of intestinal occlusion. The length of the invaginated cylinder is very variable. It attains large dimensions in the large intestine. A second intestinal segment often enters the invaginated cylinder, and a third segment has even been known to enter the other two.

**5. Occlusion by Obstruction.**—The accumulation of fæcal matter in the cæcum or large intestine, intestinal concretions (enteroliths), gall-stones, round worms, and foreign bodies introduced into the rectum (Verneuil), may cause intestinal occlusion.

**6. Occlusion by Pseudo-Strangulation.**—The stoppage of fæcal matter from intestinal paralysis occurs in peritonitis and appendicitis. In some cases the patient has all the symptoms of intestinal occlusion. An operation fails to reveal the obstruction. Has the occlusion its origin in a paralysis of the intestine? Is this paralytic condition accompanied by kinking of the intestine? These facts are not completely elucidated.

**Symptoms.**—The onset shows many variations. In some cases the occlusion comes on suddenly, in the absence of any apparent cause, with sharp pain, which spreads over the abdomen. Nausea, hiccough, and vomiting of food and bile then appear. In the next few days the vomit becomes **fæcal**. The abdomen is at first retracted, but soon becomes distended, and the intestinal coils stand out in more or less relief. The patient passes neither fæces nor gas. The loss of strength is rapid, the temperature falls, the general symptoms grow worse, the pulse becomes small and irregular, and death may supervene in three or four days. This variety, which somewhat resembles strangulated hernia or acute peritonitis, is not the most frequent one. We shall study it later under Tubercular Peri-



tonitis. It is, indeed, often the first index of a tubercular peritonitis which has previously been latent.

In the great majority of cases the symptoms of intestinal occlusion come on **more slowly**. The pain is neither so sudden nor so intense as in the preceding case. It is sometimes limited to the diseased region ; at other times it radiates in various directions. Constipation is not absolute till the second or third day, when the patient has emptied the lower end of the intestine. From this moment, if the occlusion is complete, the patient passes neither fæces nor flatus, though he may void a little mucus. The passage of blood or shreds (detritus of the mucous membrane) indicates sloughing in the case of invagination.

The **vomit**, which at first consists of food, mucus, and bile, becomes fæcal about the third or fourth day, or later. It is quite liquid, because it is mixed with the fluid swallowed by the patient, and is composed of the matter which is contained in the upper part of the intestine, and has regurgitated into the stomach by the antiperistalsis. After the onset of occlusion the stomach becomes distended, the meteorism grows excessive, and in some cases, especially during the paroxysms of pain, the intestinal coils stand out in relief around the umbilicus or in the flanks. The urine is scanty, especially if the obstacle is seated near the stomach (Barlow), while the thirst is great, and the ingestion of the smallest quantity of liquid provokes vomiting. Prostration and loss of strength are early symptoms. The pulse is small and rapid ; the face is pale, thin, and drawn ; the skin is cold and wrinkled ; the voice is reduced to a whisper.

Several terminations are possible. In the happiest, but the rarest, cases the fæcal vomiting ceases, and the patient passes flatus and fæces *per rectum*. The motions are passed freely. In other cases the acute symptoms of occlusion disappear, but are then followed by symptoms of chronic invagination, which constitute a permanent danger. Perforation and acute peritonitis in the course of intestinal invagination are rapidly fatal ; but this is not the manner in which death usually supervenes. The acute symptoms of the onset improve, the pain in the abdomen is not so severe, and we are almost tempted to speak of improvement ; but the patient grows cold, the voice sinks to a whisper, the pulse becomes irregular and imperceptible, dyspnœa, hiccough, and muscular spasms appear, the extremities become livid, and death occurs on the tenth or twelfth day—not from peritonitis, but from the **auto-intoxication** which Gubler has called **peritonism**. Its origin lies in the abdominal sympathetic nerves.

**Diagnosis.**—In order not to confound intestinal occlusion with **strangulated hernia**, the usual sites of hernia must be examined. Intestinal occlusion must not be mistaken for peritonitis from perforation of

simple ulcer of the stomach and duodenum. This question has already been discussed. Appendicitis is sometimes accompanied by marked tympanites and arrest of flatus. The reader will find this point discussed under Appendicitis. Intestinal occlusion must not be confounded with lead colic or with acute intoxication. Obstruction in the **rectum** will be recognized by digital examination.

The cause of the occlusion is often obscure. The mode of onset is not enough. It has been said that acute symptoms occur, especially in strangulation or volvulus, and that gradually increasing symptoms are due to the compression of the intestine or to the growth of a tumour in the walls, etc. These assertions are not always true, and it is not rare to see symptoms appear with all the suddenness of strangulation when the obstruction is really secondary to cancer of the intestine.

The diagnosis of the cause is, however, possible, in certain cases. If the occlusion occurs in a patient who has been suffering from alternate diarrhoea and constipation, melæna, and loss of flesh, intestinal cancer will be thought of, even though a tumour is not appreciable on digital examination of the rectum or on abdominal palpation. If the occlusion comes on suddenly during good health, with sharp pain, vomiting, which rapidly becomes faecal, and absolute constipation: internal strangulation or volvulus may be inferred. If the onset is less sudden, the pain is less severe, the faecal vomiting occurs later, while the constipation does not set in early, and a doughy mass can be felt in one part of the abdomen: it is probable that the case is one of invagination. If the symptoms of occlusion have been preceded by obstinate constipation, and if palpation reveals a painless, soft, and pasty mass in the region of the cæcum or the sigmoid flexure; a faecal accumulation is present.

Recognition of the site of the lesion is also difficult. Digital examination *per vaginam* and *per rectum* must always be carried out, the finger being pushed up as high as possible into the rectum, for we may thus discover a cicatricial stenosis, cancer, faecal accumulation, or invagination in this region. The initial seat of the pain is a valuable but uncertain sign. The absence of faecal vomiting would prove that the intestine is obliterated high up, probably in the duodenum. Ballooning of the belly, more pronounced at the centre of the abdomen than at the sides, would indicate that the obstacle is in the small intestine. Early meteorism over the whole of the abdomen is in favour of obstruction in the large intestine. An early onset and severity of the general symptoms point to a lesion of the small intestine, which is so rich in nerve plexuses.

The **prognosis** of intestinal occlusion is always formidable, even when the symptoms are slight and the obstruction is incomplete. In faecal accumulation the prognosis is good, because it generally yields to treatment.



The other varieties—invagination, volvulus, and especially strangulation—are very grave.

**Treatment.**—The means employed in intestinal occlusion are medical and surgical. Purgatives give good results when the occlusion is due to a mass of fæces or intestinal paresis; in other circumstances they are more harmful than useful. They must, therefore, be used with great care in occlusion, and their use must not be unduly prolonged.

Purgative enemata of sulphate of soda, syrup of buckthorn, infusion of senna, and enemata containing 60 grains of bicarbonate of soda and 30 grains of tartaric acid in each enema, and repeated three or four times during the day, have given good results. Large injections (Cantani), given with a douche-can and high rectal-tube, are of service.

**Electricity** (continuous currents are employed to-day) has several times been successful (Lacaille), when employed prior to any inflammatory complications.

Valuable time, however, is often lost by medical treatment, and **surgical intervention** is needed in the majority of cases.

## XII. CANCER OF VATER'S AMPULLA.

**Discussion.**—On account of its topography and symptoms, cancer of Vater's ampulla deserves special mention. In 1890 Busson had collected a dozen cases. New cases have since been published.

As the name implies, the growth affects the ampulla of Vater, where the ducts of the liver and pancreas open into the gut. Does the cancer arise in the intestine, the common bile-duct, or the pancreatic duct? In other words, is it an intestinal, biliary, or pancreatic cancer? According to Rendu, it is intestinal; according to Hanot, pancreatico-biliary; according to Bard, pancreatic; and according to Durand-Fardel, biliary.

In a recent case published by Dominici a primary epithelioma developed at the expense of the biliary and pancreatic ducts. It must be distinguished from cancer of the head of the pancreas (alveolar cancer) commencing in the pancreatic acini.

**Pathological Anatomy.**—Primary cancer of Vater's ampulla may form a plaque, or, more frequently, a mushroom-like growth of the size of a hazel-nut or a walnut. The tumour projects into the cavity of the duodenum. It is whitish, fairly soft, and rarely ulcerates. The orifices of the common bile and pancreatic ducts terminate in the tumour, which obliterates them more or less completely. In some cases the orifice of the pancreatic or of the common bile-duct may be found in the cancerous vegetation. The mass may absorb the orifice of the bile-duct and grow into the opening of this canal, as if the cancer had commenced in the orifice of the duct and had then invaded the ampulla (Durand-Fardel's case). When the opening of the common duct is obliterated, this duct, as well as the cystic and hepatic ducts and the gall-bladder, may be much dilated.

Cancer of Vater's ampulla is almost always a cylindrical epithelioma. It is limited to the ampulla, and has no tendency to become general. It does not invade the pancreas or the liver, though it may cause adenitis in the pancreatic and mesenteric glands.

The pancreas shows no cancerous lesion. It is sometimes normal, sometimes enlarged.

The liver is not invaded by the growth. It is enlarged, infiltrated with biliary pigment, and often the seat of incipient fibrosis of the portal system (Hanot). The gall-bladder is often distended and full of bile, but the remarkable fact is that it very rarely contains gall-stones (once in fifteen cases). This finding may well be compared with their extreme frequency in primary cancer of the biliary passages. The spleen is often enlarged.

**Summary.**—Whether the growth begins in the pancreatico-biliary orifice (orificial cancer) or in the intestinal mucous membrane of the ampulla, it is none the less true that it behaves “like the patches of intestinal epithelioma met with at the ileo-cæcal valve or in the sigmoid flexure. It has the same superficial disposition and the same slow course, with but little tendency to ulcerate, to become general, or to spread to the adjacent glands” (Rendu).

**Symptoms.**—The small cancer of Vater's ampulla betrays its presence by jaundice. It may, indeed, be said that jaundice is the first apparent symptom. The jaundice is due to the obstruction of the common duct by the tumour, and has therefore the characteristics of jaundice from obstruction—colour changing from yellow to olive-green, bile-pigment in the urine, and colourless fæces. As the jaundice is not always continuous, the skin resumes its natural colour, and the fæces become coloured. The intermittent character of the jaundice proves that the orifice of the common duct regains its permeability—for a time, at least. Itching, which troubles the patient day and night, is sometimes present. Pigmented spots and xanthelasma have been noticed.

Pain, either spontaneous or provoked, is a rare symptom, but it has been noticed in some cases. Rendu's patient had sharp pain in the right hypochondrium and epigastrium. Palpation was very painful in the pit of the stomach over the left lobe of the liver, so that it was a question whether the patient was not suffering from a stone in the common duct.

Vomiting of food is a fairly frequent symptom. Diarrhœa usually appears late. It may alternate with constipation.

On examining the patient, the liver is found to be enlarged. The gall-bladder is often much distended, and can be felt through the abdominal wall.

Some months after the onset of the disease general symptoms appear. The appetite disappears, and the patient becomes cachectic. In one case



fever supervenes; in another intestinal hæmorrhage appears. Œdema sets in, and is sometimes confined to the right side. Finally, after a period which varies from six to eighteen months, the patient succumbs from cachexia, or is carried off by some complication.

**Diagnosis.**—The diagnosis of cancer of Vater's ampulla sometimes presents insurmountable difficulties. On what signs are we to rely? Jaundice, which is the first and principal symptom, is also present in a number of conditions. It matters little whether the common bile-duct is compressed or obliterated by a cancer of the head of the pancreas, of the biliary passages, or of Vater's ampulla, by one or several calculi, or by the swelling of the walls which occurs in catarrhal icterus. In all these cases jaundice and its symptom-complex (bile in the urine, colourless stools, enlarged liver, etc.) may be the only symptom for weeks or months. It will be objected, no doubt, that in a case of cancer of Vater's ampulla the jaundice and the decoloration of the stools are at times intermittent; but intermission is likewise seen in calculus of the common duct and in prolonged catarrhal icterus. Cases have been seen, and I myself have published some, in which this same phenomenon—viz., reappearance of bile in the stools and momentary suspension of jaundice—was present in cancer of the head of the pancreas. This feature, therefore, is not special to cancer of Vater's ampulla.

On the other hand, it will be said that spontaneous or provoked pain is particularly the result of stones in the common duct; but I would point out that primary cancer of the biliary passages and cancer of Vater's ampulla are also accompanied by similar pain, as in Rendu's case. Diarrhœa is said to be an important symptom, but in reality it has no more value than the preceding ones in the diagnosis of cancer of Vater's ampulla, and most frequently, it must be admitted, the diagnosis is founded on conjectures. I have nothing to say on the subject of **treatment**, unless it be to suggest surgical intervention.

### XIII. DYSENTERY.

**Ætiology.**—Dysentery has every appearance of being a microbic disease. Two forms are seen—amoebic dysentery, due to a protozoon, and bacillary dysentery, due to a bacillus.

The latter occurs in temperate zones, and is common in France, but it is also found in tropical countries in conjunction with the amoebic form. The amoebic form is scarcely ever seen in temperate climates, but is met with in tropical lands. It may or may not be associated with the bacillary form.

Although the organisms are different in the two forms, the symptoms are alike in both cases.

Dysentery is sporadic, endemic or epidemic, and acute or chronic, and its character differs according to the variety.

Sporadic dysentery breaks out during the great heat of summer, and disappears towards the end of the autumn. It has been asserted that bad water, cold drinks, and green fruit favour its development.

Endemic dysentery occurs in Senegal, Cochin-China, Mexico, the West Indies, India, Algeria, etc. Are the climatic conditions and the excessive heat of these countries ætiological factors? They are, doubtless, important, but they are not the sole factor. Are the conditions of the soil and malaria causative? And are we to say, with Cambay and Dutrouleau, that dysentery, like hepatitis and swamp fever, is a manifestation of paludal intoxication? The answer is that in certain countries where malaria is endemic, dysentery is unknown or exceptional. Thus, Guadeloupe and Pointe-à-Pitre are decimated with marsh fever, while dysentery is fairly rare; and the same may be said of the Départements of Aunis and of Saintonge. The final argument is that each of these diseases possesses its specific microbe.

Epidemic dysentery is favoured by the ætiological conditions of the two preceding varieties, but as special causes of the epidemic, writers have cited overcrowding, famine, bad water, and defective hygienic conditions, such as are observed with armies in the field or in besieged towns, etc. "But," says Trousseau, "these are only occasional causes. Something else is required independent of them, and this something, which we know only through its effects, we call the epidemic constitution." The discovery of the micro-organisms of dysentery has confirmed the far-seeing observations of Trousseau.

Dysentery is contagious. This fact is well proved, and the contagion is probably due to the dejecta. Water seems to play a considerable part in the dissemination of dysentery. This fact has been clearly proved by the medical officers with our troops in Cochin-China.

**Pathological Anatomy.**—The chief lesion is an ulcerative inflammation of the large intestine. The ulcers are found in the rectum and sigmoid flexure. They present different degrees, according as the dysentery is slight or severe, but in the main they are quite analogous. We must first consider briefly the structure of the large intestine.

The large intestine is composed of several coats, which are, from within outwards, serous coat, formed of flat cells and connective tissue; next two layers of smooth muscles, the one longitudinal and the other circular; and, lastly, an internal or mucous coat. The two last-named coats present the following peculiarities: The superficial portion of the mucous membrane is composed of tubular glands, analogous to Lieberkühn's glands in the small intestine, but somewhat longer. These glands are united by a very fine fibro-vascular framework, and their cylindrical epithelium is continuous with that of the mucosa. Next to this glandular layer we find smooth muscular fibres and connective tissue, which is more like ordinary connective tissue than the reticu-



lated tissue of the small intestine. The closed follicles in this cellular coat are less numerous but larger than those of the small intestine. The cellular coat is traversed by large vascular branches, which form a compact plexus (Doellinger's vascular membrane) at the inner limit of the cellular coat, whence the perpendicular branches arise, and ramify between the tubular glands. The lymphatics are much less abundant than in the small intestine.

**1. Benign Dysentery.**—The lesions attack the rectum and the sigmoid flexure. At the onset the mucous membrane is red, thickened, ecchymosed, and granulating. It is covered with a yellowish, puriform mucous exudation, streaked with blood, and analogous to the stools characteristic of this period. The hypertrophied lymphatic follicles stand out in relief. A few days later punched-out **ulcers**, as well as larger and more irregular ones, are found on the mucous membrane. On microscopic examination, the vessels of the glandular layer appear swollen and surrounded with connective tissue that is infiltrated with embryonic cells. The tubular glands are compressed and elongated. The subjacent vascular network is likewise surrounded with fibrinous exudate and connective tissue, infiltrated with embryonic cells. The swollen follicles, which are also infiltrated with lymphatic cells, stand out on the surface of the mucous membrane. The rounded follicular ulcers are due to softening and suppuration in the centre of the follicles. The more extensive ulcers arise from gangrene of the mucous membrane, which lacks proper blood-supply, because it is infiltrated and compressed by new elements. The ulcers extend and suppurate. In slight cases, however, the process rapidly ends in recovery.

**2. Grave Dysentery.**—The lesions are similar to those just described, though they are much more severe. They are spread over the large intestine, and may be found in the small bowel. The **ulcers** are extensive, deep, and distributed over the whole of the large intestine. In places the mucous membrane resembles a piece of wood eaten by worms (Kelsch). In other cases the mucous membrane is removed in patches over a large extent, and we find on the cellular coat only a few tubular glands, which are the remnants of the exfoliated mucous membrane. The ulcerated surfaces are reddish, granular, and covered with purulent débris; the edges of the ulcers are clean-cut. The vascular network subjacent to the glandular layer is infiltrated with embryonic cells and coagulated fibrin. The vessels are dilated and congested with blood, and their walls have returned to the embryonic condition, while the submucous cellular tissue resembles a phlegmon spread like a sheet over the glandular layer. These vascular changes sum up the chief lesion in dysentery. The morbid process is centred in the vascular zone of the cellular coat and in its peripheral expansion in the mucous membrane (Kelsch). As the mucous membrane is deprived of its blood-supply, it necroses *en masse*, and is cast off in the form of sloughs.

The parts underlying the glandular layer (cellular coat) are twice or thrice as thick as in the normal state. This swelling is due to the fullness of the bloodvessels, the congestion of the lymphatics, and the infiltration of the connective tissue. Perforation of the intestine is very rare. The ulceration may lead to scar tissue, which causes stenosis of the gut. The ileum often shows catarrhal changes in the grave forms of dysentery. Lieberkühn's glands are in part replaced by embryonic tissue, and the vascular walls are likewise converted into new tissue; but the changes do not attack the cellular layer, the follicles, or Peyer's glands.

In acute dysentery we find suppurative adenitis of the **mesenteric glands** and softening and hypertrophy of the spleen. Hepatitis and abscesses of the liver will be described later (see Liver).

**3. Chronic Dysentery.**—Chronic dysentery and the chronic diarrhœas of hot countries present analogous changes. The divergence between these two morbid states is chiefly in the symptoms. In chronic dysentery the mucous membrane of the large intestine is congested in places, and at certain spots there are small rounded ulcerations, which are not very deep, and have a brown or slaty surface. The ulcers arise in the closed follicles, and are the end of the orifices which lead into small cavities. These cavities or follicular depressions are elliptical or rounded. They are formed of one or three pockets, and contain a gelatiniform mucus, which may be extracted by pressure. It is probable that the follicular diverticulum occupies the place of the destroyed follicle.

The submucous connective tissue which separates the muscular layers of the intestine and the subserous tissue are thickened and fibrous, so that the intestine tends to become converted into a canal with rigid walls. In the chronic diarrhœa of hot countries this change also affects the small intestine.

**Bacteriology.**—The bacillus of dysentery, discovered by Chantemesse and Widal, is a short, slightly mobile rod, which neither stains with Gram nor liquefies gelatine. It does not coagulate milk. It does not cause sugar to ferment, and it never produces indol, so that it somewhat resembles the typhoid bacillus. Shiga has proved that it is a specific bacillus by obtaining the agglutination of cultures only with the blood-serum of patients suffering from dysentery. Krase in Germany, Flexner in the Philippines, Strong and Murgrave in Manilla, Rosenthal in Moscow, and Vaillard and Doepter, have all found the same bacillus, with some slight modification. It is exceedingly abundant in the stools, but not in a pure state.

Vaillard and Doepter, by the subcutaneous inoculation of the bacillus or its toxine, have produced in certain animals the symptoms and the lesions characteristic of epidemic dysentery, and have thus furnished a fresh proof of its specific nature. Amœbic dysentery is only seen in hot



countries, and is noteworthy because it gives rise to abscess of the liver. The blood of patients suffering from amoebic dysentery does not agglutinate the bacillus of Chantemesse and Widal.

**Symptoms.**—The description of dysentery varies in benign or grave cases as well as in different epidemics, and I shall select, therefore, a case of average severity as my type, indicating subsequently the different forms of the disease.

At the onset the patient is taken ill with **diarrhoea** and abdominal pains. At the end of twenty-four or forty-eight hours the stools become dysenteric, and contain whitish mucus analogous to partially coagulated albumin. The mucus is sometimes transparent and mixed with shreds of blood; at other times it has the rusty appearance of pneumonic sputum. Intestinal ulcers have not yet formed. The evacuations are preceded by a painful feeling of tension and constriction at the anus (spasms and tenesmus), and by an incessant desire to stool, but the patient is unable to pass more than a teaspoonful of matter. The tenesmus sometimes spreads from the rectum to the bladder (dysuria). The tenesmus, which is so painful, is not due, as was believed, to the convulsive contraction of the sphincter, because the anus is gaping, but to the acute inflammation of the passage.

In addition, colic is felt around the umbilicus and along the course of the large intestine. The pain is increased by pressure, especially in the left iliac fossa.

When ulcers are present, the stools change, and contain, in addition to mucus, pure blood and shreds of ulcerated mucous membrane. It is not uncommon to find moulded faecal matter in the stools. At this period the effort of defæcation is repeated several times an hour, and in very grave epidemic cases the patients have as many as 100 to 200 stools in the twenty-four hours, and the total quantity of matter voided may amount to 7 or 8 pints.

During the second week the stools, which are horribly foetid, contain practically no mucus, because the mucous glands are partially destroyed, but consist of a serous and reddish liquid in which shreds of membrane float. They may also contain pus in considerable quantity at this time.

I shall next describe the general symptoms, which vary in different epidemics and in different countries.

1. In the **benign** form the temperature does not rise above 100° F., and the stools do not exceed twelve to fifteen a day. The patient, however, wastes rapidly, and his appearance changes quickly. These cases recover in about a week.

In the **grave** forms the general symptoms rapidly become alarming. There is great thirst, the skin is dry, and the pulse variable. The evacua-

tions are incessant, and the intolerable pain wears out the patient. His strength declines suddenly, and the loss of flesh is considerable. Prostration, somnolence, and chilling often complete this picture, and death may supervene from the fourth to the twentieth day of the disease.

2. The **inflammatory** form is characterized by marked febrile reaction, with quickening and hardness of the pulse. The tongue, instead of being coated, is red and dry. The stools are scanty, and when they become more frequent, the fever speedily falls.

3. In the **bilious** form, contrary to what is observed in dysentery, there is conjointly diarrhoea, and the dysenteric stools are mixed with yellow and greenish bilious matter. The patients complain of nausea and vomiting, and the tongue is covered with a thick fur, but the fever is not high.

4. The **rheumatic** form, says Stoll, is remarkable for metastases in the joints. In the medical parlance of to-day we say: dysenteric infection produces an arthropathy which belongs to the class of pseudo-rheumatism. This variety of arthropathy is observed in the slight forms of dysentery more often than in the graver cases. It appears chiefly about the period of convalescence. It commences generally in the knee, and then affects several joints, such as the ankle, the shoulder, and the small joints of the fingers. Dysenteric arthropathy supervenes suddenly, causing neither fever nor acute pain. In some cases its appearance coincides with the arrest of the dysenteric flux. Alternation of the intestinal and articular localizations have been recorded twice or thrice in the same individual. These cases of arthropathy are obstinate, and may last for weeks or months, but they are not accompanied by any of the visceral complications of true rheumatism. During an epidemic observed at Montargis (Huet) the rheumatic complications took the form of arthritis analogous to blennorrhagic arthritis.

5. The **intermittent** form is characterized by remissions, followed by exacerbations, and must not be confounded with dysenteric **pernicious fever**.

6. The **adynamic** and **ataxic** forms are characterized, on the one hand, by prostration and depression, leading rapidly to coma, and, on the other, by delirium, restlessness, carphology, and subsultus tendinum. These terrible forms occur in association or in succession, and constitute **malignant dysentery**.

7. **Chronic dysentery** is frequently seen in hot countries, and generally follows after several acute attacks. It is characterized by the symptoms of acute dysentery, though greatly modified. Thus the evacuations are serous or puriform, and rarely bloody. The anus is gaping, and the tenesmus is replaced by a painful feeling of weight. The abdomen is retracted and



painful on pressure. Apyrexia is complete, and the appetite is normal or increased. In spite of this, the loss of flesh proceeds, and the patient generally becomes cachectic. The disease lasts for months and years, and its progress is sometimes interrupted by acute attacks. Cure is possible, but death is the most frequent termination.

**Complications.**—The complications of dysentery are as follows: (1) **Perforation** of the intestines, which may cause local or general peritonitis, phlegmon localized to the rectum or to the cæcum, and intestinal hæmorrhage, which may be copious enough to cause death. (2) **Parotitis**, characterized by inflammation and suppuration in the parotid glands and the surrounding cellular tissue. The pus may invade the cellular tissue of the neck and dissect out the muscles. (3) Partial and temporary **paralyses**, such as follow acute diseases. (4) **Cicatrices** of the intestine, a late complication, which may cause stenosis and symptoms of intestinal occlusion. (5) **Abscess of the liver**, which is the most typical complication of dysentery.

**Diagnosis.**—The diagnosis of acute dysentery presents no difficulties. The finding of the microbe in the stools and especially the sero-diagnosis are specific. In difficult cases they clear up the diagnosis. The **chronic diarrhoea of Cochin-China** must not be confounded with chronic dysentery. In chronic diarrhoea bloody stools and tenesmus do not occur. The dejections are mucous or bilious, and the course of the disease is not interrupted by acute phases, as in dysentery.

The **prognosis** is generally benign in sporadic dysentery, but it is much graver in the endemic form, and becomes alarming in certain epidemics.

**Treatment.**—Trousseau advocates the following measures: At the onset ipecacuanha in emetic doses should be prescribed—45 grains, divided into four powders to be taken at intervals of ten minutes, until vomiting occurs. On the next day a neutral salt, such as sulphate of soda or Seignette salt, in doses of 3 to 5 drachms, should be given, and purgation continued every day, or twice a day, until the stools become fæcal. At the same time the disease should be attacked by means of enemata, containing 3 to 10 grains of nitrate of silver in 10 ounces of water. The patient must be fed with soups, thick panadas, or eggs beaten up in broth. Rice-water, albumin-water, or the white decoction of Sydenham, may be given to drink. Enemata of 1 quart of water, with a few drops of laudanum added, may help to relieve the spasms and the tenesmus.

The treatment of chronic dysentery is different. Enemata of nitrate of silver and saline purgatives in small doses are recommended, and we may also employ subnitrate of bismuth in large doses. Milk diet and the use of raw meat constitute the most essential part of the regimen.

As precautionary measures we should filter and boil water, avoid sudden changes of temperature, and destroy putrid foci as far as possible. The patient must be isolated. Contagion must be avoided, and soiled linen and fæcal matter must be disinfected.

Vaillard and Doepter have immunized horses against the bacillus, and have obtained a serum that has given good results in bacillary dysentery.

Their conclusions are as follows: In an adult 20 c.c. of serum usually causes rapid relief of the symptoms, and recovery in cases of moderate severity. If the symptoms are not better at the end of twenty-four hours, the injections are repeated, but in diminished doses.

In grave cases 40 to 60 c.c. are at once injected. A similar dose is given next day if need be. The serum is then given in diminishing doses.

In the worst cases it is necessary to give 80, 90, and even 100 c.c. This quantity is given in divided doses twice daily until the intestinal symptoms improve. The serum must then be continued in decreasing doses. In children the amount is reduced to one-half of the adult dose. Very satisfactory results have been obtained by this treatment.

Prophylactic injections of 10 c.c. give immunity which lasts for ten or twelve days.

#### XIV. INTESTINAL WORMS.

Of the **worms** (helminthes) which live in the intestine of man, some are ribbon-like (**Cestodes**, from *κεστός*, festooned), and others are cylindrical (**Nematodes**, from *νήμα*, thread). The former are the *tæniæ* (from *ταινία*, ribbon), and the cylindrical worms are the *Ascaris lumbricoides*, the *Oxyuris vermicularis*, and the *Trichocephalus*.

**Ascaris Lumbricoides.**—This worm, which is white or reddish, cylindrical, and tapered at both ends, attains a length of 6 to 10 inches. The ascarides live in the small intestine, and several hundred may be present. They often pass unnoticed, until their presence is revealed by the escape of a worm from the anus or the mouth. At other times they cause vomiting, ballooning of the abdomen, colic, diarrhœa, and reflex nervous complications, such as convulsions, mental troubles, or paralysis. It is probable that the ascarides enter the digestive passages in the form of eggs contained in non-filtered water (Davaine). The anthelmintics most used against ascarides are semen-contra (10 to 30 grains), santonin (1 to 4 grains), and Corsican moss (30 to 60 grains).

**Oxyuris Vermicularis.**—The oxyuris is a small white worm, which is  $\frac{1}{2}$  inch in length. It is very common in children, and is usually found in the rectum and around the anus, where it causes severe itching. The oxyuris may, however, invade the vulva and the vagina or the prepuce and



the urethra. It may also pass up the digestive tube and be vomited by the mouth. The chief symptom is pruritus, which may become intolerable just after the patient has retired to bed. Saline enemata generally suffice to destroy or drive away the worms. If this treatment is not efficacious, enemata composed of mucilage of gum, holding in suspension 1 to 4 grains of calomel, should be given.

**Trichocephalus.**—This is a little worm 1 to 2 inches in length. Its posterior extremity is swollen. It is generally found in the cæcum, but it does not give rise to special symptoms. Metchnikoff thought it played some part in appendicitis.

**Bothriocephalus—Cachexia.**—The bothriocephalus, so called because it has two dimples (*βόθριον*, a dimple) on the lateral parts of the head, is very common in Switzerland, Finland, Livonia, etc., but is somewhat rare in France. This fact is not sufficiently borne in mind when rapid cachexia occurs, and the signs recall those of cancer of the stomach and of pernicious anæmia. The diagnosis, however, is important, because we can effect a rapid cure in a patient who appears doomed to an early grave. The following case at the Hôtel-Dieu has been published by Nattan-Larrier :

A patient was admitted for acute epigastric pain. He vomited after every meal, had lost 16 pounds in weight, and was extremely weak. His colour was waxy, his eyes were dark, his conjunctivæ and his lips were colourless, and he had slight œdema of the legs. The diagnosis of cancer had been made.

The illness had commenced five months before with epigastric pain, followed by diarrhœa, anorexia, pallor, vertigo, daily vomiting, and wasting. No hæmatemesis, no melæna, and no induration of the stomach, which would have pointed to cancer. The blood-count differed from that of cancerous anæmia, the figures being : red corpuscles, 1,400,000 ; white corpuscles, 2,500 ; percentage of white corpuscles : polynuclears, 48 ; mononuclears, 52 ; no eosinophiles, no myelocytes, no nucleated red corpuscles ; a few deformed red corpuscles ; hæmoglobin coefficient, 1·2. The number of red corpuscles, though small, did not put cancer out of count ; the other features of the count, however, did so. In cancerous anæmia the hæmoglobin coefficient is always below normal ; in this case it was above normal. In one-third of the cases of anæmia due to new growths we find nucleated red corpuscles ; in this case both normoblasts and megaloblasts were absent. In cancerous anæmia the number of leucocytes is increased ; in this case it was diminished.

Was the blood-count compatible with pernicious anæmia ? In this disease we find similar anæmia to that of our patient, but the characters of the anæmia are more marked. The red corpuscles fall below a million ; the hæmoglobin coefficient is above the normal ; the blood contains myelocytes and nucleated red cells, which were absent in this case ; lastly, in pernicious anæmia the morphology of the red corpuscles is more profoundly modified.

Could the affection be a severe secondary anæmia due to lead, malaria, or syphilis ? These factors could all be eliminated. We had to find out whether the anæmia was due to an animal parasite, the ankylostoma, or the bothriocephalus.

Neither his trade nor his home was in favour of ankylostomiasis. Was he, then, suffering from anæmia due to the bothriocephalus ? On inquiry, he said that he had had “ a solitary worm ” for many years. A dose of castor-oil led to the expulsion of an intestinal worm 10 inches long and  $\frac{1}{2}$  inch broad, of a greyish colour. It had broad,

short segments, which were all similar. The genital pores were situated in the middle line. The head was almond-shaped ; it had no hooklets, but possessed two lateral slits. The slits contained innumerable eggs,  $70\ \mu$  in length, and  $45\ \mu$  in breadth. Each had a double wall, and a valve at one pole. The worm was a bothriocephalus.

Oil of male-fern was given, and some hours later he passed eight worms, varying in length from 4 to 12 inches. Next day he was much better, and had a good appetite. In a week he gained 3 pounds in weight. The red corpuscles rose to 3,000,000, while the white corpuscles were 4,000. Slides showed nucleated red corpuscles and myelocytes, indicating regeneration of the blood. He left the hospital in excellent health.

**General Description.**—In many persons the bothriocephalus may be present for many years, and only cause intestinal troubles and moderate anæmia. The scene at times changes suddenly, and cachexia is established within two weeks. Careful examination, however, shows that for two or three years the patient has suffered from general weakness, headache, vertigo, and buzzing in the ears, before the slight anæmia changed into marked cachexia.

The gastro-intestinal symptoms of cachexia due to the bothriocephalus simulate those of malignant cachexia, and thus render the diagnosis obscure. The rapid anæmia leads to a diagnosis of an essential pernicious anæmia. The colour of the patient is an ivory-white, and the mucous membranes are colourless. Anæmic murmurs are heard over the precordial region and the vessels of the neck. Retinal hæmorrhages often occur. In a few months, or even in a few weeks, the wasting is considerable. In about half of the cases slight œdema is present over the malleoli. At the same time the dyspeptic symptoms grow worse : the anorexia is constant, vomiting is frequent, and diarrhœa is commonly present.

The red corpuscles may fall below a million. The hæmoglobin is rarely diminished, and is never much increased. The corpuscles are not much deformed ; microcytes are numerous, while the macrocytes are said by Schaumann to be scanty. Normoblasts and megaloblasts have often been found, but they were not present in my case. At times the myelocytes show the participation of the bone-marrow in the blood regeneration. An increase of the eosinophiles has never been found during the stationary stage of the disease.

Certain differences in the blood-counts distinguish this disease from pernicious anæmia. Examination of the stools yields the eggs of the tænia, provided it has not been expelled. They are so numerous that their discovery is easy. Their appearance is so characteristic that doubt cannot exist. If the patient has just passed fragments of the worm, examination will give the diagnosis.

The pathogenesis of anæmia and cachexia due to the bothriocephalus is a matter of theory. It is evidently an intoxication, but the mechanism is imperfectly understood, and in spite of many experiments it is not easy



to say why the worm may be harmless for many years and then suddenly give rise to rapid cachexia.

We cannot explain, moreover, why in certain countries (Livonia, Finland) the bothriocephalus often tends to cause anæmia and cachexia, and why these troubles are rare in other countries.

**Trichina.**—The trichina is a parasite of the muscles, and only attains its perfect state in the alimentary canal. Trichinosis is common in pigs. In man the trichina ingested with the food is set free in the alimentary canal, gives birth to rectilinear trichinæ, which perforate the intestine, and lodge in the muscles.

**Tæniæ.**—The tæniæ which are met with in the intestine of man are the *Tænia inermis* and the *T. armis*, or *T. solium*. The following description is taken from Laboulbène's monograph : The patient brings in a bottle the fragments of a worm which he has passed. We can tell at once whether it is a case of *T. inermis* or *T. solium*. If the segments of the worm are separated or isolated, and if they are voided involuntarily and without the patient's knowledge, it is a case of *T. inermis*. If, on the contrary, the fragments consist of five or six to ten rings, and if they have not been passed without the patient's knowledge, but have been expelled in the stools, then it is a case of *T. solium*. The following are the distinguishing characteristics :

The *T. inermis* (*mediocanellata*), which is twenty times as frequent as the *T. armis*, looks like a ribbon of 12 or 15 feet in length. One of its extremities is pointed, and terminates in a slight swelling, which is the head. The head, or **scolex**, is provided with four suckers, by which the animal adheres to the intestine ; the body is formed of segments, of which the last ones are longer than they are broad, and are called **cucurbitins**, on account of their resemblance to a pumpkin-seed. They are filled with eggs. Each segment contains both male and female organs of generation, except the posterior segments or cucurbitins, in which the male organ is atrophied. The genital pores are found on the sides of the segments. The unarmed tænia of man comes from the cysticercus of the ox, or from measly veal. The *T. solium*, or armed tænia, is thinner than the unarmed tænia. Its head is provided with four suckers and hooklets, arranged in a double crown. Each of the segments forming the body of the animal contains both male and female organs of generation, and the genital pores are most frequently alternate, whilst they are placed in series on the same side in the unarmed tænia. *T. solium* comes from the cysticercus of the pig, in which it constitutes measles.

The **symptoms** occasioned by tænia are multiple. Amongst the digestive troubles I may mention salivation, vomiting, attacks of nausea, and gastro-intestinal troubles. Amongst the nervous troubles of a reflex

kind we see vertigo, laryngeal spasms, nasal pruritis, spasm, hiccough, and epileptiform convulsions. Eosinophilia has not the diagnostic importance which has been attributed to it.

**Anklostoma—Ankylostomiasis.**—The invasion of the system by duodenal ankylostoma is, also, marked by **pernicious progressive anæmia**. This anæmia has all the characteristics which we have just described when dealing with bothriocephalic cachexia. The eosinophiles are even more numerous. The absence of eosinophiles, however, must not prevent the search for the ova of the parasite in the stools of the patient.

This research is very easy indeed. It is only necessary to place a small quantity of faecal matter between the slides to see the characteristic ova.

This research must be carried out in the case of every anæmic patient coming from tropical countries (India, Central America, etc.), which, as is known, are devastated by ankylostoma. These countries are, likewise, ravaged by paludism and, in the presence of cachectic patients suffering



FIG. 46.—OVA OF ANKYLOSTOMA.

“The ova of the ankylostoma are of oval shape, and measure from  $55\mu$  to  $65\mu$  by  $22\mu$  to  $43\mu$ . Their delicately transparent covering allows the contents to be seen divided into two or four segments” (Jeanselme and Rist).

from paludism, coming from these countries, the search for the ova of ankylostoma in the stools is indispensable. Let us, however, not forget that Europe is not free from ankylostomiasis; that this curse afflicts a large number of coal districts in France (Calmette), Germany, Belgium, and in Austro-Hungary. The diagnosis of ankylostomiasis is made, almost solely, by the examination of the stools. The importance of the prognosis will be understood, because the disappearance of the parasites by appropriate treatment is quickly followed by the cure of the patient. After administering thymol, it is possible to examine the parasite, the description of which is given herewith, in the stools.

The ankylostoma is a nematode worm. The male, which is from 8 to 11 millimetres long, bears, at its posterior extremity, a trilobed sack, B, similar to an umbrella, which has eleven ribs. The female, which is somewhat thicker, has an obtuse tail. The vulva, V, opens towards the posterior third of the body. In both sexes, the body diminishes in size from the tail to the neck. This is provided with “a powerfully armed buccal capsule with a distinct protuberance. The free edge of this remarkable organ possesses four powerful hooks, similar to claws, and two conical teeth.”



The vermifuges generally employed are ethereal oil of male-fern, the root of the wild pomegranate-tree, kousso, and pelletierine. I obtain almost constant success with the male-fern, which I administer according to Trousseau's prescription. The method of administration is as follows:

The patient is placed on a milk diet for twenty-four hours. Next morning he takes fasting 12 to 15 capsules, each containing 5 drops of ethereal oil of male-fern. One capsule is taken every three minutes. A quarter of an hour after the last capsule he takes  $\frac{1}{2}$  ounce of castor-oil, and half an hour later he again takes  $\frac{1}{2}$  ounce. This is the method which

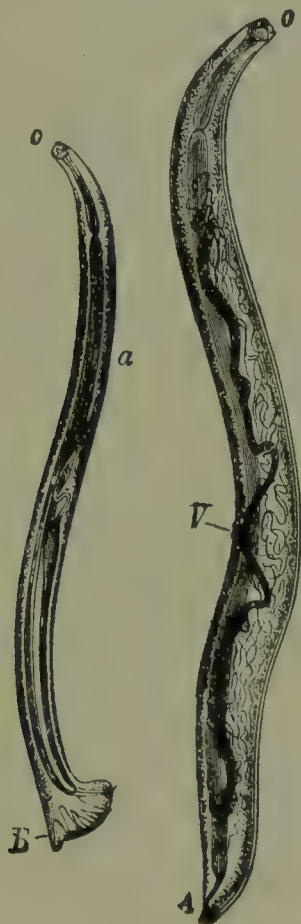


FIG. 47.—MALE AND FEMALE ANKYLOSTOMA.

I had inaugurated at the Necker Hospital, where I used to see every year more than a hundred patients suffering from tænia. It has succeeded in cases where other remedies have failed. The doses which I have given are for an adult, and must be modified in the case of a child.

Male fern gives good results in ankylostomiasis, but thymol is the best remedy. It is administered, according to the age of the patient, in doses of from 4 to 8 grammes daily, or in four days. Each dose should consist of from 0.25 to 0.5 centigrammes. The last dose must be followed by an oilless purgative. It must be remembered that thymol is toxic and, hence, must not be absorbed by the intestinal mucosa. The following precautions must, therefore, be taken: (1) Put the patient on a low diet for a day or

two ; (2) Give no medicine, no food capable of making the thymol soluble (fat, oil, ether, alcohol, chloroform). The urine must be carefully watched, and the remedy be discontinued on the slightest appearance of a blackish colouring.

In order to verify the effect of the medicine, it suffices to examine the fæces eight days after the administration of the thymol. If ova of the ankylostoma be still present, the treatment must be resumed.



## CHAPTER VI

### DISEASES OF THE PERITONEUM

#### I. INTRODUCTION TO THE STUDY OF PERITONITIS.

DURING the past few years a radical change has been brought about in the study of peritonitis. From the peritoneal chaos we have established clearly-defined types, which have taken their place in the nosological list, and have been substituted for the mistakes of the past. The old classification of peritonitis admitted two groups: on the one hand, secondary peritonitis, and on the other, so-called primary or idiopathic peritonitis. The causes of secondary peritonitis were perforations of the stomach, intestines, or biliary passages; lesions of the uterus and its adnexa, puerperal infection, tuberculosis, cancer, etc., were described. The commonest variety of secondary peritonitis—I mean appendicular peritonitis—was unknown. Grisolles wrote on typhlitis, perityphlitis, and iliac phlegmon, but appendicitis and appendicular peritonitis were passed over in silence. A few lines on perforation of the appendix by foreign bodies were inserted in the description of intestinal perforations.

In direct opposition to the incongruous group of secondary peritonitis, writers described the so-called primary or idiopathic peritonitis, which was believed to come *a frigore*, and which Grisolles had called “simple acute peritonitis.” This peritonitis served as the model for the classical description of peritonitis, and was called “acute peritonitis,” without any other designation.

This so-called simple or idiopathic peritonitis does not exist. Many of these cases are in reality appendicular peritonitis. We know how the toxi-infection elaborated in the closed cavity traverses the walls of the appendix, and causes acute peritonitis without obvious perforation. These facts were formerly unknown. The peritonitis was called simple or idiopathic, because the original focus in the appendix had remained unnoticed.

Writers also placed to the credit of simple acute peritonitis the peritoneal infections which are known to be caused by the pneumococcus and the streptococcus, the epithet “primary” being applied to cases in which the entrance-gate of the infective agent remained unknown.

It follows, therefore, that we cannot to-day give the general description of acute peritonitis. The description must be split up according to the origin of the peritonitis, because each variety may present a clinical picture which differs according to the cause.

**Puerperal Peritoneal Infection.**—The uterine cavity in the normal condition or after accouchement does not contain microbes; consequently, the usual agents of puerperal peritonitis—streptococcus, coli bacillus, or other microbes—must have been carried to the woman by the accoucheur, the midwife, the instruments, the linen, and the dressings, by attempts to procure abortion, or by erysipelas. The microbes pass through the lymphatic vessels eleven times out of twelve (Widal), and in some cases along the mucosa of the Fallopian tube. This variety of puerperal infection has become exceedingly rare.

Amongst the varieties of peritonitis peculiar to women and girls I may mention inflammation consecutive to pyosalpinx. Blennorrhagic peritonitis has been well described by Charrier. It usually begins with acute pain in the lower abdomen (Bernutz's cry of alarm). The onset often coincides with the end of the first menstruation which follows infection of the uterus by the gonococcus. In addition to the usual symptoms of peritonitis, local examination reveals induration in the culs-de-sac and fixation of the pelvic organs by adhesions. This perimetro-salpingitis, which is benign as far as life is concerned, is a possible cause of sterility (Charrier). Blennorrhagic vulvovaginitis in young girls is also a cause of peritonitis. The detailed description of blennorrhagic peritonitis will be given under Blennorrhagia.

**Appendicular Peritonitis.**—As I have described this form in detail under Appendicitis, I shall simply mention it here. General or local peritonitis and remote peritoneal abscesses are seen in the course of appendicitis. The appendix may or may not be perforated. I have explained the process when describing the **closed cavity**.

**Peritoneal Infection due to Perforation of Organs.**—This group contains cases of peritonitis resulting from trauma, perforation of the stomach (simple ulcer) and of the intestine (ulcer of the duodenum, typhoid and tubercular ulcerations). I shall here merely mention these varieties of peritonitis. They are discussed in detail in the sections on Ulcer of the Stomach, Ulcer of the Duodenum, Typhoid Fever, etc.

This group contains the varieties of peritonitis due to perforation of the bile-ducts. The bile itself is harmless and sterile, and the passage of aseptic bile into the peritoneum does not cause peritonitis. Acute peritonitis, however, develops if the bile is contaminated with pathogenic microbes (rupture of the gall-bladder or of the bile-duct, calculous obstruction).

The same remark applies to the urine. Physiological urine is inoffensive to the peritoneum, as to all other tissues, because it does not contain microbes. If the urinary tract is infected by the streptococcus or the coli bacillus, perforation of the bladder or of the pelvis of the kidney will be followed by peritonitis.

**Peritoneal Infection due to Purulent Collections.**—To this category belong the various kinds of peritonitis due to contact with or to rupture of perirenal suppurations (Albarran), abscesses of the liver, iliac phlegmons, inflammation of the abdominal wall, purulent pleurisy, etc.

**Primary Peritonitis.**—Primary, pneumococcal, and streptococcal peritonitis will be described later.

**Anatomy of the Peritoneum.**—It will be useful to note a few anatomical facts which are indispensable in studying diseases of the peritoneum.

The peritoneum, like every serous membrane, presents a parietal and a visceral layer. The parietal layer is more resistant and thicker. It is lined with cellular tissue, which plays an important part in certain regions. This cellular tissue is loose over the anterior wall, where it forms the **fascia propria**, and is abundant in the iliac fossæ and the pelvis, where it readily becomes inflamed. It is still more abundant around the kidneys, where its inflammation constitutes perinephritis. The parietal layer is easily separated from the diaphragm, and still more easily from the other regions which I have just enumerated.

The visceral peritoneum is thin and transparent, being so intimately united with certain viscera (spleen, liver, ovary) that it forms, as it were, part of their structure; but elsewhere it is separable as a thin sheet (stomach and intestines).

The peritoneum forms three varieties of folds in the abdominal cavity: (1) Folds which pass from the posterior walls of the abdomen to the digestive organs. The name of the organ into which they have been inserted, with the prefix **meso**, has been given to them, and in this way the names **mesocolon**, **mesorectum**, **mesentery** have originated. (2) Folds which proceed from the walls of the abdomen to organs other than those of the digestive tube. To these the name of **ligaments** has been given—



coronary ligament; right and left **triangular ligaments**, which pass to the liver; and **broad ligaments**, which pass to the uterus. (3) Folds which extend from one viscus to another one. They are three in number, and are called **omenta**. They are the small omentum, or gastro-hepatic omentum, which unites the stomach to the liver; the great omentum, or gastro-colic omentum, which unites the stomach to the transverse colon; and the gastro-splenic omentum, which passes from the liver to the spleen.

All these folds of the peritoneum are formed by the two layers of the serous membrane. Between the layers there is cellulo-fatty tissue in which the vessels and the nerves ramify.

In the normal condition the peritoneal cavity does not contain any micro-organisms.

**Description.**—As the symptoms of peritonitis vary according to the exciting cause, I shall sketch three principal types which answer fairly to the clinical pictures of acute peritonitis.

In the first type the peritonitis arises suddenly, and is acute or super-acute. It follows trauma, perforation of an organ (ulcer of the stomach or duodenum), opening of an abscess, rupture of the pelvis of the kidney or of the bladder, and perforating appendicitis. Violent pain, with or without rigors, opens the scene. The pain, which is at first local, extends rapidly to the whole of the abdomen. It is acute and at times agonizing, being increased by the least movement, contact with the bedclothes, cough, hiccough, vomiting, or intestinal peristalsis.

The abdomen soon becomes distended, and the **meteorism** is excessive. At the onset, however, the abdomen is **flat**, hard, and tense, and the muscles are contracted. **Hiccough** is frequent, and **vomiting** occurs at more or less close intervals. The vomited matter at first consists of mucus, and later of bile, and the patient, after much straining, vomits streams of bitter, greenish fluid (**porraceous vomiting**). Constipation is the rule. Dysuria is frequent, and the urine is scanty.

The fever is sometimes high, but the temperature may fall below normal. Fluid, in variable quantity, accumulates in the peritoneum. The general symptoms rapidly become more marked. On the second or third day the pulse becomes quick and thready; the tongue is dry and cracked, and there is great thirst. The face is thin and pinched, the nose is cold, the eyes are hollow, the weakness is excessive, the extremities grow cold, the respiration is short and jerky, the voice is thin and husky, and collapse is imminent. All these symptoms become accentuated, except the pain, which diminishes or disappears. The patient, not having lost consciousness, congratulates himself on the disappearance of the pain; but this calm is generally the prelude of death, and the sufferer dies speaking, as Grisolles said. In this variety of peritonitis recovery is the exception (except in the case of early surgical intervention). Death may supervene on the first, second, or third day (peritonitis due to perforation), though it rarely takes place before the sixth or seventh day. In some cases, especially in children,

a slow phase follows the sudden onset (pneumococcal peritonitis), and the pus often finds its way through the umbilicus. Recovery may then occur.

In the second type we have still to deal with acute diffuse peritonitis, but the complication appears in subjects who are **already sick**, and suffering, for example, from typhoid fever. In such a case the symptoms of peritonitis are not very marked, and the reaction is less acute. The pain, hiccough, and vomiting are not as severe as in the former type. A sudden fall in the temperature is frequent, and the abdominal meteorism is very marked.

In the third type the different symptoms of acute peritonitis are less pronounced, and the chief feature is the **general condition** of the patient, when a rapid tendency to collapse (primary streptococcal peritonitis) is seen.

**Partial Peritonitis.**—Partial peritonitis has its seat of election in the iliac fossæ, the subphrenic region, and the peri-uterine region. It is neither so acute nor so grave as general peritonitis, and often causes an abscess in one of the regions just mentioned. Abdominal palpation and vaginal and rectal examination will aid in the diagnosis of partial peritonitis. The description of these varieties of encysted peritonitis will be found under Appendicitis and Simple Ulcer of the Stomach.

**Chronic Peritonitis.**—General peritonitis does not pass into the chronic state. The chronic form is seen in partial peritonitis and in **tubercular or cancerous** peritonitis.

**Pathological Anatomy.**—In acute peritonitis the peritoneum is found to be injected, thickened, and infiltrated with pus. The fibrino-purulent infiltration exists in the folds of the great omentum and of the mesentery, in the connective tissue of the serous membrane, and upon its surface. Fibrinous adhesions rapidly form between the intestinal coils, as well as between the various organs of the abdomen and the parietal and visceral layers. The great omentum is thickened, vascular, and adherent to the intestines and to the abdominal wall. The false membrane is covered with puriform mucus. A quantity of fibrino-purulent liquid is found in the peritoneal cavity, and varies in amount from a few ounces to several pints. This liquid is sometimes very foetid. To these lesions must be added the changes in the organs which have caused peritonitis.

**Treatment.**—There is no medical treatment for acute peritonitis. The pain is treated with injections of morphia. Ice-compresses are applied to the abdomen, and ice is given to the patient to suck. Surgical intervention is the only real treatment. We must act, however, without delay, and if too much precious time has not been lost in medical treatment, the greater are the chances of saving the patient. This question of surgical treatment is given in detail under Appendicitis, Ulcer of the Stomach and of the Duodenum, Primary Pneumococcal and Streptococcal Peritonitis, Peritonitis in Typhoid Fever, etc.



## II. ACUTE AND SUBACUTE TUBERCULAR PERITONITIS.

Tubercular peritonitis may be partial or general and acute or chronic. I shall first consider acute and subacute peritonitis.

**Peritonitis due to Perforation.**—A patient is suffering from tubercular ulceration of the intestine. The sufferer is seized with symptoms of acute peritonitis from perforation; he has terrible pain in the abdomen, vomiting, hiccough, etc., and succumbs in a few days. Post mortem, general peritonitis due to perforation of a tubercular ulcer of the ileum is found. Although the peritonitis is not tubercular in the true sense of the word, because it is not due to the tubercle bacillus, but to virulent microbes which have passed from the intestine into the peritoneum, it is none the less true that a tubercular lesion of the intestine has caused the perforation, so that we may clinically place this variety under Tubercular Peritonitis.

It must not, however, be supposed that these cases are common. As far as I am concerned, I have never seen one. Tubercular ulcerations of the intestine, though common, **very rarely** cause perforation and peritonitis. Barbe, in his thesis on perforation of the small intestine, only mentions two cases, and adds that the average of these perforations in tubercular patients amounts, according to Potain, to 2 per cent.

**Miliary Peritonitis.**—The tubercular process does not affect the peritoneum alone, but forms part of a more or less general miliary tuberculosis. An acute febrile disease which often simulates typhoid fever in adults and meningitis in children declares itself. The typhoid symptoms in the one case and the meningeal symptoms in the other are so marked that the peritoneal symptoms pass almost unnoticed. They may, however, be fairly severe, and the abdominal pain, porraceous vomiting, meteorism, and ascites prove the presence of peritonitis. The patient succumbs, and at the autopsy we find a small quantity of sero-fibrinous or sero-purulent liquid, which is rarely hæmorrhagic, in the peritoneum. The peritoneal cavity is not partitioned off by false membranes, and we find on the layers of the peritoneum (especially on the parietal one) small, discrete, or confluent tubercles, which resemble grains of semolina, and are often surrounded by ecchymotic patches. The intestinal coils are more or less congested and matted together by fibrinous exudate. Miliary tubercles are likewise found in the lungs, pleura, meninges, pericardium, etc. The peritonitis is, therefore, merely a local manifestation of general tuberculosis (tuberculosis by blood infection). As Marfan says, when miliary tuberculosis attacks young people, "the bacillus seems to have a certain affinity for the peritoneum, pleura, meninges, and pericardium."

**Subacute Pleuro-Peritoneal Tuberculosis.**—In the adult the course of events is somewhat different, and we often see a subacute variety, which takes its origin in tuberculosis of the lung, and invades successively the pleura and the peritoneum. This variety, described by Fernet under the name of "Subacute Pleuro-Peritoneal Tuberculosis," is much less common in children than in adults. It is much less severe than general tuberculosis, the fever being less marked, the symptoms more moderate, and the course less rapid. Even the origin is different: general tuberculosis is the result of infection through the bloodvessels, but in pleuro-peritoneal tuberculosis the bacilli seem to follow the lymphatic passages, and pass from the pleura to the peritoneum through the diaphragm.

In one case of pleuro-peritoneal tuberculosis we were able to trace the extension of the tuberculosis from the pleura to the peritoneum through the lymphatic tracts in the diaphragm.

Reference to the published cases shows that the patient usually had well-marked disease in the pleura when the peritonitis appeared. The symptoms of pleurisy are at times quite evident; in other cases the pleural stage is latent, and it is only discovered when the alarm has been given by the peritoneal symptoms.

In this variety, as in the other varieties of tubercular pleurisy, we find more or less abundant fluid, which may be hæmorrhagic, but very rarely purulent, except in recurrence of the effusion after thoracentesis, etc.

The peritoneal stage is rather insidious than acute. In the latter event we find pain, fever, vomiting, distension of the abdomen, and ascites; in the former the peritonitis is not very painful, and betrays itself only by digestive troubles and enlargement of the abdomen. The liquid is generally free in the peritoneal cavity. It is usually yellow in colour, and may amount to 8 or 10 pints.

We find post mortem not only the lesions of pleuro-peritoneal tuberculosis, but also tubercular lesions in the lungs and liver, perihepatitis, perisplenitis, etc.

The diagnosis of pleuro-peritoneal tuberculosis is often very difficult, because hepatic cirrhosis may produce peritoneal and pleural effusion. In cirrhosis, however, pleural or peritoneal pain is absolutely wanting, a collateral circulation is established, and the patient has signs of cirrhosis or of precirrhosis.

The laboratory measures described under Tubercular Pleurisy may be employed in the diagnosis of peritoneal effusions. Although the peritoneal fluid does not lend itself as well to investigation as the pleural effusion, it nevertheless supplies valuable information. I have several times made use of cyto-diagnosis, sero-diagnosis, and of injections of the liquid into the mamma of a nursing guinea-pig.



**Summary.**—I have just reviewed three varieties of tubercular peritonitis. The first is superacute, and very rare. It results from perforation of a tubercular ulcer. The second is acute, and fairly frequent in children. It is characterized by peritonitis, which forms part of a general infection. The third or subacute variety is often curable. It is usually found in adults, and is characterized by the pleuro-peritoneal syndrome.

### III. CHRONIC TUBERCULAR PERITONITIS.

Chronic tubercular peritonitis comprises three varieties of lesions: (1) more or less abundant peritoneal effusion; (2) caseous, ulcero-caseous, or fibro-caseous lesions; and (3) fibro-adhesive lesions. This cicatricial process has a curative tendency, although it sometimes oversteps its object, and creates vicious adhesions. These three kinds of lesions are found in every case of chronic tubercular peritonitis. Sometimes, however, one of them is so marked that it gives a special character to the chronic peritonitis, and I shall therefore describe an ascitic and a fibro-caseous form.

#### A. Ascitic Form.

In some cases the effusion is so large that the condition at first sight appears to be ordinary ascites. These cases of chronic peritonitis, in which the peritoneal effusion is the dominant symptom, may be compared with those cases of tubercular pleurisy in which a large effusion is the only sign. The ascitic form is far more common in children than in adults; it is, indeed, the **most common** form of tubercular peritonitis in youth.

**Pathological Anatomy.**—Abdominal surgery has revealed the following lesions: Abundant fluid, which is citron-coloured, transparent, and hardly ever sero-purulent or sero-sanguineous. The quantity of liquid is rarely as large as in hepatic ascites, and rarely calls for tapping. The liquid is free in the peritoneal cavity, which presents no septa. The peritoneum is injected, dull, and covered in places with fibrinous exudate. Tubercles of every size and age are found on the peritoneum, which is more or less vascular. These greyish or yellowish granulations are superficial, or inserted into the wall. Caseous and fibrous lesions are absent in most cases.

**Description.**—The onset is often febrile in infants, the temperature rising to 101° or 103° F. The child complains of colic, nausea, and vomiting, and the abdomen is distended and painful. Examination reveals ascites without dilatation of the abdominal veins, and sometimes a little liquid in the pleura. After this acute onset the disease enters on a more silent stage, in which the pains and the fever disappear, and the ascites alone

persists, sometimes increasing, sometimes diminishing, but without reaching—in the child, at least—such proportions as to necessitate tapping. During this stationary stage, which may last several months, the child grows pale and thin, the appetite is poor, and yet the condition does not grow worse, because the other organs generally remain healthy. The ascitic fluid is often absorbed spontaneously, and cure occurs in more than half the cases. The disease, therefore, well deserves its name of **benign chronic tubercular ascites of youth** (Marfan). In some cases, however, which are rarer in children than in adults, fibro-caseous peritonitis may be associated with the ascitic form, and when the peritoneum is tapped, thickening of the omentum and of the peritoneum is found.

The **diagnosis** of chronic tubercular ascites is often very difficult, because ascites due to cirrhosis and cardiac or renal disease may present much analogy to it. In these different varieties, however, fever and pain are absent at first. Furthermore, in cirrhotic ascites the liver is small, and the collateral circulation is well developed. In cardio-hepatic ascites the causative lesion is found in the heart, and, finally, in the ascites of Bright's disease we can discover symptoms of Brightism and albuminuria. The diagnosis of tubercular ascites may be confirmed by cyto-diagnosis, sero-diagnosis, and injection of the ascitic fluid into the mammæ of guinea-pigs.

### B. Chronic Peritonitis—Fibro-Caseous Form.

This is the usual form in adults, and is chronic tubercular peritonitis, without other designation.

**Pathological Anatomy.**—On opening the belly, the viscera may be hidden by thick, greyish false membranes, forming tumours or nodules that may have given rise to errors in diagnosis. The peritoneum, the mesocolon, the mesentery, and the great omentum are much thickened. The thickening is due to sero-purulent infiltration, tubercular granulations, and embryonic tissue between the layers of these membranes. In some cases the thickened and indurated omentum forms, as it were, a transverse band, which is clearly felt during life.

The anterior abdominal wall is often adherent to the omentum and the intestines. Adhesions are formed between the layers of the peritoneum, between the omentum and the intestines, and between the intestinal coils, which are matted together, and cannot always be separated without tearing. After separation of these adhesions, little cavities, filled with serous fluid and altered blood, or with pus mixed with fæcal matter, are sometimes met with. Cavities filled with caseous matter, forming cold abscesses in the peritoneum, may also be found. The diameter of the intestine is contracted, and its walls are sodden, friable, and atrophied. The intestinal



canal may be only one-third of its normal length (Grisolles). Tubercular ulcerations are found on its surface. The omentum becomes retracted, and the mesentery draws the small intestine towards itself. This matting of the intestinal coils may simulate a tumour situated above the umbilicus. The false membranes may compress the portal vein, and cause thrombosis.

Perforations and communications between two adherent coils of bowel are sometimes met with. In exceptional cases, when the adhesions have not had time to establish themselves, the rupture of an ulcer may cause acute peritonitis.

The peritoneal effusion is not, as a rule, abundant; sometimes it is absent. The effusion is serous, sero-purulent, and flaky. It encysts itself in the dependent parts, and becomes caseous. The fluid may in some cases have a hæmorrhagic tint. There are at times true hæmorrhages into the peritoneum. Baumgarten reports a case in which the hæmorrhage was so copious as to form large clots in the abdominal cavity.

The vessels and the lymphatic glands always take part in the tubercular process. The glands are sometimes so large (mesenteric phthisis) that they may cause œdema of the lower limbs from compression of the vena cava. Tubercles are often found in the other organs of the abdomen (liver, spleen, female genital organs, bladder and ureters in children). The lungs often contain tubercles, which are few in number and of recent growth. When the tubercular lesions end in a fibro-adhesive process, almost complete adhesion is found between the intestines and the abdominal wall. The omentum retracts the transverse colon and the stomach, while the mesentery draws the small intestine backward, thus favouring intestinal occlusion.

**Description.**—Tubercular peritonitis is almost always chronic from the first, and we may say that, with the exception of a few cases and cancer excluded, it sums up the history of chronic general peritonitis. In twelve cases of chronic peritonitis, says Grisolles, eleven were tubercular. The disease chiefly affects young people and adults, and not patients with advanced phthisis, but those who are in the early stage. As a consequence, the abdominal lesions assume a **predominant importance**, and the peritonitis seems to represent the entire disease, whereas it is sometimes only an episode in phthisis.

In some cases chronic tubercular peritonitis is preceded by an acute or subacute phase, but generally it is **chronic from the outset**. The patients complain of abdominal pains, with alternate diarrhœa and constipation. The abdomen is sensitive to pressure; meteorism and ascites are present. The peritoneal effusion is not abundant, and exploration of the abdomen reveals a feeling of induration, especially in the umbilical and hypogastric regions. The induration is due to matting of the intestinal coils. The fibroid thickening of the omentum gives rise to an **omental**

**band**, stretching transversely from one hypochondrium to the other. The liquid in the peritoneal cavity does not vary in position, and is often encysted by the false membranes. At the points where the membranes glide over one another a kind of **rustling** is perceptible both on palpation and auscultation.

The **digestive functions** are impaired from the first (vomiting, diarrhœa, and lientery); the appetite fails, and progressive wasting follows. The general symptoms of fever, sweating, œdema, and cachexia are especially marked when phthisis is also present. The **course** and **duration** of the disease depend greatly on the state of the lungs. Chronic tubercular peritonitis runs a slow course, that is sometimes interrupted by subacute attacks. It lasts from one to two years, and often has a fatal ending, but **recovery is possible** when the disease is limited to the abdomen, and the lung remains healthy.

**Peritoneal Abscesses and Fæcal Fistulæ.**—During the course of chronic peritonitis fistulæ and peritoneal abscesses may form in the neighbourhood of the liver, of the spleen, of the cæcum, in the pelvis, in the omentum, or between two intestinal coils. These encysted abscesses contain pus and caseous matter. Fever and other symptoms may be absent, as in a cold abscess. The fever often declares itself with rigors, evening rise of temperature, pain, and vomiting. These infectious attacks may terminate in resolution, but they more often end in the perforation of a neighbouring organ, and the evacuation of the focus by the ileum, colon, cæcum, or bronchi.

In some cases the intestinal matter enters the open fistula, and makes its exit in the umbilical region, or elsewhere. Cutaneous fistulæ, discharging fæcal and purulent matter, are thus formed.

**Intestinal Occlusion.**—Lejars, in speaking of the causes of fæcal obstruction in the course of tubercular peritonitis, describes four kinds. In the first variety the intestinal occlusion is due to a band which strangles the intestine. These fibrous bands are very common in tubercular peritonitis. The strangulation may affect the small or the large intestine. Occlusion by a band may supervene at any stage of tubercular peritonitis; it may be the first indication of tubercular peritonitis which has been previously latent. Occlusion may also occur when the tubercular peritonitis is cured, the fibrous band being a relic of the peritonitis, as in the following case:

A young woman had been operated on by Marchant and cured. Four years later she came to me at the Necker Hospital for intestinal occlusion. An operation was performed by Routier, who found strangulation due to a band, and the patient recovered.

In the second variety we have intestinal occlusion by kinking of the intestine. Lejars has figured a very good illustration of this, in which the intestine is astride a band, occlusion being the result. Here also the symptoms of occlusion were the first sign of latent tubercular peritonitis.



In the third variety the occlusion is due to matting of the intestinal coils. In this matted lump we find adhesions, bands, kinks, and compression by tubercular masses or by an encysted collection. Surgical intervention, which is so favourable in the preceding varieties, is here rarely successful.

In the last variety intestinal paralysis is present. The fæcal matter no longer passes, owing to the absence of peristalsis, and we have a pseudo-strangulation—frequent, by the way, in all varieties of peritonitis.

The symptoms of intestinal occlusion in tubercular peritonitis may be sudden or slow in their appearance (Lejars). When this occlusion takes place suddenly, it is almost always the first signal of **latent** tuberculosis of the peritoneum. “The patient is seized suddenly with intestinal occlusion, and there is no suspicion of tubercular peritonitis. The abdomen is opened, and we find a band, a kink, paralysis of the intestine, or a crop of tubercular granulations, especially in the parietal layer of the peritoneum.” This proves once more that tubercular peritonitis is often latent during the first phase of its evolution. The symptoms of sudden occlusion are complete arrest of fæces and gas, ballooning of the abdomen, vomit of varying composition including fæcal matter, abdominal pains, and subnormal temperature. Nevertheless, tympanites and fæcal vomiting may be absent if the intestinal occlusion is very high up, as in the duodenum, for example.

Intestinal occlusion of the **slow type** is chiefly seen in confirmed tubercular peritonitis. It commences with constipation, and tympanites then sets in. Temporary improvement occurs after each fresh attack. The situation becomes worse, and the occlusion complete. This slow form is almost always due to matting of the intestines, and offers, therefore, less chance to the operator than the preceding variety.

**Diagnosis.**—While the diagnosis of chronic tubercular peritonitis is very difficult at first, it is far more difficult later, when the abdominal walls become deformed, and indurations appear, with or without ascites. Tubercular peritonitis is sometimes so insidious that it is hard to define its existence, because the patient's health is excellent, and the lungs appear to be healthy.

We shall see later that the distinction between hydatid cysts of the peritoneum and tubercular peritonitis is sometimes very difficult.

Chronic tubercular peritonitis is not to be confounded with the peritonitis of alcoholics, and with the rare cases of peritonitis in Bright's disease.

When the effusion is great, symptomatic ascites from **atrophic cirrhosis** may be thought of. In cirrhosis the ascites is not encysted, the fluctuation is not limited, and the collateral circulation is well marked; the liver is small, and the spleen is hypertrophied. On the other hand, in the ascitic form of tubercular peritonitis the indurated and retracted

omentum sometimes gives the feeling of a tense band in the umbilical region (Aran). Finally, it must be remembered that tubercular peritonitis and cirrhotic tuberculosis of the liver may coincide, and the diagnosis is then particularly difficult (Hanot).

In the dry form of the disease the masses of false membranes or the matted intestinal coils may simulate a solid tumour or an ovarian cyst. The diagnosis from ovarian cyst, though generally easy, is in certain cases a matter of great difficulty. It will be sufficient to recall the celebrated case of Spencer Wells, who performed laparotomy for an ovarian cyst, and found tubercular peritonitis. The fluid was evacuated, the wound sutured, and the patient recovered.

We must also exclude cancer of the peritoneum. In this case the yellow, straw tint is characteristic, the inguinal glands are often enlarged, the abdominal pains are very sharp, and if effusion supervenes, it is generally hæmorrhagic.

**Ætiology.**—Tubercular peritonitis generally attacks children between six and twelve years of age. It is not uncommon in adults, and especially amongst young soldiers, but it is exceptional in old people. Privation, overwork, insufficient food, traumatism of the abdominal region, chills, and alcoholism favour the onset of the disease.

Tubercular peritonitis is due to Koch's bacillus. How does the tubercle bacillus reach the peritoneum?

When I undertook the discussion of this subject at the Faculté, I had arrived at the same conclusion as Marfan: It seems at first sight that the tubercular ulcers in the intestine would be the most likely point of origin for peritonitis. This opinion has been upheld by many authors (Grancher, Koenig), and yet I cannot accept it. However paradoxical the assertion may seem, I do not think that the peritonitis arises from the lesions in the intestine.

Clinical medicine teaches us that many patients suffer from tubercular peritonitis, and yet they present none of the symptoms of tubercular enteritis. On the other hand, we find tubercular enteritis in phthisical patients who do not suffer from peritonitis.

Pathological anatomy teaches us that most phthisical patients who die with ulcers in the gut have neither general nor local peritonitis. We certainly find infected lymphatics proceeding from the intestinal ulcers, as well as chains of caseating mesenteric glands, but no peritonitis—or, at least, only slight traces of peritonitis—are met with. In favour of this assertion, I know of no more convincing argument than the work of Tchistovitch. In his monograph on "Intestinal Tuberculosis in Man," Tchistovitch, although he is not dealing with peritoneal tuberculosis, discusses the way in which intestinal infection is brought about, and the migration of the tubercle bacilli through the intestinal walls is effected by way of the lymphatics.



I have studied the account of the autopsies on which this work is based, and I find that the intestine (ileum, cæcum, large intestine) was the seat of deep, extensive, and serpiginous tubercular ulcers in seven cases; but though they were in appearance so favourable to the development of tubercular peritonitis, no trace of it was present. In the three other cases, in which no tubercular lesion of the intestine was found, tubercular peritonitis was present. It is true that in these three last cases tubercular pleurisy (pleuro-peritoneal tuberculosis) was also present.

From a careful study of these ten autopsies I draw the following conclusions: chronic tubercular peritonitis is not brought on by intestinal tuberculosis, and the infection travels from the pleura to the peritoneum by the lymphatics which pass through the diaphragm.

**Treatment.**—We must remember that chronic tubercular peritonitis, when primary, is often cured without surgical intervention by hygienic and medicinal measures (superalimentation, injections of cacodylate of soda, etc.). When I say that chronic tubercular peritonitis is often curable by medical and hygienic means, I include both the ascitic form seen in young subjects, and also tubercular peritonitis in adults, in whom we find peritoneal effusion, fibro-caseous lesions in the omentum and mesentery, peritoneal thickening, and intestinal matting.

I remember a cavalry officer who suffered from chronic peritonitis, with peritoneal abscess and a peri-umbilical fistula. He was completely cured. A few years ago, in company with Berger and Barth, I saw a young girl suffering from chronic peritonitis, with peritoneal matting and false fluctuation in the right iliac fossa, and all these lesions ended in a cure without any surgical intervention. For some two years I attended a young woman suffering from chronic tubercular peritonitis. In this case hygienic measures, cod-liver oil in large doses, salt baths, and superalimentation brought about marked improvement. In consultation with Hammon, I saw a young girl with chronic tubercular peritonitis of a febrile type and grave prognosis, but she is now convalescent.

In other circumstances, however, surgical intervention is indicated. The question has been ably discussed by Legueu, whose conclusions are: The ascitic type of chronic and subacute peritonitis is naturally the one in which the best results are obtained. In children below fifteen years of age, 40 operations resulted in 35 cures, 4 of which remained so after one year, 1 after two years, and 2 after fourteen years. In adults, out of 131 cases, 99 cures were obtained, 56 of which remained so after one year and 25 after two years. Out of the total cures (children and adults), recurrences have been observed seventeen times.

The fibro-caseous type is less suited to surgical intervention. Out of twenty-two patients operated upon, thirteen were cured, and in several instances the cure has been perfect, except for a fæcal fistula. The sclero-adhesive type gave, on an average, a cure in a third of the cases.

**Summary.**—In well-selected cases, when the indications are well-defined, surgical intervention has given most encouraging successes, and saves patients from certain death. There are, however, cases in which laparotomy is not indispensable, and tapping of the peritoneum, followed by injections of various kinds, has given good results.

In 1890 Debove reported a case of tubercular peritonitis cured by tapping and the injection of 3 pints of a warm saturated solution of boric acid. In 1893 Rendu reported a case of peritoneal tuberculosis cured by injections of camphorated naphthol. In 1895 Caubert obtained a cure by means of lavage with sterilized water at a temperature of 115° F. I would add, however, that **medicated** injections into the peritoneum are to be avoided as a routine practice.

#### IV. PRIMARY PNEUMOCOCCAL PERITONITIS—ASSOCIATION OF THE COLI BACILLUS.

**Pneumococcal peritonitis** is almost always **primary**, especially in **children**. It breaks out in healthy subjects who have had no other pneumococcal infection. The secondary form, which is far less common, is most often met with in adults. A moment's thought will show how exceptional is the appearance of pneumococcal peritonitis in pleurisy or pneumonia. I have seen it twice in patients suffering from general pneumococcia. In one of the patients we were able to prove that the peritonitis was on the road to recovery, thus showing that the secondary form may terminate by resolution. Secondary peritonitis figures only in five out of twenty-seven cases which I have collected, the peritonitis being primary in twenty-two.

This is not because the pneumococcus does not reach the peritoneum. In many fatal cases of pneumonia the pneumococcus has been found in the peritoneum. But it seems, says Boulay, that this serous membrane opposes the development of the pneumococcus, or, at least, that the microbe does not flourish on it. Thus, in a man already infected with a pneumococcal lesion, the pneumococcus may emigrate to the peritoneum, but it does not cause peritonitis. A similar thing occurs in experiments. An animal dies after inoculation, and though pneumococci are found in its peritoneum, no peritonitis is present. When pneumococci are injected into the peritoneum of a guinea-pig or of a rabbit, the animal dies of general infection, but peritonitis is usually absent.

The following case \* is one of primary pneumococcal peritonitis :

A little girl eight years of age was in perfect health on November 25, 1896. She ate her supper and went to bed at nine o'clock. About two o'clock in the morning

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\* This case is taken from my lecture on "Pneumococcal Peritonitis" (*Clinique Médicale de l'Hôtel-Dieu*, p. 396).



she was awakened by sharp pain in the belly, and commenced to vomit. The vomiting was incessant. At first food and then bile were rejected, but later the vomit became greenish. Profuse diarrhœa soon appeared, the motions being very frequent. Dr. Schmidt saw the patient, whose symptoms led him to think of appendicitis. The presence of the diarrhœa and the absence of well-defined local symptoms in the appendicular region induced him to reserve his diagnosis. Next day the child was able to get a few hours' sleep; the vomiting ceased, but the diarrhœa persisted. On the morning of the 27th the child's temperature stood at 100.5° F. Rénon, who saw her at six in the evening, found the belly slightly distended, with general tenderness on pressure. The diarrhœa persisted. In spite of the absence of spots, the possibility of typhoid fever was thought of, and cold compresses were applied to the abdomen. On the 28th the diarrhœa still persisted. The general condition was satisfactory, and the temperature morning and evening was 101° F. On the 29th rectal tenesmus, which came on two evenings previously, became so painful that it provoked a desire to stool five or six times every hour. The pains in the abdomen were stationary, and the urine did not contain albumin. Typhoid fever was feared more and more, and to decide the point sero-diagnosis was performed, with a **negative** result.

What was the matter with the child? The disease continued its course. During the early days of December, the abdomen being more distended and indurated, especially in the right iliac fossa, the diagnosis was appendicitis complicated with a cæcal abscess. I was called in consultation with Routier, and on December 13 we went to see the little patient, with Schmidt and Rénon. When I saw the child I was surprised to find her looking so well. She certainly had no peritoneal facies, and gave accurate information concerning her condition. The abdomen was more distended on the right side than on the left. The impaired resonance was extensive, but it was difficult to define. Palpation gave the sensation of a huge liquid collection, which bulged under pressure. From the onset and the course of the disease I suggested pneumococcal peritonitis, basing my diagnosis on the age of the child, the sudden onset of the disease, the diarrhœa, and the presence of a peritoneal collection, which, though extensive, seemed to have a tendency to become localized. I suggested an exploratory puncture in order to ascertain the virulence of the pneumococcus. The puncture was made on the following day, December 14, at eight o'clock in the morning by Routier, who drew off 2 pints of creamy pus. It was homogeneous, greenish, odourless, and contained a number of fibrinous flakes. The bacteriological examination, which was carried out an hour later, revealed the presence of the pneumococcus in a pure state. A mouse inoculated with two drops of pus died in forty-eight hours, and its blood contained pneumococci. The diagnosis of pneumococcal peritonitis having been verified, we decided to perform immediate laparotomy, and Routier did so. A few weeks later I saw the child, who had completely recovered.

**Description.**—Pneumococcal peritonitis is rare in the adult, and I find it noted only five times in twenty-seven cases. It is especially frequent between the ages of three and twelve years, and is far more common in girls than boys. This difference may perhaps be due to vulvitis (Marfan), which has been noticed in several cases. On the other hand, as the pneumococcus has been found in the uterine cavity (Boulay), "it is permissible to ask," says Brun, "whether pneumococcal peritonitis does not have the same pathogenesis as other kinds of peritonitis—passage of the micro-organisms from the uterus into the peritoneal cavity by the lymphatics or by the tubes. However hypothetical this opinion may be, it may rest on this fact—that in all cases where the localization of the lesions has been clearly

defined, it is always in the lower portion of the serous membrane, the iliac fossa, and the true pelvis, that they are met with." However seductive this theory may be, it takes no account of pneumococcal peritonitis in boys.

The present description refers to primary pneumococcal peritonitis in children, for the disease behaves somewhat differently in adults, being more frequently secondary, and more subject to extra-peritoneal complications; it may be accompanied by multiple thromboses (Sylvian, radial, and popliteal arteries).

The first point to mention is the **sudden onset**, in the midst of excellent health, with sharp pain in the belly, comparable to the **pain in the side** of pneumonia. The cases collected in my clinical lecture show in almost every instance this sudden onset, with pain and fever. The little patient to whom I have referred had eaten a good supper, and had gone to sleep quietly about nine o'clock. About two o'clock in the morning she was awakened by sharp abdominal pain, followed by vomiting.

Let the pain be situated in the right iliac fossa, as it often is, and it is clear that at first sight pneumococcal peritonitis, with its pain and vomiting, closely simulates appendicitis. We shall see later how to differentiate them. The onset of the disease is generally accompanied by fever. The sudden rise in temperature ( $102^{\circ}$  or  $103^{\circ}$  F.) has even been compared with the similar rise of temperature in pneumonia. The comparison is not absolutely justified, for though there are cases of peritonitis in which the temperature is raised from the outset, there are others in which during the first two days the temperature is not above  $99^{\circ}$  F.

The onset of pneumococcal peritonitis is further characterized by **vomiting** of food and bile, which is never absent, and which may be incessant. I do not find hiccough mentioned in any of these cases—a strange fact, because it is a common symptom in other kinds of peritonitis. On the other hand, in almost every case there is special mention of **diarrhœa**, which is a very exceptional symptom in other forms of peritonitis. Though I am unable to explain the cause of this diarrhœa, it is certain that its constancy and its characteristics are important in pneumococcal peritonitis. It has an especial value from a diagnostic point of view.

It may, consequently, be said that pain in the belly, vomiting, and diarrhœa, with more or less high fever, form the characteristic syndrome of pneumococcal peritonitis, and the recognition of this syndrome simplifies the diagnosis considerably. The early diagnosis of pneumococcal peritonitis is not free from difficulties, chiefly because we are liable to forget it. The onset is much like that of appendicitis, and in most cases appendicitis is at first thought of. This is the more easily understood as the early age at which pneumococcal peritonitis is seen is the usual age for appendicitis,



Nevertheless, close inspection shows that there is a difference between the two affections. Though the pain of pneumococcal peritonitis may commence in the iliac region, it is not, as a rule, clearly marked at McBurney's point. Furthermore, diarrhoea, which is hardly ever absent at the onset of pneumococcal peritonitis, is very rare in appendicitis.

**Evolution of Pneumococcal Peritonitis.**—For some days the symptoms show but little change. The temperature is about 102° F., the abdominal pain may persist or grow less, and the fits of vomiting may cease; but the diarrhoea, as a rule, continues. On the fourth, fifth, or sixth day of the disease we find fever, diarrhoea, abdominal pains, and ballooning of the belly; but if we take care to find out our position by considering the **syndrome of the onset**, the idea of typhoid fever may be dismissed. It must be admitted that this diagnosis might be somewhat embarrassing—at least, for a few days—did not Widal's sero-diagnosis remove all doubt.

As the disease goes on new symptoms appear. In this phase the fever has sometimes disappeared; it may, indeed, have ceased as suddenly as in pneumonia. This sudden defervescence, considered by some authors as a sign of pneumococcal peritonitis, is wanting in many cases. In proportion as the abdomen becomes swollen, the dullness generally limits itself to the hypogastric region and to the flanks. Palpation reveals induration and resistance in the same region, and fluid is noticed. The diagnosis of peritonitis is then obvious, and if the peritonitis is limited to the right ilio-inguinal region, the diagnosis of appendicitis with peritoneal abscess is naturally made. One sign at this period of the disease is, however, of great value—viz., the unfolding of the umbilicus, which becomes red and prominent. The abscess bursts, giving exit to a variable quantity of pus.

In many cases pneumococcal peritonitis causes an umbilical fistula. Three, four, or five weeks after the onset, a reddish, shiny, fluctuating swelling of the size of a nut appears in the umbilical region. If intervention is delayed, the abscess opens, and the pus in the peritoneum is evacuated, so to say, by a "peritoneal vomica." The pus is generally greenish, creamy, inodorous, and mixed with false membranes. Spontaneous opening at the umbilicus is found in half the cases, but it might be avoided by timely operation. In some cases spontaneous opening has occurred not only at the umbilicus, but also in the vagina (Brun, Pochon). The fluid is sometimes very abundant, and as much as 5 pints have been found in the abdomen of a child eight years of age. The fluid has not, however, the mobility of ascitic fluid, and collects in foci, limited by thick false membranes.

Primary pneumococcal peritonitis may be cured by spontaneous evacuation of the pus, just as some cases of purulent pleurisy recover by vomical. In less favourable cases it causes pneumonia, pleurisy, endopericarditis, or otitis, and death is the result. Hence the rule of operating in pneumococcal

peritonitis before the infection has had time to become general. In some cases the disease, if left to itself, drags on: the pus does not make its exit, the abdomen remains large, and the fever returns every evening, while the child grows thin and cachectic, as in phthisis (Brun). Tubercular peritonitis is incorrectly diagnosed, and, for want of proper information or of timely operation, the patient is allowed to die.

Such is the course of pneumococcal peritonitis. I have already indicated the diagnosis in each stage of the disease, but I return to this subject in order to add a few words. Sudden onset, very sharp pain in the belly, vomiting of food and bile, abundant and foetid diarrhoea, and rise of temperature, form the **syndrome** characterizing the onset of pneumococcal peritonitis. This syndrome is not found in other peritoneal diseases, and must, therefore, be our guide at every stage of the malady. **As soon as the peritoneal fluid is appreciable**, we must employ an exploratory aspiration, and look for the pneumococcus.

Pneumococcal peritonitis is often **encysted**, the purulent collection occupying one large or several pockets. The surgeon must not forget this fact at the time of the operation. In one of Brun's cases the abdominal viscera were covered on their anterior surface with an extensive false membrane, thicker in the lateral than in the middle portions, and circumscribing a large suppurating cavity that extended upwards and to the left as far as the diaphragm, upwards and to the right as far as the anterior edge of the liver, and descended into the pelvis as far as the lowest part of Douglas's pouch. In Brault's case the peritonitis occupied the right lower half of the abdomen. In Sevestre's case the peritonitis was encysted below the umbilicus. In Gaillard's case the peritonitis was encysted, and Jalaguier had to open several pockets.

The peritoneal fluid is greenish, thick, creamy, and fibrino-purulent, and has all the characteristics of pneumococcal pus. In one case, however, it was not purulent, but sero-fibrinous (Weichselbaum's case), an exception also found in pneumococcal pleurisy which may not be purulent. Numerous flakes and thick membranes float in the fluid.

**Very Grave Forms.**—Although pneumococcal peritonitis is not grave as a rule, it may be formidable, as in two cases reported by Brun:

A little girl awoke at six o'clock in the morning with pain in the abdomen, vomiting and diarrhoea. Next day the vomiting persisted, and the child was sent to the hospital. The general condition was bad: the facies was peritoneal, the eyes were hollowed, the pulse was 160, and the temperature 100° F. The belly was slightly ballooned, but tense and painful, especially on the right side. Immediate intervention was decided upon. The disease was of three days' duration, and Brun found in the peritoneum a small quantity of odourless, turbid fluid, and some false membranes over the intestinal coils. The appendix, which was apparently healthy, was resected. After the operation an injection of serum was given. The little patient passed a very restless night,



and died the next morning at five o'clock, on the fourth day of the disease, the clinical picture being that of primary streptococcal peritonitis.

At the autopsy hardly any fluid was found in the peritoneum, excepting at the posterior surface of the stomach and on a level with the spleen, where greenish pus was discovered. False membranes were scattered over part of the abdomen, the mesentery was packed with glands, and the right tube was congested. False membranes covered the right pleura, but were, however, less abundant than in the peritoneum. The lungs were healthy. The histological examination of the appendix proved its healthy condition.

The bacteriological examination of the pus was made by Zuber, and on the slides he found only encapsuled, lanceolated diplococci, which stained by Gram. The pus, sown on the surface of agar-tubes, after successive dilutions, produced colonies of the same diplococci. This pus, sown in the deeper parts of the agar-tubes for the purpose of demonstrating anaerobic microbes, gave colonies of this same diplococcus. The inoculation of the pus under the skin of the thigh in a mouse caused death in twenty-four hours, and the pneumococcus was found in a state of purity in blood from the heart. The little girl succumbed, then, to an attack of primary pneumococcal peritonitis.

A little girl of four years and a half was taken ill with sore throat, fever, dysphagia, and headache on April 9, and a whitish exudate was also noticed on the tonsils. On April 14 the sore throat was better, but she was then seized with pains in the abdomen and frequent vomiting. The belly was ballooned, and as the symptoms became worse, she was admitted into hospital on April 16. Her condition was very bad indeed: dyspnoea; pulse uncountable; the eyes were surrounded by black circles; the look was dull; and the belly was distended and painful, but there were no areas of dullness. Half an hour after admission into hospital the child vomited copiously, and as she was on the point of death, an injection of serum was administered. At ten o'clock Brun operated. On opening the peritoneum a small quantity of greenish odourless pus flowed forth. This small collection was not seated around the appendix. The intestinal coils were covered with thin false membranes. Immediately after the operation injections of serum, ether, and caffeine were administered. The patient died on the afternoon of the third day from **superacute** peritonitis. The clinical picture in this case was that of primary streptococcal infection of the peritoneum.

At the post-mortem examination a few spoonfuls of pus were found in the pelvis, and false membranes were adherent over the whole of the intestine. The appendix was normal, and all the viscera were healthy. Bacteriological examination of the pus removed during the operation was made by Bernheim, who found only the pneumococcus.

These two cases of **superacute** peritonitis differ in a singular manner from the ordinary pneumococcal peritonitis. Clinically, the early intensity of the symptoms, the rapidity of the infection, and the extreme gravity of the prognosis, bring them into close relation with streptococcal peritonitis. I see only one way of making a rapid diagnosis, and that is to employ sero-diagnosis at once by Bezançon and Griffon's method, as described under Pneumonia.

**Association with the Bacillus Coli.**—The pneumococcus is the only microbe in pneumococcal peritonitis, but at the autopsy the *Bacillus coli* which has invaded the peritoneum post mortem is often found. In cases of **umbilical fistula** other microbes may also invade the peritoneal cavity (secondary infection).

I am not sure that the *Bacillus coli* may not be associated with the pneumococcus during life, as the following cases seem to show :

On March 16, 1898, in consultation with Blache, I saw a child of nine years of age who had suffered for the past few days from abdominal symptoms. Several children in the same family had been laid up with influenza, and our patient had had a slight attack. During the night of March 14 the child awoke with pains in the stomach. Next day the pains persisted, but had no definite localization. The child vomited, and the temperature rose to 102° F. On March 16 I saw the patient with Blache. In a child with sharp pain in the stomach, fever, and vomiting the first thought is of appendicitis. In this case local pain and muscular resistance were not present at McBurney's point. The pain was diffused over the lower abdomen. The subumbilical region was prominent, painful, and tympanitic, but not uniformly resonant. The pulse was quick, and the vomiting continued. The child had passed a bad night, and the facies was changed. We were face to face with peritoneal infection, but of what kind ? The diagnosis of appendicitis was eliminated, because the signs of appendicitis were absent. Still, the child was suffering from peritonitis, as shown by the sharp pain, vomiting, fever, distension of the abdomen, and subumbilical tympanites.

When a child is suddenly taken ill with abdominal pain, vomiting, tympanites, and fever, and when he has not appendicitis, we must think of primary pneumococcal peritonitis. The age of the child, the sudden onset, the fever, the intense pain, the vomiting, and the limitation of the peritonitis to the lower abdomen, were all in favour of pneumococcal peritonitis.

Diarrhœa alone was lacking in the clinical picture. As a matter of fact, diarrhœa is usually present from the onset. I said to Blache : " If the child had diarrhœa, there would not be any hesitation as to the diagnosis of pneumococcal peritonitis." Though this peritonitis is not dangerous in the early stage, the idea of surgical intervention came to my mind. We agreed to see the child on the following day. As soon as I arrived, I was told of the appearance of the expected symptom, diarrhœa. During the night the child passed about fifteen yellowish loose stools.

The malady was undoubtedly primary pneumococcal peritonitis. We had then to think of operation, which, however, was not urgent, since pneumococcal peritonitis is not as grave as appendicular and primary streptococcal peritonitis. The latter, even from the first, causes alarming symptoms : drawn look, acceleration of the pulse, delirium, and prostration, which rapidly end in collapse. Moreover, the operation must be performed early. As regards appendicular peritonitis, the danger does not come solely from the peritonitis : it comes from the toxi-infection, which demands early intervention.

Pneumococcal peritonitis is quite different. We have just seen that this kind of peritonitis runs a somewhat slow course, and that patients operated upon on the eighth, tenth, or fifteenth day recover. I therefore saw no harm in postponing the operation till the following day (sixth day of the disease), and I believe, too, that there is no case in which the operation for pneumococcal peritonitis has been performed before the tenth or twelfth day. The operation was therefore fixed for the next day. When I arrived I was quite surprised to note the change in the patient. The child had passed a bad night, with insomnia, restlessness, and much crying. The facies was peritoneal, the pulse was much accelerated, the pain and tympanites were general. The condition did not in the slightest way resemble the first phase of pneumococcal peritonitis. Had we made an error in diagnosis, and was this peritonitis not due to the pneumococcus ? A doubt arose in my mind. As the operation was urgent, Routier performed it that day. As soon as the peritoneal cavity was opened we noticed the tainted odour indicative of the *Bacillus coli*. A large quantity of sero-purulent, foetid fluid flowed out from the peritoneal cavity, together with thick greenish fluid and fibrinous membranes.



Routier asked himself whether the appendix was not the origin of this peritonitis. It was, however, found to be healthy.

At first sight the fibrinous membranes and the homogeneous pus gave the idea of pneumococcal peritonitis. On the other hand, the foetid sero-purulent fluid bore witness to peritonitis caused by *Bacillus coli*. Bacteriological examination showed the presence of both microbes—pneumococcus and *Bacillus coli*. There were no other varieties. The patient was very ill for some days, and then slowly recovered.

We have here, then, a case of peritonitis with mixed infection which occurred in two stages. During the first stage the peritonitis was pneumococcal, and was characterized by its sudden onset, pain and vomiting, subumbilical pain, and diarrhoea. The general condition was not bad, and to look at the child no one would have said that he was suffering from peritonitis. On the fifth day the scene changed. **The facies grew peritoneal, the general condition became alarming,** and the *Bacillus coli* entered upon the scene.

What do we actually know of **primary** peritonitis due to the coli bacillus? Nothing. I do not know of a single published case. Of all kinds of secondary peritonitis, peritonitis due to the *Bacillus coli* is the most frequent. It is the peritonitis caused by perforation of the intestine, and especially the peritonitis of appendicitis, whether there is perforation of the appendix or migration of the microbes through the unperforated walls of the appendix.

Such, however, was not the case here. How, then, was the poly-infection to be explained? I do not know. It may be said that, thanks to the pneumococcus, the *Bacillus coli* penetrated into the peritoneal cavity.

Charrin and Veillon have published a case of pure pneumococcal peritonitis which was invaded **after death** by the *Bacillus coli*, but this microbial association did not take place during life, as it did in our patient.

**Treatment.**—Pneumococcal peritonitis is often fatal if left to itself. The patient succumbs from cachexia or from general intoxication, with secondary troubles in the pleura, lungs, heart, etc., and death is sometimes very rapid (Brun's case). It is therefore right to intervene **at an early date**, and not to wait for secondary infections. Numerous recoveries have been obtained by opportune surgical intervention. The failures, on the contrary, must be placed to the debit of late operation. The patient operated on late succumbs to infection of the lungs, the pericardium, or the pleura. **As soon as peritonitis is recognized, we must operate.**

It may be asked if laparotomy is necessary in the case of spontaneous opening. Cases of cure are cited, but the spontaneous evacuation of the pus cannot, in my opinion, be relied upon. In this event two complications are to be feared—viz., the penetration of a new and formidable microbe, such as the streptococcus or the staphylococcus, into the focus, or fresh infection in one of the peritoneal fistulæ which have been incompletely emptied. Early surgical intervention is therefore the only rational treatment for pneumococcal peritonitis.

## V. PRIMARY STREPTOCOCCAL PERITONITIS.

The following case is taken from a clinical lecture on the subject at the Hôtel-Dieu :\*

On January 9, 1900, a young girl was taken ill with "sore throat, accompanied by fever and lassitude. She kept her bed for three days without sending for a doctor. On the 12th she fancied she was better, and got up, but remained in her room. On the 13th she drove out to see a friend, but became so ill that she had to be brought home. A doctor was called in on the 14th. He thought it was a case of influenza, and advised the patient to go into hospital. She passed a bad day on the 15th, and came into my ward the same evening. The temperature reached 105° F. During the night she vomited porraceous matter. The scene changed rapidly, and on the morning of the 16th, when I came to the hospital, I found her suffering from peritonitis. She was prostrated, and incapable of giving any information, with sunken eyes and peritoneal facies. The stools were liquid, and were passed unconsciously; the temperature was 103° F., and the pulse uncountable; the extremities were cyanosed and cold, and collapse was imminent.

The abdomen was slightly distended, not hard and retracted, as in peritonitis due to perforation of a gastric or duodenal ulcer. It was painful on exploration, as far as could be judged from the complaints of the patient. Arrest of fæces and flatus; blood-stained fluid flowed from the vulva.

I diagnosed superacute general peritonitis. The uterus and its adnexa seemed healthy. I eliminated the hypothesis of peritonitis due to appendicitis or to perforation of an ulcer of the stomach or duodenum, and I likewise put on one side the idea of pneumococcal peritonitis, because of the rapidity and severity of the symptoms. I ordered an injection of artificial serum, and requested Marion to perform laparotomy. The abdomen being opened, the great omentum was found to be adherent, and beneath it a layer of non-foetid, homogeneous, but not very thick, pus. The coils of the small intestine did not form masses covered with thick membranes, as in pneumococcal peritonitis. The appendix was healthy. In the pelvis pus was found around the right tube, but no salpingitis. The adnexa were healthy, the uterus was normal, and Douglas's pouch was blocked up by soft purulent adhesions.

During the evening the temperature fell to 98° F., the pulse was bad, and the patient became collapsed. She was very restless during the night, the temperature rose to 103° F., and she died next day.

At the post-mortem examination we found general peritonitis, but no lesion to explain this peritonitis. The stomach, uterus, tubes, and ovaries were healthy. The gall-bladder and the liver were normal. The kidneys, heart, pleura, and the lungs showed not the slightest lesion—in short, as there was not a lesion anywhere, primary peritonitis was the verdict.

Griffon made a bacteriological examination of the pus removed during the operation, and found only a streptococcus (long flexuous chains without capsules), but not a trace of the pneumococcus.

The pus was sown on agar broth and rabbit serum. The surface of the agar showed a growth of very fine colonies, less transparent than those of the pneumococcus. There were no other foreign colonies. The microscope showed that these colonies were formed of streptococci. In the broth culture the clots characteristic of the streptococcus were seen. In the rabbit's serum streptococci in long, flexuous chains, without capsules, and no pneumococci, were found. The absence of any other microbe during the examination made anaerobic cultures unnecessary.

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\* *Clinique Médicale de l'Hôtel-Dieu*, 1903, 11<sup>me</sup> leçon.



**Summary.**—The girl succumbed in a few days to peritonitis, the streptococcus being present alone, without other microbes. The intensity of the symptoms and the superacute course of the disease show how great the virulence of the infection must have been.

In this case, how had the pathogenic agents entered? Can we blame the angina from which the patient was suffering a few days before? As I did not see her before the onset of peritonitis, and did not verify the angina, I do not dare to express an opinion as to the pathogenesis of the peritoneal infection. This hypothesis, however, cannot be rejected.

I have collected a few cases of primary streptococcal peritonitis, which closely resemble my case. They will serve to show the details of this terrible infection.

The following case was published by Milian and Herrenschmidt :

On June 13, 1899, at 11 a.m., a young woman of nineteen years of age was brought to the Lariboisière Hospital, and placed under the charge of Landrieux. She had been ill for a few days. She was examined at 2 p.m. She was lying on her back, prostrated; the lips and cheeks were cyanosed, the skin was clammy, the extremities were cold, the pulse was uncountable, the tongue was parched, the respiration was quick, and the temperature was 97° F.

The patient passed her motions under her. The belly was hyperæsthetic and moderately tympanitic. No hiccough, no vomiting. She was collapsed, and could hardly answer questions. We found out, however, that she had been ill for about a week, and that the disease had commenced with a sore throat. As adynamia accompanied by diarrhoea, distension, and abdominal pain were present, we thought of typhoid fever with cardiac failure. The parents told us later of the existence of porraceous vomiting, with obstinate constipation. The patient died during the night.

At the post-mortem examination we found peritonitis and pus which was turbid and not homogeneous. A very thin layer of fibrin covered the intestinal coils. In spite of a minute search, it was impossible to discover any lesion to explain the peritonitis. All the organs were healthy. It was, therefore, a case of **primary** peritonitis. The bacteriological examination proved that the peritonitis was **streptococcal**. Had the angina been the entrance-gate of the microbe? It is possible.

The following case has been published by Meunier :

A young girl came under the care of Millard. Two evenings prior to her admission into hospital she had suddenly been seized with a sharp rigor, followed by headache, prostration, repeated vomiting, and severe diarrhoea. During the night she became delirious; the restlessness was extreme, and the menses, which had appeared two days previously, stopped suddenly. Next day the vomiting, diarrhoea, and abdominal pain continued. On the third day the symptoms became worse, and she was admitted to hospital. The patient was so weak that it was impossible to take her history or locate the nature of the infection. Her lips were covered with sordes, her face was drawn, and her speech was short and halting. The pulse was 156 and the temperature 102° F. The abdomen was slightly distended and tender on pressure in the left iliac fossa. The motions were liquid and passed under her. Nothing found on auscultation of the heart and the lungs. The condition did not alter through the night, and on her admission (fourth day of the disease) she was completely prostrated, with cold sweats and cyanosis of the face. She died on the fifth day.

The autopsy revealed acute sero-purulent peritonitis, with false membranes; no adhesions, and no matting of the coils. The pelvis contained some ounces of turbid but non-fœtid fluid. The case was, therefore, apparently one of primary peritonitis.

Bacteriological examination of the peritoneal fluid taken away one hour after death showed, after staining with gentian violet, innumerable little chains of streptococci, which did not stain with Gram. Agar cultures gave a rich growth, having all the characteristics of colonies of streptococci. The peritonitis was therefore primary, and due to the streptococcus.

The epithet "primary" is, perhaps, not strictly applicable to this case, for, as Meunier says, the cause and the origin of the peritoneal infection may be as follows: The patient was a servant, and was tending a lady suffering from severe facial erysipelas, with high fever, invasion of the face and of the scalp, suppuration, and eye complications, which called for surgical intervention. The servant, whose menses had just appeared, had, to keep herself clean, used a towel upon which she had wiped her hands after dressing her mistress. While out for a walk she was suddenly taken ill with a violent rigor, headache, and vomiting. She was brought home and put to bed. Fever, diarrhoea, restlessness, and delirium appeared, and the menses suddenly stopped. In this case the virulent streptococcus of erysipelas may have caused the peritonitis, being conveyed by the contaminated towel used as a napkin by the servant.

This pathogenesis is quite acceptable, because we know that, on the one hand, the streptococcus can cause erysipelas and abscess (Widal), and, on the other hand, we know that immediate contagion through the uterine mucous membrane may be brought about by virtue of the menstrual loss of tissue (Dolérís). Furthermore, this case of streptococcal infection following erysipelas may be reconciled with the views put forward by Trousseau,\* who wrote an admirable chapter on the relations between the puerperal condition, erysipelas, and peritonitis. He noticed that a new-born child suffering from facial erysipelas might die of peritonitis.

It is interesting to compare with this case two of Tarnier's cases, which also prove that the menstrual loss of tissue (the uterus being perfectly healthy) may be the entrance-gate of peritoneal infection or of septicæmia. Tarnier reports in his thesis, that during an epidemic of puerperal fever two pupil midwives who were menstruating were attacked by septicæmia, with rigors, fever, and nervous symptoms. One of them died of peritonitis, and the diagnosis was verified at the post-mortem.

I have still to quote two other cases of primary peritonitis. The first is a case of Cornil. On January 26, 1901, a woman fifty-six years of age was admitted into the Hôtel-Dieu at ten o'clock at night. She walked doubled up, and was suffering from her stomach. The pain had been present for the past three days. In the surgical wards, where she was first admitted, appendicitis or ileus was at first thought of. The patient had a drawn look and a thready pulse. The extremities were covered with a cold sweat, and the temperature stood at 99° F. The stomach was distended and very painful on palpation, but without any appreciable effusion. The pain was not limited to McBurney's point. There was no lesion in the genital organs. The evening before, the patient had had nausea, vomiting, and two liquid stools. The diagnosis of peritonitis was made, but it was not possible to determine the cause. The patient was so weak that an operation was out of the question. On the following day at 6 a.m. Celos was called to the patient, who had just had a copious hæmatemesis. The bedclothes and the floor were soiled with a blackish fluid like coffee-grounds. Death supervened a few moments afterwards.

At the autopsy we found acute peritonitis, with diffuse redness of the parietal peritoneum; the coils of the small intestine were congested, and covered in places with thin fibrinous membranes infiltrated with pus. There was no gas, and the fluid was in an unappreciable quantity. The appendix was normal, and the genital organs were healthy. The stomach contained no blood, but a few small ecchymoses were present on the mucous membrane. The bacteriological examination revealed the presence of

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\* Trousseau, *Clinique Médicale de l'Hôtel-Dieu*, t. i., p. 187.



streptococci in small chains. A mouse inoculated with the culture-broth died in twelve hours, and its blood contained diplococci without capsules. "In this case we therefore had to do with **primary streptococcal purulent peritonitis**, or at least with infectious peritonitis accompanied by copious hæmatemesis, without being able to discover the cause."

I will close this account with Leyden's case, which occurred in 1884, and is the first case of primary streptococcal peritonitis verified by bacteriology: On January 28 a young woman was taken ill with rigors, repeated vomiting of green fluid, and sharp pains in different parts of the abdomen. The patient could give **no** cause for her condition. Three days later, on January 31, she was taken to hospital. The fever was not high; the greenish vomit persisted; the prostration was great; the voice was feeble; the breathing was shallow and quick; the abdomen was distended, and the pains were sharp, particularly in the hypochondria; the stools were loose, and the urine was albuminous; the temperature was 101° F., and the pulse 116. During the night of January 31 the vomiting recurred several times, and was copious.

On February 1 the patient's eyes were hollow and the pulse was small; the stomach was very painful. The ingestion of the smallest quantity of fluid was followed by vomiting. The respiration was 44 and the pulse 144. The following night the patient was delirious. In the morning the collapse was complete: the pulse was hardly perceptible, the extremities were cold, and the facies was hippocratic. Death took place at seven o'clock in the evening. The diagnosis had been: Diffuse peritonitis. The persistence of the diarrhoea, however, had given rise to the idea of typhoid fever, with perforation of the intestine.

The autopsy proved that it was not a case of typhoid fever, but of general peritonitis. The peritoneal fluid was abundant, purulent, odourless, and contained some fibrinous flakes. The intestines and the omentum were covered with fibrino-purulent deposits. The stomach, intestine, and appendix showed no lesions. All the organs were healthy. The peritonitis was held to be primary. Bacteriological examination revealed the presence of streptococci in straight and curved chains.

All these cases were streptococcal peritonitis of very rapid course and fatal prognosis. I have reserved a special place for the exceptional case of Lorrain, which differs from the others in the relative benignity, the slow course of the disease, and the success which followed a double operation.

The case is as follows:

On November 21, 1896, a little girl of eight years of age was admitted into D'Heilly's ward. She had had no previous illness. Her mother had died in childbed a fortnight previously. The little girl had been ill for three weeks. No precise information was available. It was known that she was suffering from her stomach, and a doctor who saw her several times diagnosed typhoid fever. The child was always complaining of her stomach. She was thin, her skin was dry, her nose was slightly pinched, and her eyes were hollow. The stomach was distended and hard. Pressure caused pain and muscular resistance. Neither tumour nor induration could be felt. The right iliac fossa was free. The spleen was not enlarged, and no lenticular rose-spots were found. The patient had alternate diarrhoea and constipation, with some vomiting. On auscultation the heart and the lungs were normal. The urine was albuminous; the temperature oscillated between 99° and 101° F. The general condition was bad. During the next few days there was no change. The vomiting ceased, but the diarrhoea persisted. The stomach was still distended and painful on pressure. The symptoms of loss of flesh, fever at night, diarrhoea, and ballooning of the stomach led to the idea of enteritis, with probable extension to the peritoneum.

On December 2 a hard mass, which increased rapidly in size and reached the surface, was found in the left iliac fossa. On December 6 the tumour was clearly localized in the left iliac fossa, but was not painful. On December 7 an operation was performed by Brun.

An encysted collection of pus was found. The pus was yellowish, odourless, and homogeneous. No false membranes were seen. The quantity of pus evacuated was estimated at 2 pints. During the next few days the temperature fluctuated greatly—103° F. in the evening, and 99° F. in the morning. On January 1 an induration was felt in the right iliac fossa, and a second operation was therefore performed. The appendix was found to be healthy, but the cæcum was adherent to the small intestine. The child was finally discharged cured.

The case appeared to be one of pneumococcal peritonitis. The encysted abscess, the homogeneous pus, the slowness of the peritoneal infection, the relative benignity of the peritonitis, and the recovery in no way correspond to streptococcal peritonitis. Bacteriology, however, proved that it was a case of streptococcal peritonitis. Direct examination of the pus on slides showed the presence of diplococci in chains, staining with Gram, and the absence of other microbes. The pus, sown on broth and agar, gave pure cultures of streptococci at the end of twenty-four hours. Inoculation of the cultures in a rabbit caused streptococcal infection. In this case, therefore, we must admit as an exception primary streptococcal peritonitis of a benign nature.

**Description.**—Let us now study **primary streptococcal peritonitis** as a whole. It is far less common than primary pneumococcal peritonitis. Furthermore, streptococcal peritonitis deserves the name of primary much less than pneumococcal peritonitis. If the reader will refer to the section on Primary Pneumococcal Peritonitis, he will see that in almost every case it is impossible to find the entrance-gate of the pneumococcus. The disease breaks out in the midst of good health, and we are unable to find the small lesion which has opened the door to the infection.

It is not always the same in so-called primary streptococcal peritonitis. It is true that in many cases the term “primary” is perfectly applicable in the sense that the peritonitis occurs in a healthy subject, and that the most minute examination may not reveal the starting-point of the infection. In the cases of Cornil and Leyden the name “primary” holds good. In my case, as well as in that of Milian and Herrenschildt, the peritonitis was likewise primary. The tonsil may, perhaps, be the origin of the infection, but this is pure hypothesis. In Meunier’s case the peritonitis was primary in the sense that it was not possible to find the causative lesion, but it is likely that the streptococcus of erysipelas was carried by the contaminated towel. In Lorrain’s case the peritonitis may be considered primary, because it was not caused by any lesion. The child, however, lived in the midst of surroundings infected by her mother, who died later in childbed, and here, too, it is permissible to think of contagion.

Primary streptococcal peritonitis, like primary pneumococcal peritonitis, is chiefly found in the female sex; but the former has a predilection for adults, whereas the latter is much more frequent in children. The peritoneal lesions vary in the two cases. Pneumococcal peritonitis tends to form encysted pockets, and is rarely general. It gives rise to thick, fibrinous membranes, which cause adhesions of the intestinal coils and of the organs.



The pus is greenish, thick, homogeneous, and laudable. Streptococcal peritonitis, on the contrary, has no tendency to become encysted, but is prone to become general. It gives rise to thin membranes, which cause but slight adhesions. The pus is neither thick nor homogeneous, but a sero-purulent liquid, comparable to tainted broth. The onset of both varieties may be sudden and violent. In each case the patient is attacked in the midst of good health. Rigors, fever, vomiting, and abdominal pain are the first symptoms. However, the "abdominal stitch" seems to me to be more marked in pneumococcia, and, on the other hand, the greenish vomit appears to be more frequent in streptococcia. Hiccough is rare in both cases. I would call special attention to the diarrhœa. It is too often thought that constipation is the usual symptom in peritonitis. Constipation is particularly frequent in appendicular peritonitis and in peritonitis following perforation of the stomach or duodenum, but in pneumococcal and streptococcal peritonitis diarrhœa is the rule. In pneumococcal peritonitis, diarrhœa appears very early in the disease, and for several days the stools are liquid, frequent, and bilious. In streptococcal peritonitis diarrhœa is found in almost all cases, and has led to the erroneous diagnosis of typhoid fever.

**Summary.**—In pneumococcal and streptococcal peritonitis the onset is sudden, and certain symptoms, such as pain, vomiting, and diarrhœa, are common to both diseases. The real distinction is the general condition of the patient, which differs from the first in the two cases. In streptococcal peritonitis the prostration, the acceleration and smallness of the pulse, the delirium and the ataxo-adyndamia, and the rapid tendency to collapse and coma, are the chief features, even early in the case. With few exceptions, nothing of the sort occurs in pneumococcal peritonitis, which may last for weeks without fatal consequences.

The prognosis in streptococcal peritonitis is more serious than in pneumococcal peritonitis. In the latter case the toxi-infection remains for a long time abdominal, and does not affect the remainder of the system, whereas in the case of streptococcal peritonitis the toxi-infection is severe from the outset. It is the rapid poisoning which dominates everything, and in a few days leads to collapse, coma, and death.

This parallel between pneumococcal and streptococcal peritonitis is applicable in the great majority of cases. There are, however, exceptions. On the one hand, streptococcal peritonitis may be neither very virulent nor superacute; it may run a slow course, and be cured after operation, as in the case reported by Lorrain. On the other hand, pneumococcal peritonitis, from its great virulence or the presence of other organisms, may be as severe as streptococcal peritonitis. Cases of this kind are described under Pneumococcal Peritonitis.

The **treatment** of primary streptococcal peritonitis is early surgical intervention. The operation **must be performed as quickly as possible**, and in this event a cure may be hoped for.

## VI. CANCER OF THE PERITONEUM—CANCEROUS PERITONITIS.

**Ætiology.**—The great omentum is the only peritoneal fold attacked by primary cancer. Cancer of the peritoneum is always secondary, and has the same structure as the primary growth (carcinoma, epithelioma, sarcoma, lymphadenoma). This question has been carefully discussed in Aslanian's thesis, from which I have taken the following statistics :

Cancer of the stomach invades the peritoneum in the proportion of 1 to 4 ; cancer of the intestine invades the peritoneum in the proportion of 1 to 8 ; primary cancer of the liver invades the peritoneum in the proportion of 1 to 8 ; primary cancer of the biliary passages invades the peritoneum in the proportion of 1 to 2 ; cancer of the pancreas invades the peritoneum in the proportion of 1 to 3 ; cancer of the spleen invades the peritoneum in the proportion of 1 to 13 ; cancer of the kidney invades the peritoneum in the proportion of 1 to 15 ; cancer of the uterus invades the peritoneum in the proportion of 1 to 18 ; carcinoma and sarcoma are rare in the ovaries, but epithelioma of a papillary type is common, and invades the peritoneum in the proportion of 1 to 3 ; cancers of the thoracic cavity practically never spread to the peritoneum, but cancer of the peritoneum often spreads to the thoracic organs, glands, pleuræ, and lungs.

**Pathological Anatomy.**—The cancerous process takes place in two stages : it commences as a local infection near the primary growth, and then becomes general. The peritoneal cavity can only be infected when the serous membrane around the cancerous organ is involved. The seats of election of the peritoneal infection are the anterior parietal peritoneum, the diaphragmatic peritoneum, the pelvic peritoneum, and the large omentum.

In the **miliary** form cancer takes the form of tubercles as large as a pea, a lentil, or a nut. In the **ulcerous** form the tumours present crateriform ulcerations. In the **vegetating** form, which usually follows papillary growths of the ovaries, the cancer resembles a soft mushroom, bristling with papillæ that may be as large as a walnut, or larger. In the **cystic** form, which is usually secondary to cancer of the ovaries, but which may also occur primarily in the great omentum, the cancer takes the form of cysts, from the size of a pea to that of an apple. In the **infiltrating** form, which is seen especially in the parietal peritoneum and omentum, the cancer takes the form of hard and thick patches, or of infiltrated sheets. All these forms may be combined. Carcinoma of the peritoneum is accompanied by more or less intense inflammation of the serous membrane. Sometimes we find only a fibrinous exudate, with matting of the intestinal coils and yellowish fluid ; but at other times the cancerous peritonitis is clearly pronounced, and there are numerous adhesions between the layers of the peritoneum



and the abdominal organs. These neo-membranes are vascular, sometimes hæmorrhagic, and often invaded by the cancerous growth.

These cancerous masses, fused with the organs and with the glands, and themselves attacked by cancerous degeneration, sometimes form enormous tumours.

The ascitic fluid in cancerous peritonitis may amount to several pints. The fluid is sometimes citron-coloured and sero-fibrinous, or sometimes hæmorrhagic. The fluid is nearly always hæmorrhagic in papillomatous cancer (Terrier). The sero-purulent effusion is much less common, and an ascites which is citron-coloured at the first puncture may, like pleuritic fluid, later become hæmorrhagic, or *vice versa*.

The hæmorrhagic fluid may be rosy, reddish, brownish, or blackish. Ascites, the liquid being citron-coloured or hæmorrhagic, is a much more frequent complication of abdominal neoplasms than the invasion of the peritoneum by cancer. "In ovarian cysts Terrier found twenty-nine cases of ascites, and only six cases of peritoneal metastasis, in 175 ovariectomies."

Hanot and Gilbert, in their researches on diseases of the liver, found that in thirteen cases of cancer of the liver, ascites was present in ten cases, whilst general infection of the peritoneum existed in only three cases. "Whatever organ be invaded by the cancer, we have always found that ascites predominated over peritoneal metastasis" (Aslanian). Ascites is not only more frequent than cancerous metastasis in the peritoneum, but it is also the earliest phenomenon, and seems to precede the appearance of cancer in the peritoneum.

**Description.**—Cancerous peritonitis has an insidious onset, and is not ushered in by any acute symptom. The abdomen becomes distended, ascites appears, the patient complains of pain, and it may be said that the symptoms of peritonitis are subinherent to the symptoms of cancerous infection.

A little later, in the stationary stage, these symptoms are more clearly marked: the abdomen is more enlarged, the ascites is greater, and a collateral circulation often appears in the abdominal wall. Palpation is rarely painful, but it allows us to make out indurations and tumours of various forms and dimensions, especially after evacuation of the fluid. The ascitic fluid is generally free in the peritoneal cavity, and a fluid thrill is readily obtained. The inguinal glands may be attacked by the cancerous degeneration, which may also be met with in other regions (supraclavicular glands). Numerous complications may appear from one moment to another (intestinal occlusion, jaundice from compression, retention of urine). In the thorax we may find malignant growths in the pleura, lung, or mediastinum.

Intraperitoneal hæmorrhage is so frequent that it may be looked upon as a symptom rather than as a complication. Fever is far from being

constant, but we sometimes see the temperature rise to 102° or 103° F. after a period of complete apyrexia.

The patient quickly becomes cachectic; the loss of flesh and asthenia make rapid progress. The yellow, straw-coloured complexion, with peripheral œdema, appears, and death supervenes within a year of the invasion of the peritoneum.

## VII. HYDATID CYSTS OF THE PERITONEUM.

**Pathogenesis.**—Hydatid cysts of the peritoneum are fairly common. It is thought that the embryo reaches the peritoneum, either directly by perforating the walls of the intestine, or indirectly through the bloodvessels and lymphatics. Peritoneal cysts are sometimes secondary to a cyst in another organ. In some cases the disease may be grafted on the peritoneum by the heads of the *tæniæ*, liberated in consequence of natural or artificial rupture of a cyst (Soupault). We have then to deal with a case of secondary echinococchia.

**Pathological Anatomy.**—The hydatid cyst of the peritoneum is rarely solitary; we usually find from three to fifty or sixty cysts, and, indeed, two hundred and more have been counted. Their size varies greatly. The more numerous they are, the smaller their size, but they are never all of the same size. One or two cysts of the size of an orange are sometimes met with, and also other cysts as large as a lentil, a grape-pip, or a walnut.

The hydatid is found in the omenta, then in the mesentery, the mesocolon, the peritoneal folds of the pelvis, and the broad ligaments. The cysts may be discrete or confluent, and are sometimes aligned in chains, after the fashion of streptococci, or at other times agglomerated in bundles, like the staphylococcus, or, lastly, heaped up like a bunch of grapes. Davaine and Charcot have proved that the cyst **never** develops in the peritoneal cavity, but in the subserous cellular tissue. The peritoneum covers the cyst, and helps to form the pedicle when the cyst spreads from its point of origin into the pelvis.

The structure of the cyst is given under Hydatid Cysts of the Liver, but the peritoneal cyst is surrounded by a more distinct membrane, formed by the subperitoneal tissue, and covered by the serous membrane. This covering may become thick and fibrous, thus helping to isolate and fix the cyst. The hydatid cyst may give rise to more or less thickened false membranes, with matting of the intestinal coils and fibrinous bands. It is not rare to find ascites, due to compression of the portal vein.

**Description.**—Hydatid cysts of the peritoneum develop silently for some time. Enlargement of the belly is generally the first sign which betrays their presence. The belly enlarges and becomes deformed, sometimes in



one region, sometimes in another. This increase in size is soon followed by continuous or paroxysmal pain. Whatever the cause of the pain (torsion of the omenta, peritoneal traction, attacks of peritonitis), it may supervene in crises, accompanied by vomiting and constipation.

As the cyst grows, it provokes many and varied symptoms of compression. Compression of the intestine causes constipation and occlusion; compression of the portal vein gives rise to ascites and collateral circulation; compression of a large bile-duct leads to icterus; compression of the inferior vena cava is the cause of œdema of the lower half of the body; compression of the ureters brings about anuria and uræmia; compression of the nerves of the sacral plexus produces intense neuralgia. The abdomen, on examination, is frequently found to be deformed and nodular; percussion will map out dull and resonant regions. Very large cysts may give a sensation of fluctuation.

When peritoneal cysts grow towards the diaphragm, they may push it upwards into the thoracic cavity, and provoke symptoms of dyspnœa. **Pregnancy** in women suffering from hydatid cysts of the peritoneum has a marked effect on the prognosis. Porak has given a résumé of twenty-four cases, with the following results: Twice the accouchement was premature; five times the delivery was spontaneous, but difficult; and eight times the cyst had to be punctured during labour. The cyst broke in three cases. Embryotomy had to be performed in two cases, and Cæsarean section in two cases. Cephalotripsy was performed in one case, and in two cases delivery was impossible. The growth of hydatids of the peritoneum is generally very slow, and during the first period of their development they occasion no appreciable trouble. The symptoms of compression appear later, and it is only after several years that the patient reaches the cachectic stage, with loss of appetite, progressive wasting, loss of strength, and peripheral œdema.

The appearance of fever and rigors indicates suppuration in one or more cysts. Sweating and diarrhœa complete the picture of hectic fever, and the condition brings about marasmus and death.

The rupture of a hydatid cyst in the peritoneum is often accompanied by **urticaria** and symptoms of peritonitis, which is not always fatal. Hydatid disease of the peritoneum is serious because the cysts are almost always **multiple**, and, supposing that one of them recover, either spontaneously or after surgical intervention, the other cysts continue to grow, and sooner or later bring on the complications just mentioned.

**Diagnosis.**—The diagnosis is beset with extreme difficulties. The indurations and tumours which the hydatid forms resemble those of tubercular or cancerous peritonitis. Sometimes the cysts give the sensation of soft, fluctuating tumours, and at other times they give the impression of

indurated tumours adherent to the neighbouring organs. "It is easily understood that the hydatid tumour may be mistaken for a distended bladder, a gravid uterus, fibro-cystic fibroids, peri-uterine collections, cystic tubes, or cysts of the ovary" (Soupault). Aspiratory puncture is the only means of arriving at a diagnosis.

**Treatment.**—The treatment does not give such good results as are obtained in cysts of the liver, because the cysts in the peritoneum are multiple, of different ages, and develop one after the other; and even if, by an aspiratory puncture or by laparotomy, we succeed in curing one or two of the principal cysts, it is much to be feared that the other cysts will continue to grow. *Uno avulso, non deficit alter.*

### VIII. ASCITES.

**Ætiology.**—**Ascites** is dropsy of the peritoneum. It follows on mechanical and constitutional troubles. Ascites is found at all ages, even in young children. The mechanical troubles are those which offer an obstacle to the circulation in the portal vessels, and include atrophic cirrhosis of the liver, pylephlebitis, compression of the portal vein by tumours of the peritoneum, liver, spleen, pancreas, and mesentery. The constitutional troubles are those seen in Bright's disease and cachexia. Fluid containing sugar may occur in diabetics.

**Pathological Anatomy.**—The effusion may amount to as much as 60 pints. It has all the characteristics of dropsical fluid, being transparent, fluid, of a pale yellow colour, and contains a small quantity of albumin.

**Description.**—I have described the signs and symptoms of ascites under Laënnec's Atrophic Cirrhosis and Tubercular Peritonitis. I will therefore refer the reader to these diseases, and will merely say that the **course** and the **duration** of ascites are subordinate to its cause. In some cases (tuberculosis, hypertrophic alcoholic cirrhosis) ascites may recover without surgical intervention, but most frequently it is persistent, and the liquid is subject to variations which rarely end in recovery. The diagnosis of ascites comprises that of the symptom and of the cause. Ascites must not be confounded with **ovarian cyst, distended bladder, or chronic peritonitis**. The diagnosis of the cause is sometimes only possible after evacuation of the liquid. The treatment of ascites has been given under Atrophic Cirrhosis. **Cyto-diagnosis** is of value, although the results are not as exact in ascites as in pleurisy.



## IX. CHYLOUS, CHYLIFORM, LACTESCENT ASCITES.

**History.**—In 1700 we find the first mention of lactescent ascites. Vernage made a communication to the Académie Royale des Sciences concerning a peculiar case of dropsy. The lacteals had just been discovered, and people at once considered the effusion of chyle into the peritoneum as due to rupture of a lacteal. Subsequently, several cases were published. They were neither collected nor compared, and no deduction was drawn concerning their pathogenesis. In 1874 Guéneau de Mussy, referring to two cases of pleural effusion analogous to fatty emulsion, attributed the aspect of the fluid to fatty changes in the leucocytes and the fibrinous exudate. Veil and Letulle were of a similar opinion. In 1880 Debove described the existence of a special variety of effusion composed of emulsified fats, and quite distinct from serous, sero-fibrinous, and purulent effusions. This theory was supported in the thesis of Madame Perrée. In 1896 Strauss described a typical case of chylous ascites due to communication between the lacteals and the peritoneal cavity. Lion described a new variety of ascites, which had only the milky look, and differed in every other respect from chyle. The colour was due to the presence of a special albumin closely allied to casein. Other cases support this interpretation (Achard, Merklen, Sainton, Apert).

Widal and Prosper Merklen have shown that the morphology of the leucocytes in the effusion is of great importance in the diagnosis of lactescent ascites. My clinical assistant, Kahn, has published two cases of chyliform ascites in my wards at the Hôtel-Dieu. In these two cases we had to deal with secondary cancer of the peritoneum and of the primary lymphatic passages, and in one of them he proved the presence of the mono-nuclear leucocytes pointed out by Widal.

**Examination of the Lactescent Fluid.**—The quantity of fluid is not as great as in common ascites. It varies from 2 to 20 pints (Schmidt), and in one case only did it amount to 30 pints. The liquid has a tendency to reform. As its name indicates, it has the appearance of chyle. It is bluish-white (Quinke), milky, or creamy-white, like an emulsion of almonds (Marshall Hughes). Its density is lower than that of blood-serum, which is 1028. Hirtz and Merklen give the following figures: 1007, 1023, 1020, 1013. Nearly all authors agree that the lactescent fluid is quite homogeneous, and this feature persists sometimes for a fortnight (Kahn). One of its most remarkable characteristics is its great power of resisting putrefaction. Samples have been kept for some weeks in summer without giving off the least smell. In the long run the fluid separates into two layers—the lower one serous and almost limpid, the upper one whitish and creamy. The reaction is neutral or slightly alkaline.

On chemical examination, the proportion of fatty matter is large (15 parts per 1,000), though it may be present in much smaller quantities. It is, as a rule, easily soluble in ether. Albuminous matter also enters largely into the composition of the fluid (36 parts per 1,000). The weight of the mineral salts attains the average of 5 parts per 1,000. They are largely composed of chlorides, of phosphates, and of a small proportion of sulphates. In very rare cases the presence of cholesterine has been noticed. Sugar is often found. Bargebuhr considers the presence of sugar a distinctive sign between true chylous and chyliform ascites, but his sign has lost its value, since Richard, Reichel, and Rotman have proved that all pathological fluids poured out either in serous cavities or in the subcutaneous cellular tissue, with the exception of pus, contain sugar in variable proportion.

On microscopic examination, the effusion is composed of a large number of very fine rounded granules, isolated from one another, refractile, and showing Brownian movements. They are generally very small, but at times oily drops are visible. They dissolve in ether, and stain brown with osmic acid. It is not uncommon to find degenerated leucocytes. Widal and Merklen have shown the importance of the leucocytic formula of the effusion as regards the origin of lactescent ascites. The recognition of

the mononuclear leucocytes, to the exclusion of every other variety of white corpuscles, proves the lymphatic nature of the ascites. The white mononuclear corpuscles are the only ones transported by the lymph. Red corpuscles are sometimes seen.

In two cases the patient's serum was found to be lactescent. During the past few years some cases have been published in France under the name of "non-chylous milky ascites." The first is due to Lion, who has shown that ascites may be lactescent without containing any of the elements of chyle. In the case in question the effusion appeared in a woman suffering from cancer of the ovary. The fluid, analyzed by Winter, did not contain fat, but an albuminoid substance analogous to casein, which might be classed amongst the glyco-proteids of Hammerstein. Under the name of "non-chylous milky ascites," Achard has reported a case in which the fluid contained only an infinitely small quantity of fat. The microscope showed innumerable fine, refractile granules, unstained with osmic acid, and similar to those which Widal and Siccard have described in the lactescent serum of patients suffering from albuminuria. Similar granulations have been found by Sainton in the liquid of a non-chylous milky ascites in a patient suffering from cancer of the pylorus. Finally, a fourth case, entitled "non-chylous milky ascites," has been published by Apert. In this case the milky ascites supervened in a cardiac patient, and was characterized by the complete absence of fat globules, and by the presence of a fine powder visible only with very high magnification.

**Pathological Anatomy.**—Lactescent ascites does not properly belong to any abdominal lesion. It may be observed in almost all the affections which cause ascites, with or without lesions of the peritoneum. It has been noticed in alcoholic atrophic cirrhosis, without peritonitis (Depoix, Merklen, and Widal). In another case Remond and Rispal found post mortem only chronic nephritis. The lesions of a concomitant affection and lesions of the lymphatic system, such as rupture of the lymphatic ducts, compression by tumours or by glands, are generally associated. Tuberculosis and cancer of the peritoneum are the most frequent causes of lactescent ascites. In tuberculosis injection and pigmentation of the serous membrane are seen, and near Bauhin's valve a very fine injection of the chyloferous vessels, without appendicular lesions, is often noticed. The peritoneum and the intestinal coils adhere at the level of the two layers of the mesentery. The lesions of the lymphatic system include constriction and obstruction of the lacteal vessels, which are twisted and distended, but rarely ruptured; presence of a clot in the thoracic duct; and fullness of the intestinal villi. Adenitis is constant.

In the case of cancer we nearly always find in one of the abdominal organs a primary growth which has spread to the lymphatic passages. I had in my wards a woman suffering from lactescent ascites. The primary cause was a small cancer of the ovary. Kahn and Nattan-Larrier made the inspection, and the results of the autopsy were as follows :

The entire mesentery was much thickened, and on section was found to be crammed with glands of the size of a hazel-nut. These glands were hard, and yielded but little juice on scraping. They were cancerous. At the junction of the small intestine with the mesentery the lacteals were involved, and showed three principal types : (1) The



lymphatic duct was, as it were, injected, as large as the lash of a whip, indented, irregular, and tortuous. It was cancerous, though not corresponding to any cancerous lesion of the mucous membrane. (2) There was distension, but no cancerous change in the lymphatics. They were distended, as large as a violin-string, and stood out in white against the surface of the serous membrane. They showed symmetrical constrictions, clearly marking the situation of the valves. (3) The above-mentioned appearances were combined. Sometimes a cancerous lymphangitis was continued to the surface of the intestine by a large lymphatic vessel distended with lymph. Sometimes the lymphatics formed long and fine arches, interrupted at certain points by a short portion invaded by the growth. At certain points there were diffuse yellowish plaques like a thickening of the serous membrane, but due to general dilatation of the fine lymphatic capillaries. The lymphatic trunks, which ended in the receptaculum, though not invaded by the cancer, were nevertheless distended. The receptaculum was greatly distended by the neoplasm, and appeared as a nodular canal of the size of a penholder. This invasion extended for a distance of about 2 inches. No rupture was visible.

In certain cases a tear of the thoracic duct or of the chyloferous vessels places the peritoneal cavity in communication with the lymphatic circulation. Bussy's conclusions must certainly not be accepted without reserve, when he says that, in twenty-eight cases reported by him, twenty-seven may be attributed to the rupture of a chyloferous vessel. It is correct to make allowance for tears produced at the post-mortem examination, but there are indisputable cases of this lesion, as in Whitla's case :

The thoracic canal, in its inferior third, was transformed into a fibrous obliterated cord. Above it was much dilated, and below there was a perforation of the size of a pea at a point where the walls had gradually become thin.

Strauss says : " At first sight I did not perceive any chyloferous vessels standing out in relief on the surface of the mesentery, but after a careful examination I saw at two different spots a whitish liquid, flowing from two little openings on the anterior layer of the mesentery. The fluid was exactly like milk or chyle, and of a more milky colour than the ascitic fluid. These openings were ruptures of the lacteals into the peritoneum. The point of a pipette was introduced several times into these small openings, and about a cubic centimetre of chyle thus aspirated. I wish to state that this verification was made without touching the intestine, so as to avoid disturbing the coils and producing tears in the mesentery. It is certain that these two small orifices were not caused at the autopsy." Quinke has observed a similar case. It is in consequence of these cases that one of the numerous pathogenic theories which we shall now study has been built up.

**Pathogenesis.**—1. The effusion is due to the passage of the chyle into the peritoneal cavity, either by a tear or by an obstacle in the lymphatic system. This view of the question is based on facts of incontestable value. Strauss made use of a most ingenious contrivance to prove the passage of the chyle into the peritoneum during life. The patient, who was placed on an ordinary diet, was tapped, and the ascitic liquid analyzed. The patient

was then placed on strict milk diet, and an emulsion of butter was given in the milk as strong as the patient could take. Fresh effusion appeared at the end of a few days. The patient was tapped, and the analysis of the liquid revealed more fatty matter than at the first tapping. In this fatty matter there was also a much greater quantity of butyrine. It is therefore beyond doubt that the fat absorbed by the patient passed into the chyli-ferous vessels, and from them into the cavity of the peritoneum.

Apart from the cases in which rupture of the lymphatic vessels has been proved, can the transudation of the chyle through the chyli-ferous vessels be a cause of lactescent ascites? Rokitansky, Whitla, Verdelli, Bargebuhr, and Widal admit this; but an objection is raised against this theory, because experiments have never yet succeeded in reproducing chylous effusion. Potain, in speaking of this subject, insists on the numerous anastomoses of the large lymphatics.

2. Lactescent ascites is due to fatty change in a peritoneal effusion (purulent or fibrinous). Guéneau de Mussy admits that the chyliform effusion into the pleura results from the slow liquefaction of the leucocytes of the pus into large granulo-fatty masses, or from an exaggerated lipæmia. According to Robin, when the pus (which contains the elements of the chyliform liquid) cannot make its way out, it undergoes changes, amongst which fatty degeneration is one of the most frequent. The peritoneal serum is said to have an emulsifying action (Veil).

Letulle arrives at the following conclusions: (1) All cases of chyliform ascites examined post mortem are cases of tubercular, cancerous, or simply neo-membranous peritonitis; (2) the existence of chronic inflammatory lesions represents, then, one of the most constant elements in chyliform effusion; (3) granulo-fatty degeneration of the effused inflammatory products (fibrine and leucocytes) is perhaps sufficient to account for all the emulsified fat in suspension in the peritoneal serous fluid. This conception serves to explain the presence of fat in chronically inflamed serous membranes in which chyli-ferous vessels do not exist.

3. Debove opposes this theory with an argument of great value: How are we to explain the fact that the liquid preserves its chyliform character when it recurs so soon after tapping? In order to explain these facts, he proposes a theory which has the advantage of uniting all the cases without prejudging any one of them. These effusions, says he, are chyliform from the first, in the same manner as they might have been serous or purulent. The word "chyliform" merely shows that in appearance and colour the liquid somewhat resembles chyle.

4. Parasitic Theory. — In some cases of chyliform ascites Winckel observed very small entozoa, endowed with energetic movements, and said to be filaria, analogous to Bancroft's species. It is by their presence that



Lancereaux explains milky hydroceles, and Levois lymphuria. According to Lancereaux, the parasite causes the rupture of a chyliferous vessel.

Finally, the experiments of Desombry and Porcher, as well as those of Nocard, show that the intestinal microbes traverse the healthy intestinal wall, and pass into the serum when animals have been given fat to ingest. It may be that some new theory will withstand the attacks that have shaken all previous hypotheses (François).

**Symptoms.**—According to the teaching of pathological anatomy, lactescent ascites does not properly belong to any abdominal affection; it may be associated with any disease causing effusion. It has no proper symptomatology. Tapping and examination of the liquid alone reveal its presence. In the stationary stage, inspection, palpation, and percussion give the physical signs of ordinary ascites. Lymphatic varices have not been found in any case.

After tapping, cancerous or tubercular tumours may be found in the peritoneum. Ascites which is lactescent on a first tapping remains so on other tapplings. Nevertheless, it has been found first limpid, and then milky (Méry-Letulle); milky and then limpid (Siredey); or limpid, then milky, and limpid again (Rendu). Quenette has seen it associated with pleurisy of a like nature.

**Course — Duration — Termination.** — The variety of the affections accompanied by lactescent ascites explains the variable course of this syndrome. This form of ascites never proceeds in an identical manner. The stages of the process differ in aspect and in duration, but the termination is always fatal. Momentary arrest and remissions have been observed. In most cases, after a year or eighteen months, during which the tapplings follow at closer and closer intervals, the patient dies of cachexia. This fatal prognosis is perhaps the only point of difference between lactescent and ordinary ascites. Ascites with citron-like effusion can recover when the causative lesion is cured. Such cases are not uncommon.\* Lactescent ascites is never cured.

**Diagnosis.**—Lactescent ascites may be diagnosed by examination of the liquid. Fatty granules and leucocytes must be sought for, and the formula of the latter must be determined. Strauss's experiment (ingestion of large quantities of butter) might be tried, and does not present the slightest danger. As to the diagnostic value of lactescent ascites, it is of but small importance. It must be remembered, however, that it has been specially observed in tuberculosis and cancer of the peritoneum.

\* Dieulafoy, *Cliniques de l'Hôtel-Dieu*, 1898, 1899, 19<sup>m</sup> leçon.

## CHAPTER VII

### DISEASES OF THE LIVER

#### I. ANATOMY AND PHYSIOLOGY OF THE LIVER.

**Anatomy.**—Before describing the lesions of the liver, the cirrhoses which play such a large part in the pathology of this organ, the degenerations of all kinds (fatty, pigimentary, amyloid) which affect the cell in various ways, the changes in the bloodvessels and biliary canals, etc., it is essential to describe briefly its normal structure and functions.

The liver occupies the right hypochondrium, and weighs about 50 ounces. It receives two kinds of afferent vessels: (1) the hepatic artery for purposes of tissue nutrition; and (2) the portal vein, which brings the venous blood from the intestine, stomach, and spleen to the liver. It has only one set of efferent vessels—the hepatic veins, which pour the blood into the vena cava. In a section of the liver the portal veins may be easily distinguished from the hepatic veins. The portal veins are accompanied by the ramifications of Glisson's capsule, do not adhere to the tissue of the liver, and collapse, whereas the hepatic veins adhere to the tissue of the liver and remain open. On section of the liver we can observe with the naked eye or with a low-power microscope a number of small islands, coloured deeper at the centre than at the circumference, and giving to the liver a grained appearance. These islands represent the hepatic lobules.

The liver is formed by the union of lobules, and, according to Sappey, contains 1,200,000. If, therefore, we know the structure of one lobule, we know the structure of the whole liver. The **hepatic lobules** are small masses 1 millimetre wide and 2 millimetres long, and fixed on to the divisions of the hepatic vein like glandular acini to their excretory ducts. The polyhedral form of the lobules is due to mutual pressure. They are separated from one another by fissures and by triangular spaces caused by the blunting of their angles. In the centre of the lobule we find the suprahepatic vein, which is adherent to the tissue of the liver, and penetrates the lobule. It is a short branch of the extralobular hepatic vein. The suprahepatic vein is formed of fibro-elastic tissue, with a few smooth muscular fibres, and is lined with epithelium. In pathological conditions this fibrous thickened vein is an important histological landmark. Around the lobule, in the triangular spaces and fissures, are the portal veins, the hepatic artery, the biliary ducts, and the lymphatic vessels, bound together by connective tissue. The importance of these fissures and spaces in the pathological anatomy of the liver will be already apparent. Between the hepatic vein, which is central, and the portal veins, which are peripheral, we find capillaries carrying the blood from the circumference to the centre of the lobule, forming a venous network in the interior of the lobule, and united with one another by transverse anastomoses. In pathological conditions these capillaries are often obstructed by red corpuscles and microbes. In the meshes of this capillary network are the hepatic cells, directly connected with the network of the biliary canaliculi.



1. The **hepatic cells** constitute the essential and specific element of the liver. They are small blocks of a soft and granular substance, and their variety of shape is due to the pressure exerted on them by the surrounding cells and vessels. After a fast the cells are small and somewhat ill-defined, but during digestion they become large and very distinct. They are readily isolated. They are flattened, polygonal, so that each cell is in contact with six or seven of its neighbours, as well as with the blood and bile capillaries. The hepatic cells do not appear to have an envelope, and are formed of granular protoplasm containing one or two nuclei. They contain yellow granules of biliary pigment, with red granules of blood pigment and fatty granulations. They contain glycogen and the ferment by which the glycogen is transformed into sugar. This glycogenic matter is especially abundant in the cells at the centre of the lobules. It appears in little amorphous masses, which are almost fluid, and turn a mahogany-red colour with tincture of iodine. The cells radiate from the centre towards the circumference, thereby following the disposition of the vascular meshes, and are placed in a network at the periphery of the lobule (Frey).

The radiation of the cells gives them the appearance of little columns, sometimes called trabeculæ. Eberth has compared these trabeculæ with tubes, the tube being formed of hepatic cells surrounded by a membrane. This tubular disposition of the liver, which exists in some animals, especially in the seal, is seen in man in pathological conditions.

2. The **portal veins** which surround the hepatic lobule run in the spaces and fissures between the lobules. Each lobule is penetrated by capillaries derived from several portal vessels. These capillaries make their way in a radial direction from the periphery towards the centre of the lobule (Kolliker). They are united one to another by transverse anastomoses, and they anastomose with the capillaries of the central hepatic vein. It is by means of this network that the blood of the peripheral portal veins, after having traversed the mass of the cells, pours into the hepatic vein. It is in this network that the hepatic cells are contained. The blood capillaries make their way in the gutters hollowed out at the junction of the vertical edges of the hepatic cells. Hence, in a section of the liver perpendicular to the capillaries of the lobule, the hepatic cells have four sides, and their excavated angles receive a blood capillary, whereas the bile capillaries travel over the faces, and not over the angles, of the cells.

3. The **biliary canals** surrounding the hepatic lobule are formed of a thin enveloping membrane, lined internally by cubical epithelial cells. These canals are the terminal portions of the intralobular biliary canaliculi. The intralobular canaliculi make their way through the hepatic cells, forming narrow meshes. Their path is rectilinear, and their meshes are somewhat elongated, like those of the blood capillaries. "The biliary canaliculi pass between the faces of the cells, and consequently do not meet the capillaries of the blood, from which they are distant by at least half the face of a hepatic cell."

Some doubt exists regarding the structure of the intralobular canaliculi. Some authors assign to them a wall of their own, formed of juxtaposed flat cells (Legros); others say that they have only a borrowed wall, "formed by the condensation of the surface of the hepatic cells into a cuticle." The hepatic cell, without a proper wall, is said to be a simple modification of the epithelium of the biliary canaliculi, and to circumscribe them, after the manner of the secretory cells (Farabœuf). In any case, these intralobular canaliculi serve some purpose in the biliary secretion. They receive the bile secreted by the hepatic lobule.

4. The **connective tissue** of the hepatic lobule is derived from Glisson's capsule. On the surface of the liver this envelope is formed of two layers—the one superficial, serous, and covered by the endothelial cells of the peritoneum; the other deep, thicker, and formed of fibrous tissue. From this capsule bundles of connective tissue accompany the interlobar vessels, and help to fill the fissures and spaces between the lobules.

The perilobular connective tissue, in its turn, sends out an intralobular network which is adherent to the wall of the capillaries or extends between the capillaries in the form of a reticulated tissue. This tissue, united with the blood capillaries, forms a woof supporting the hepatic cells.

The **perilobular lymphatic vessels** form networks that accompany the portal vein. These lymphatic vessels are probably formed by lymphatic capillaries arising in the interior of the lobule (MacGillavry). The hepatic artery (nutrient vessel) supplies the lobules and the walls of the veins and biliary canals.

The hepatic lobule forms an **anatomical unit**, but this unit in no way corresponds to the ordination of pathological lesions. If we wish to discover our bearings in many lesions of the liver, we must come back to the conception of Eberth, who looks upon this organ as a tubular gland. His conception is borne out by comparative anatomy and embryology. The hepatic lobule constitutes in the human species the anatomical unit, but the pathological unit is the **biliary lobule** (Sabourin). The biliary lobule, the liver being considered as a tubular gland, "has as its glandular domain the parenchyma grouped around a portal space—a domain extending excentrically as far as the first system of subhepatic veins. This domain is not homogeneous, because it is composed of several hepatic lobules. Each hepatic lobule is, therefore, formed of distinct segments, each one belonging to different biliary lobules" (Sabourin).

**Physiology.**—The liver performs multiple functions, some well known, others still under study. I shall review them briefly.

1. The liver makes bile. It is not made, as was formerly held, by the biliary glands contained in the biliary canals. The biliary canals, no matter what their size, are only excretory ducts. The **secretory** organ of the bile is the hepatic cell. **Biligenesis** is one of the most important functions of the hepatic cell, which, bathed in the blood of the portal vein, extracts and elaborates the primary matter of the biliary excrements. The bile made by the cell is poured into the biliary canaliculi, which in turn lead it into the larger excretory canals.

It is chiefly during periods of fasting that the liver accomplishes its biligenic function, and it is towards the end of the meal that the excretion of bile into the intestine takes place. Man secretes about 40 ounces of bile in twenty-four hours, but a portion is reabsorbed. Fresh bile has a beautiful yellow colour, due to bilirubin. The first stage of oxidation causes the yellow bile to become green, the bilirubin being converted into biliverdin. Bilirubin is derived from the hæmoglobin of the blood, which is converted into hæmatin by the biliary acids, takes up water, and loses its ferruginous elements. Hæmoglobin is not present in normal bile. In the spectroscope bilirubin extends to the left of Fraunhofer's B line.

The biliary acids are likewise formed by the hepatic cells. Cholic and choleic acids are eliminated as salts of soda.

The bile shares with the pancreatic juice the power of emulsification and absorption of fat. A fatty condition of the fæcal matter is therefore seen in obstructive jaundice.

As the bile passes through the intestine, it has been thought to act as an antiseptic (Charrin and Roger).

The bile is a toxic liquid, and the intravenous injection in an animal of 5 or 6 grammes of bile per kilogramme of weight produces convulsions and death. The toxic action depends on the acids and colouring matter.

2. The liver makes glycogen, and the hepatic cell is charged with this important function. The question of hepatic glycogenesis, first discussed by Claude Bernard, will be treated in greater detail under Diabetes.

The liver possesses the power of making glycogen from every kind of food. I say every kind advisedly, because the formation of glycogen takes place in the hepatic cell, no matter what the kind of food may be, and apart from any starch and sugar.



Glycogen, or animal starch, is not the simple result of a transformation, but of a formation which is effected throughout the whole animal series by a mechanism analogous to that observed in the vegetable kingdom.

The sugar, absorbed by the intestine, does not remain in the liver as sugar, but is immediately transformed into glycogen, which is in its turn transformed into glucose, and poured into the general circulation as the economy demands it for nutrition and heat production.

In the formation and evolution of the immediate sugar principle there are two distinct phenomena: (1) The creation of the amylaceous matter in the hepatic cell—that is to say, the secretion of glycogen; (2) the chemical phenomenon, causing this principle to undergo successive transformations. A portion of the glycogen contained in the cell is stored there as a reserve ration; another portion is continually subject to the action of a ferment produced in the liver. By this ferment the glycogen is converted into glucose, which passes into the hepatic veins, and so to the whole system, to aid in the general nutrition and production of heat. Glycogen appears to undergo other successive transformations (carbonic acid, lactic acid, and no doubt fat).

During life these two phenomena—the secretion of glycogen and its transformation by the ferment—take place at the same time; after death the secretion of glycogen, which is the vital act, stops, whilst its decomposition into secondary products, which is the chemical act, continues. Accordingly, the liver of an animal may be washed several times, and still yield traces of glucose.

Writers have wished to make the glycogenic function general. Rougey, having found glycogen in other tissues—in the muscles, for example—though only in small proportions, wished to make glycogenesis a general nutritive act, and not a particular function of the liver. I cannot enter here into the full details of this question, but it is certain that the presence of glycogen in the muscles, testicles, ovaries, and placenta has not the importance formerly attributed to it. “These are phenomena subject to all the eventualities of feeding, and to all the varieties that are observed in the accidental phenomena of the economy, and must be distinguished from the constant functions” (Claude Bernard). The constant, invariable, and necessary function has devolved on the liver.

3. The formation of fat in the liver (I am not speaking of fatty degeneration) seems to result from a change in the glycogen similar to the production of wax by bees which nourish themselves on sugar, and the production of fat in geese, who, after an exclusive diet of starch and sugar, end by having an enormous liver (Persoz). The formation has not its exclusive seat in the liver, but this organ participates largely therein.

4. Lehmann's view that the liver is concerned in the production of the red corpuscles is no longer admitted. It is now held that the liver is an organ in which the red corpuscles are destroyed.

5. The theory advanced by several authors, and notably by Murchison, that the liver may be charged with the production of urea, has been upheld in France (Brouardel). According to the old theory, urea, considered as a phenomenon of combustion, or as the result of organic splitting up, was produced in the tissues of the whole economy. According to the new theory, urea, considered as the result of organic splitting up, is produced exclusively in the liver. We see the deductions which result therefrom. Pathological conditions, which exaggerate the normal functions of the organ, favour the increased production of urea. Pathological conditions, which tend to impair or destroy the function of the liver (acute atrophy), produce a noticeable diminution in the quantity of urea excreted in the urine. We shall see in the course of the following articles that this theory is not absolutely in accordance with facts, but we must nevertheless recognize that physiologically the liver takes an active part in urogenesis.

6. According to Schiff, another function of the liver is to arrest, neutralize, or destroy, the toxic substances absorbed in the intestine and contained in the portal

vein. The hepatic cell has to stop in part the alkaloids resulting from the putrid fermentation that goes on during intestinal digestion.

"In the intestine infection is constant. In every man and at every hour the agents of intestinal putrefaction are found in the intestinal cavity giving birth to toxic products which the intestine absorbs, and intoxication in various degrees is extremely frequent. If this intoxication is not more frequent, or if it is not of daily occurrence, it is because the organism is supplied with the means of self-protection. The liver arrests, destroys, or converts a part of the poisons absorbed by the intestine; the blood burns a part, and the kidneys eliminate the surplus" (Bouchard).

"The poisons of the gastro-intestinal tract are to a large extent produced by microbes which decompose the tertiary and quaternary substances of food. On the other hand, these very microbes produce toxines, so that infection and intoxication are thus intimately connected" (Hanot).

"The intestine is generally the open door for the poisons of intoxication, but the liver protects the organism by arresting the passage of the poisons in order to neutralize them or to return them into the intestine" (Bouchard).

"The liver, then, is not only the storehouse for the organism: it is also the advance fortress against intoxication. The recognition of the antitoxic power of the liver and of the auto-intoxication of intestinal origin show the pathological relations between the liver and the intestine" (Hanot). The liver arrests the vegetable alkaloids (morphia, quinine, curare) in the proportion of 50 per cent. It arrests the toxic substances of the bile which have been reabsorbed into the intestine.

## II. CONGESTION OF THE LIVER.

The extreme vascularity of the liver and the richness of its double system of capillaries, interposed between the general venous circulation and the heart, are favourable to congestion. In congestion of the liver, as in that of the lungs, or of the other viscera, we have not to describe a definite morbid entity, but only a morbid condition, made up of dissimilar elements. This chapter in pathology is therefore an enumeration of facts, most of which have no connection one with another, and in order to group these facts it is customary to unite them into active and passive congestion. The latter has acquired such an importance in connection with "the cardiac liver" that I shall devote a special section to its description.

**Active Congestion.**—Active congestion is caused by an excess of pressure in the afferent vessels (portal veins). The fullness of these vessels causes a marked increase in the size of the liver. Heavy meals, errors of diet, alcoholic excess, and purgatives, modify the portal circulation and stimulate the hepatic cells, causing vaso-dilatation and congestion. I may say the same of affections of the small intestine (tuberculosis) and of the colon (dysentery), in which case the portal blood carries irritating substances (microbes or toxines) to the liver. Congestion of the liver is frequent in the intermittent fevers and in the bilious remittent fevers of hot countries (Dutrouleau). It forms the first stage in certain diseases of the organ (cirrhosis, hepatitis). Suppression of the menses or of a hæmorrhoidal flux



may cause congestion of the liver. An attack of gout is sometimes preceded by congestion of the liver.

Amongst the causes which produce congestion of the liver, I may mention dyspeptic troubles and auto-intoxication of gastro-intestinal origin.

Dilatation of the stomach enters into the pathogenesis of hepatic congestion. "In 389 personal observations of dilatation of the stomach," says Bouchard, "I found swelling of the liver in the proportion of 23 per cent."

Congestion of the liver shows itself by a feeling of discomfort and heaviness in the right hypochondrium. Slight jaundice may be present, and the urine contains urobilin or biliary pigments. The liver is tender on pressure, and can be felt beyond the false ribs. Percussion gives an increase of several centimetres in the vertical diameter. This swelling increases or diminishes; it is subject to recurrences, and is sometimes the first stage of a commencing cirrhosis.

The **treatment** of hepatic congestion varies according to the cause.

### III. PASSIVE CONGESTION—CARDIAC LIVER.

**Pathogenesis.**—Passive congestion of the liver is caused by increased pressure in the efferent vessels (sublobular veins, vena cava). This venous stasis is due to many causes—diseases of the heart (lesions of the mitral and tricuspid valves), diseases of the lungs, which diminish the field of hæmatisation (emphysema and fibrosis), intrathoracic tumours, which impede the circulation in the inferior vena cava—but the most frequent causes are lesions of the mitral valve and changes in the cardiac muscle. When mitral lesions are ill-compensated, when the cardiac muscle is affected, and the left auricle is not completely emptied, there results an obstruction which extends throughout the whole of the lesser circulation to the vessels of the lungs, the right ventricle, and the right auricle. This blood-stasis hinders the return circulation in the inferior vena cava, and the hepatic veins and causes congestion of the liver. This congestion, which is at first temporary, ends by becoming permanent, and causes the anatomical changes of the **cardiac liver**.

The value of this mechanical theory for the cardiac liver, as created by Beau and Gendrin, must not be exaggerated. Defective hydraulics, due to the cardiac lesion, certainly play a large part in the determination of the hepatic changes, but the mechanical obstacle to the circulation is not the only cause. It is not rare to find people suffering from disease of the heart in whom the liver is attacked before the intermediary circulation—that of the lungs, for instance—and before the peripheral circulation (œdema of the legs) has been influenced by the cardiac lesion. In such a case it might

be held that the liver, having, under the influence of multiple causes, become an organ of *minoris resistentiæ*, is more liable than the other organs to feel the cardiac lesions.

These considerations are absolutely applicable to the auto-intoxications of intestinal origin, prolonged gastro-intestinal dyspepsia, alcoholism, malaria, gall-stones, and more or less general arterio-sclerosis. The liver withstands the effect of the cardiac lesions the better as it is prepared by its relations.

**Symptoms.**—The clinical picture of the cardiac liver varies. Let us select the most common types. In one variety the cardiac lesion is not advanced. The patient suffers slightly from palpitation, and can neither ascend a staircase nor walk quickly without getting out of breath. Malleolar œdema is common, but there are as yet no serious symptoms. Later, in consequence of fatigue, of excesses in the use of drink and food, or even without any appreciable cause, the patient suddenly experiences digestive troubles, distension of the belly, and a feeling of weight in the liver. He complains of sharp pains in the right hypochondrium, and has “a weight on his stomach.” The dyspnœa and the cardiac arrhythmia grow worse, as if the liver, in its turn, reacted on the cardio-pulmonary circulation (Potain). On examining the patient, we notice a yellowish tint of the conjunctivæ and the face, but the urine does not always contain bile pigment. There is no ascites, but the œdema of the lower limbs progresses. On auscultating the heart, a mitral lesion is found, and in the lungs râles, due to congestion and œdema, are heard at both bases. The urine is scanty, of a red-brown colour, and contains urobilin. At this period the liver is large and but slightly indurated, being as yet only congested. It is known as the **nutmeg liver**, and only becomes hard and cirrhotic (red atrophy) later, if the hepatic lesion persists, or if it is complicated by further changes. If we institute efficient measures, such as leeches or wet-cupping to the hepatic region, a milk diet, and diuretic wine or small doses of digitalis, in a little while the congestion of the liver will disappear, and for the time being the danger is removed.

In another patient events may take a different course. The hepatic mischief is more advanced, the mitral lesion is of longer duration, and the cardiac muscle is losing its strength. The lungs are much congested, the kidneys act badly, the urine is of a deep colour, and contains a brownish pigment, the yellow colour of the skin is more evident, and the œdema tends to become general. Palpation and percussion show that the liver is hard and painful; in size it is either enlarged or normal, but the ascites often makes exact examination impossible. The treatment referred to above is put into practice, but does not produce the same effect, because the cardiac liver is now cirrhotic.



In short, the hepatic congestion of cardiac patients is transitory before becoming permanent. It may be considerable during the attacks of asystole. Feelings of heaviness and of pain in the hypochondrium, a yellowish colour of the skin, and dyspeptic troubles are rarely absent. The liver is tender on pressure, and increased in size. Later the cirrhotic liver may diminish in size, the symptoms become more severe, ascites is frequent, and symptoms of **icterus gravis** often supervene.

In certain cardiac cases the hepatic troubles precede the other manifestations of heart disease; in others (tricuspid insufficiency) the reflux of the blood produces hepatic pulsation (Potain).

**Pathological Anatomy.**—The cardiac liver varies according to the period at which it is examined. In the first period it is congested, hypertrophied, and weighs 4 to 6 pounds, while its sharp edge becomes rounded and its surface is smooth. On section, the lobules are increased in size, and form islets, red in the centre and greyish at the periphery. This double coloration, which is only an exaggeration of the normal condition, gives the liver a granular appearance, whence the name of **nutmeg liver**. Under the microscope the central part of the lobule is of a mahogany-red colour, from the dilatation and congestion of the hepatic vein, from the enlargement and fullness of the neighbouring capillaries, and from the changes in the flattened and atrophied liver cells, the protoplasm often containing granules of pigment and crystals of hæmatoidin. The peripheral portion of the lobule is greyish or opaque, because it is relatively anæmic, and also because many of the hepatic cells are spherical and rich in fatty granules, the blood in the portal veins becoming stagnant at the periphery of the lobule, and leaving the fat derived from digestion in the peripheral cells. At a more advanced period the liver may atrophy (**red atrophy**); its surface becomes slightly granulated, and the double colouring of the islets is less distinctly defined. Under the microscope we see that the central vein and the central capillaries are dilated and congested with blood, so as to resemble the blood tumours of the liver; hence the name of “red atrophy” given to this stage of the cardiac liver. The hepatic cells of the central zone have partially disappeared, and are replaced by young connective tissue, with periphlebitis and thickening of the external coat of the vessels. The liver is also indurated, and this fibrous change in the congested cardiac liver has given to the lesion the name of **cardiac cirrhosis**.

Some authors do not admit this central fibrosis of the hepatic lobule, or, at least, they consider it inconstant, rudimentary, and limited to the walls of the hepatic vein only, whilst there is at the periphery of the hepatic lobule a fibrosis which accompanies the ramifications of the portal vein, and is somewhat analogous to the lesions of Laënnec's atrophic cirrhosis. Thus understood, cirrhosis in the cardiac liver would be a **bivenous**

cirrhosis, and the hepatic lobule would be invaded from all sides—at its centre from the hepatic vein, and at its periphery from the portal veins. We should find, therefore, **endo-periarteritis** of the hepatic arterioles. This opinion has been upheld by Wickham Legg and Talamon.

Recent researches have given to each of these changes their proper value. As a matter of fact, perilobular fibrosis and endo-periarteritis are found amongst the lesions in the cardiac liver, but this peripheral fibrosis is not directly associated with the lesions of the cardiac liver, properly so called. They are superadded fibrous lesions, and form a part of a more general fibrous process. Thus, to quote examples, patients attacked with these bastard forms of cardiac cirrhosis suffer at the same time either from Bright's disease, or are alcoholics, and have in consequence more or less general arterio-sclerosis. The characteristic lesion of the nutmeg (period of vascular enlargement) and cirrhotic liver (period of connective tissue formation) chiefly affects the centre of the hepatic lobule—that is to say, the area of the hepatic veins. The lesions are more or less extensive, according as the process that produces the cardiac liver is cortical, subcapsular, or general.

In "this complete remodelling of the hepatic parenchyma" (Chauffard) the microscopic appearance of the sections presents certain peculiarities, carefully described by Sabourin. We see hepatic islets which have no longer the hepatic vein in the centre of the islet, but a porto-biliary space, and around these islets zones of trabecular atrophy form a network in which, at the points of convergence, the hepatic veins are found. The type of the normal hepatic lobule is thus reversed, and the porto-biliary canal actually forms the centre of the figure."

The lesions of the cardiac liver are sometimes accompanied by catarrh of the bile-ducts and jaundice.

The **treatment** of passive congestion of the liver consists in lowering the blood-pressure in the venæ cavæ and hepatic veins. Diuretic drinks, milk diet, Trousseau's diuretic wine, digitalis, leeches applied to the anus, leeches and wet-cupping over the liver, saline purgatives, and Vichy, Carlsbad, or Tharasp water, are the means most employed.

#### IV. CIRRHOSIS OF THE LIVER IN GENERAL.

The word **cirrhosis** has been preserved to conform to the usage established by Laënnec. Cirrhosis of the liver, otherwise called sclerosis, or chronic hepatitis, forms a large part of the pathology of this organ. It is characterized by the overgrowth of the normal connective tissue, extending from Glisson's capsule into the hepatic lobules. In the first stage the fibroplastic tissue is embryonic, and formed of cells analogous to leucocytes. In this embryonic connective tissue flat cells and bundles of fibrils appear, and the morbid process, continuing its growth, ends in a morbid tissue which is more



or less invading, more or less dense, and more or less fibrous and retractile, as the case may be.

The formation of cirrhotic tissue is not a matter of chance, and the fibrous tissue is not produced here or there in an indeterminate manner. It is almost **systematic** in its invasion. Thus, according to the case, the cirrhosis follows the course of the veins, the biliary canaliculi, or the arterioles. Venous cirrhosis is associated either with lesions of the central vein of the lobule (cardiac liver), with the combined lesions of the peripheral portal veins, or with the combined lesions of the central and peripheral veins (bivenous cirrhosis, Laënnec's atrophic cirrhosis, hypertrophic alcoholic cirrhosis). Biliary cirrhosis is associated with the lesions of the biliary canals, and the most important variety is called hypertrophic biliary cirrhosis. In many circumstances the toxic agent attacks the hepatic cells themselves, and produces divers changes. It was formerly said that the fibrous lesions might arise from changes in the cells of the hepatic lobule, and that there was a double lesion—**interstitial hepatitis**, having its origin in the connective tissue; and **parenchymatous hepatitis**, or **visceral cirrhosis**, having its origin in the hepatic cells. The epithelial change is now denied. "In reality, the doctrine of epithelial cirrhosis has had its day" (Letulle).

These varieties of origin, extension, topography, and evolution of the morbid connective tissue are associated with the condition of the cells (atrophy, disappearance, fatty degeneration, pigmentation, or adenoma) that imprints on fibrosis of the liver its peculiar characteristics.

**Division.**—Fibroses of the liver may be **partial** or **general**, **primary** or **secondary**, and **isolated** or **associated** with other diseases, such as syphilis, alcoholism, malaria, diabetes, arterio-sclerosis, or diseases of the heart and kidneys.

1. **Partial** fibroses occur as **secondary** lesions in several diseases of the liver. Fibrous tissue is found around tumours, in the neighbourhood of syphilitic gummata, hydatids, or angiomas. In these examples of partial fibrosis the overgrowth of connective tissue is not of great interest.

2. Fibrosis in some cases occurs at the same time as some other lesion of the liver. We meet with fatty livers which are likewise fibrous, and side by side with the cells filled with fat we find hyperplasia of the connective tissue, which sometimes penetrates the interior of the lobule. The liver is indurated, smooth on section, and increased in size. I shall describe a type of this variety later under the name of **hypertrophic fatty cirrhosis** (Hutinel-Sabourin type). I shall also describe cirrhosis associated with **tuberculosis** of the liver. When fibrosis is combined with **amyloid** degeneration, the liver has not the enormous size of the purely lardaceous organ, and is perhaps even below the normal size.

3. We find **bastard secondary** fibroses in which the lesion in the liver is associated with disease of the heart and kidneys. I have described elsewhere the changes found in the **cardiac liver**, so that I need not return to them. We shall see later under Diseases of the Kidneys that hepatic cirrhosis may accompany the complex process of **Bright's disease**. This form, as also **malarial** and **diabetic** cirrhosis, will be described in due course.

4. For the time being I will limit the description of cirrhosis of the liver to certain well-defined varieties—viz., Laënnec's atrophic cirrhosis, hypertrophic alcoholic cirrhosis, hypertrophic biliary cirrhosis, and syphilitic cirrhosis—but yet some of these varieties are not always clearly defined, and may give rise to mixed or intermediate forms.

5. **Adenoma** is in some cases associated with cirrhotic lesions. The adenoma is sometimes discrete, but at other times it takes a most important place.

The question of the **regeneration** of the liver will also claim our attention (compensatory hyperplasia).

Before describing cirrhosis of the liver, it will be useful to give a historical sketch of this question.

**History.**—The history of cirrhosis of the liver dates from Laënnec's time, and it may even be said that, following Laënnec's example, only one variety of hepatitis was for a long time described—viz., **atrophic cirrhosis**. Laënnec used the name **cirrhosis** (κίρρος, red) on account of the colour of the cirrhotic liver. Although this term is imperfect in that it only applies to an inconstant trait of the lesion, and although the name **sclerosis** (σκληρός, hard), or chronic **hepatitis**, might be substituted, I shall nevertheless retain the name, so as to conform to custom. Laënnec was mistaken as to the nature of the lesion, because cirrhosis, in his mind, represented a production which he called "cirrhosis," and which he believed to be analogous with scirrhus; but in all other points his description is typical. With admirable precision he sketched in a few words the characters and the course of a disease previously unknown. Hanot had the happy idea of giving to this disease the name of Laënnec's cirrhosis.

In the year 1827 Bright stated positive ideas with regard to sclerotic hepatitis. He cites as a cause alcoholism. As lesions, he describes the changes in the liver, fibrous peritonitis, sclerosis of the intestine, and the illustrations in his book show several types of cirrhosis. He noticed that the lesions "produce general obstruction of the circulation through the branches of the portal vein, and thus become the immediate cause of the ascites, independently of the morbid conditions which may affect the blood from the fact that it has not left behind in the liver the substances which ought to be eliminated by the bile."

With Kiernan the pathological anatomy of cirrhosis takes a decided step forward. The English author, in his remarkable "Researches on the Structure of the Liver," proves the normal existence of a connective web which surrounds and penetrates the hepatic lobule, and he attributes cirrhosis to the abnormal development of this web. In Gubler's thesis for the Fellowship, which bears the date 1853, the ideas common to that period are collected. It shows us that up to that time only one variety of cirrhosis, with very few exceptions, was admitted—viz., that which ended in atrophy of the liver. We see that the cirrhotic liver may show itself in the hypertrophic form, but for a long time to come Laënnec's **atrophic cirrhosis** was the only variety known. Nevertheless, post-mortem examinations revealed **enlarged** and cirrhotic livers that had not the appearance of common cirrhosis; but, as the profession was convinced that cirrhosis, even when it commences with hypertrophy, must end in atrophy, all enlarged livers were considered to represent the initial phase of ordinary cirrhosis, the morbid process commencing with hypertrophy of the organ, and ending with atrophy. Moreover, this manner of regarding matters has been admitted by certain German authors (Birch-Hirschfeld). This interpretation perpetuates an error. I do not say that atrophic cirrhosis cannot commence with temporary hypertrophy; I do not say that in certain **mixed** forms which we shall study later Laënnec's cirrhotic liver may not weigh more than normal; but these cases are in no way opposed to the existence of so-called **hypertrophic cirrhosis**, where the liver remains increased in size during the whole disease.

The autonomy of **hypertrophic cirrhosis** had been foreseen by Requin, and clearly formulated by Told. Jaccoud had observed and discussed it, but it was only accepted in France after the publication of Olliver's monograph, in which he points out the clinical characters of this affection, and assigns to it anatomical characters previously indicated by Charcot and Luys.

From this epoch hypertrophic cirrhosis served as a text for numerous works. Hayem studied the disposition of the sclerotic tissue. Cornil observed the inflammation, the dilatation of the biliary canaliculi, and the formation of an intra- and extra-lobular network of bile-ducts. Hanot proved that the inflammation of the small biliary canals in the portal spaces is the starting-point for the lesions. He created hypertrophic biliary cirrhosis, to which the name of "Hanot's disease" has been rightly given.



On the other hand, the experiments begun by Legg and completed by Charcot and Gombault prove that permanent ligature of the common bile-duct produces in animals angiocholitis and periangiocholitis, and consequently connective hyperplasia, somewhat analogous to the change seen in hypertrophic biliary cirrhosis. Hypertrophic cirrhosis is, therefore, an absolutely distinct variety. Its pathogenesis and its anatomical and clinical characters differentiate it from Laënnec's atrophic cirrhosis, and are so distinct that it seems impossible to reconcile these two species. The distinctive characteristics may be thus stated :

1. Laënnec's **atrophic cirrhosis** is of bivenous origin (periportal and hepatic). This cirrhosis is annular, multilobular, and extralobular. The liver is atrophied and granular. The clinical characteristics are as follows : ascites, the development of a collateral circulation, frequent hæmorrhage, absence of jaundice, and average duration twelve to eighteen months.

2. **Hypertrophic biliary cirrhosis** is of biliary origin—that is to say, the process commences with angiocholitis of the bile-ducts. The sclerosis is monolobular, insular, extra- and intralobular. The liver shows general enlargement. The clinical characteristics are as follows : progressive and persistent jaundice, enormous enlargement of the liver, enlarged spleen, absence of ascites and collateral circulation, little hæmorrhage, average duration three to eight years. Do these two cirrheses really form two distinct types ? Are not their lesions and their symptoms often combined ? Cannot they give rise to mixed and various forms ? I shall attempt to answer these questions, but I may state at the outset :

1. Side by side with Laënnec's atrophic cirrhosis there is room for other atrophic varieties of cirrhosis.

2. Side by side with hypertrophic biliary cirrhosis there is room for other forms of hypertrophic cirrhosis, with or without jaundice.

3. Between the atrophic bivenous type of cirrhosis and the hypertrophic biliary type there is room for **intermediate forms**. This question will be discussed under **Mixed Cirrheses**.

## V. VENOUS CIRRHOSIS—LAËNNEC'S ATROPHIC CIRRHOSIS—CIRRHOSIS BY GASTRO-INTESTINAL AUTO-INTOXICATION.

**Pathological Anatomy.**—In atrophic cirrhosis the liver is always diminished in size, and the atrophy principally affects the left lobe (Freichs). The theory that atrophic cirrhosis commences with hypertrophy must not be rejected altogether, for there are cases in which the initial congestion increases the size of the organ. At an advanced period the liver is atrophied and may not weigh more than half the normal weight. Its colour is brown, reddish (*κίρρος*, red), yellowish, or greyish, according to the predominance of the biliary or fatty elements. The edge of the liver is blunted, and often furrowed by fibrous bands. The surface of the liver is nodular, and studded with little hard masses that are mammillated, of a ruddy yellow, and varying in size from a pin's head to a hazel-nut. These **granulations** are formed by larger or smaller masses of hepatic lobules, surrounded by fibrous tissue. These details are better seen after removal of Glisson's capsule, which is generally adherent, opaque, and thickened. The granulations sometimes resemble the heads of nails, whence the name **hobnailed liver** of English

writers. In some cases the parenchyma is strangled by the fibrous zones, protrudes like a cauliflower, and the liver is thus divided into lobes.

The tissue of a cirrhotic liver is not easily torn. It is hard, creaks under the knife, and is not friable. On section, granules of various sizes are found, similar to those on the surface. They are surrounded by fibrous tissue, which forms rings around them, and from which they emerge, as though they were about to become enucleated. The granules may be enucleated when the specimen is macerated in water.

**Histological Examination.**—Atrophic cirrhosis of the liver consists in hyperplasia of the connective tissue in **various phases of growth**—embryonic cells, fusiform cells, fibrillary and fibrous tissue. The process commences as an embryonic neoplasia, and ends in **fibrous retractile** tissue.

The fibrous tissue of atrophic cirrhosis is formed in the **portal spaces around the portal veins**, and in the centre of the lobules around the **hepatic veins**. These two cirrhotic systems, the one peripheral and the other central, occur together, and are united by numerous anastomoses. The cirrhosis is thus **bivenous**.

In the midst of the fibrous connective tissue around the portal spaces, which stains red with picrocarmine, the portal veins, biliary canaliculi, and capillaries are seen. The portal veins, attacked by phlebitis and periphlebitis, are particularly striking; but the biliary canaliculi, so prominent in hypertrophic cirrhosis, where they are attacked by angiocholitis, are here much less apparent than the veins.

The fibrous tissue surrounds a certain number of hepatic lobules *en bloc*, and thus forms large granulations, whence secondary tracts arise and form smaller ones. Even the smallest granulations are nearly always made up of several hepatic lobules, whence the name “multilobular cirrhosis.” They are often surrounded by a ring of cirrhotic tissue, whence the name of “annular cirrhosis.”

In many specimens orientation becomes impossible, because the normal topography of the lobule is completely changed. The hepatic lobules in cirrhosis are more or less fused together. Neither the spaces nor the fissures are seen, as in the normal condition. “Orientation is made the more difficult because the central vein, the landmark *par excellence* in physiological conditions, is here very difficult to find.” In the interior of the lobules there is the same disorder; the trabeculæ have no longer their radial disposition, and some of the lobules are cut off and represented by only a few cells.

This complete recasting of the lobules produces an unnatural topography, even at the commencement of the lesions.

Under the influence of the sclerogenous process, what becomes of the different elements in the hepatic lobule?

1. The **hepatic cells** lose their radial disposition in the lobule, and



leave no free space between their columns. They are heaped up, overturned, and deformed, especially at the periphery. Some of them show simple atrophy; others are infiltrated with fat and bile pigment. This atrophy of the cells, which is the rule at some period in atrophic cirrhosis, is the exception in hypertrophic biliary cirrhosis, where the cells are hypertrophied, preserving, as a rule, their normal characteristics, being neither infiltrated with fat nor with pigment (Hanot).

2. The **ramifications of the portal vein** in the portal spaces are invaded by phlebitis and periphlebitis. They are dilated at the commencement of the cirrhosis, during the period of embryonic growth. Later they are thickened, lose their elasticity, and become the seat of coagulation, and occasionally of extensive thrombosis. When the cirrhotic tissue has become fibrous, the tissue is still vascular, tortuous vessels of new formation being seen. "They are canals, hollowed out in the indurated connective tissue, and their walls, modified by the inflammation, have become blended with the neighbouring tissue, so as to form one structure with it." According to most authors, this vascular network is formed at the expense of the portal vein. According to others (Frerichs, Ackermann), a rarefaction of the portal network takes place, and the arterial network (hepatic artery) tends to substitute itself for the portal venules in which the circulation is embarrassed. However this may be, the **obstacle** to the circulation of the blood in the liver arises from several causes—lesions of the portal veins, thromboses in their interior, and changes in the vessel walls, entailing loss of elasticity and contractility in the vessels. Injections of fluid in the cirrhotic liver pass from the portal to the hepatic veins with some difficulty, and it is partly from this circulatory embarrassment in the portal system that a collateral development of **supplementary veins** takes place.

3. The **hepatic veins** are attacked by obliterating phlebitis. Their internal coat is fibroid and granular, and their lumen is constricted or obliterated. In some cases the cirrhosis, instead of being **bivenous**, is so clearly localized to the portal or hepatic system that it may be called **monovenous**.

4. In the sclerosed portal spaces a fairly large number of **biliary canaliculi** are seen. The canaliculi of a certain size are not attacked by angiocholitis and periangiocholitis, as in hypertrophic biliary cirrhosis, but the development of biliary capillaries is found here, as in all forms of hepatitis. The mode of formation of this biliary network will be studied under Hypertrophic Cirrhosis, in which it attains its greatest development. The more or less obliterating catarrh seen in some of the biliary canaliculi explains the slight jaundice found in some cases of atrophic cirrhosis (Hanot).

5. Cirrhosis is sometimes associated with nodules, forming a tumour. They have been described as **adenomata**. According to circumstances,

the adenoma has the size of a millet-seed, a hazel-nut, or a small egg, and invades the liver in a more or less considerable degree. The discrete or confluent islets of adenoma do not project on section, like those of cirrhosis. They are homogeneous, yellowish, old, or soft like mastic (Sabourin), and sometimes so little adherent to the wall around them that they escape on gentle washing.

Adenoma is essentially an infecting production of epithelial origin, and will be studied later under Primary Cancer of the Liver. The proof of its malignant nature lies in the fact that it spreads to the peritoneum and to the lungs. The lymphatic glands are, however, not affected. In the liver, as elsewhere (kidneys, uterus, stomach), the association of cirrhosis and adenoma is not rare, and it is the association of adenoma with hepatic cirrhosis which hastens the course of the disease and makes the prognosis worse.

The **systematization** of cirrhosis and its commencement in the **vascular** system of the liver are now called in question. Several authors would assign a more important part to the hepatic cell. "In most diseases of the liver the fibrosis is not systematic, and always has as a **primordial** cause a change in the hepatic cell."

To sum up, in the words of Brault, "it is not always necessary to look upon fibrosis of a viscus as being derived from fibrosis of its vessels, and it is indeed more natural to admit a simultaneous fibrosis affecting the vessel-walls and the interstitial tissue of the viscus. To be sure, "the reaction of the interstitial on the epithelial lesions, and *vice versa*," must be accepted, but it is more natural to admit that the different parts of the viscus, its vessels and connective tissue, are attacked simultaneously by the toxic-infectious process, which shows itself in different ways, according to the element invaded.

Finally, as far as alcoholic cirrhosis is concerned, whether the poison (alcohol) attacks the cell at the same time or before it attacks the veins, and at the same time or after the connective tissue, it is none the less carried to the liver by the portal veins, the cirrhosis being of **venous origin**.

6. The quantity of liquid in the peritoneal cavity (**ascites**) is more or less considerable. The perihepatic **peritoneum** is almost always affected, and this partial peritonitis is sometimes characterized by villous filaments, or at other times by free or adherent membranes. Adhesive perihepatic peritonitis establishes communications between the liver and the abdominal walls, and these adhesions are furrowed with veins, which, in their turn, serve for the re-establishment of the complementary circulation. The whole radicular system of the portal vein may show changes, which I shall discuss under Ascites. The **intestine** is sometimes shortened and thickened, and the **mesentery** is retracted.



**Pleurisy** must be mentioned : it may be dry or accompanied by effusion, which is rarely hæmorrhagic. The **spleen** is enlarged in half the number of cases, but not as much as in hypertrophic cirrhosis. This swelling is not solely due to blood stasis in the portal system, but to an active splenitis. The lesions of the **kidneys** are concomitant with, and not dependent on, those of the liver. The varicose dilatation of the veins of the **œsophagus** is explained by the fact that the veins of its lower two-thirds empty into the portal vein. Grave hæmorrhages sometimes result therefrom.

**Symptoms.**—The initial symptoms of atrophic cirrhosis are **insidious** and **variable**. Most frequently the patient during this indefinite phase experiences anorexia, nausea, heaviness in the right hypochondrium, and alternating diarrhœa and constipation. Itching, epistaxis, and hæmorrhoids are often present in this initial phase. In some patients the cirrhosis announces itself by congestion and pain in the liver. In many cases before any trace of ascites, the belly is distended and tympanitic. “The wind precedes the rain,” as Portal says, and, as a matter of fact, nothing is more frequent than this initial tympanites. Even in the initial period the decay of the hepatic cell is shown by alimentary glycosuria, urobilin in the urine, and pigmentary acholia.

To sum up, “Dyspeptic troubles, meteorism, constipation, urobilinuria, a jaundiced or bronzed tint, pigmentary acholia, alimentary glycosuria, pruritus, epistaxis, hæmorrhages from the gums, hæmorrhoids, localized œdema, and diarrhœa,” are the symptoms of the precirrhotic period. Hanot has given to them the name of **hepatism**, by analogy with the symptoms which I have denominated **Brightism**.

In some cases the usual symptoms of the onset are absent, or, at least, they pass unnoticed, and **ascites** is the first sign, accompanied or not by abdominal tympanites and complementary circulation. Sometimes œdema of the lower limbs is the first apparent sign. Loss of flesh may also precede the other symptoms. Whatever may be the mode of onset, a time comes when the patient's aspect is characteristic. The skin is dry and earthy, the face is marked by small dilated veins, the loss of flesh is rapid, the digestive troubles increase in severity, the belly is distended, and the œdema invades the lower limbs. The urine is brown, rich in urates, and poor in urea (Brouardel), and sooner or later three chief symptoms usually appear : (1) ascites ; (2) development of a complementary circulation ; (3) diminution in the size of the liver. The spleen sometimes increases in size.

1. **Ascites.**—Ascites must first demand our attention. Ascites, however, is almost always preceded or accompanied by **abdominal tympanites**, to which I attach great value. Sometimes the meteorism is so marked that the ascites is of minor importance. The tympanites gives an exaggerated resonance on percussion, and interferes with the exact delimitation

of the liver. Ascites generally takes several weeks to develop, and increases slowly. Sometimes its appearance is so sudden that the amount of fluid is large in a few days. When the ascites is considerable (20 to 30 pints), the abdomen has a peculiar shape. When the patient lies down, the umbilicus protrudes, and the bulging flanks recall the belly of the bac-trachians. A slight tap on the side of the abdomen, whilst the other hand is applied to the opposite side, gives a fluid wave. The dullness is complete over the whole region invaded by the fluid, and unless there is very great fullness the fluid can be readily displaced. This sign is obtained by making the patient lie alternately on the left and then on the right side. Percussion of the flanks shows that each side is dull and resonant in turn. The area and limits of the dullness change if the patient stands up. Auscultation sometimes shows the transmission of the heart and lung sounds to the lower part of the belly (Widal).

Owing to its increase, ascites becomes one of the most painful symptoms. It interferes with movement and with respiration. Although it has but little tendency to absorption, it has been observed to diminish temporarily or permanently through the action of purgatives and diuretic drugs. It may be cured by tapping of the abdomen.

The pathogenesis of ascites in atrophic cirrhosis is not clearly known. For a long time the following mechanical theory obtained: The changes of the portal veins in the liver, the transformation of their walls, the constriction of their lumen, and their obliteration by thrombosis, are an obstacle to the intrahepatic circulation, so that effusion may take place into the peritoneum, just as œdema forms in the cellular tissue of the leg when the chief veins are obliterated. There is, however, a corrective for this obstacle to the intrahepatic venous circulation—viz., the complementary circulation, which retards or lessens the production of ascitic fluid. And in order to make the theory complete, a kind of equilibrium between the production of ascites and the development of the complementary circulation has been admitted, the ascites becoming less marked as the collateral circulation becomes more developed.

The influence of these mechanical causes on the production of ascites cannot be denied, and yet we must recognize that this theory is frequently at fault. In the first place, ascites sometimes appears as the initial symptom of cirrhosis when the intrahepatic circulation is so little affected that it excites no complementary extrahepatic circulation. In such a case, therefore, it cannot be said that the ascites comes from a **mechanical obstruction**. On the other hand, cirrhosis of the liver may reach the atrophic stage without the production of any ascites, as is clearly shown by the post-mortem records published on this subject (Hanot and Lécorché). If ascites were only the result of a mechanical obstruction to the intra-



hepatic circulation, it would have occurred in the cases in question, the more readily in that there was not any complementary circulation.

If the mechanical obstacle in the intrahepatic circulation cannot alone explain the formation of ascites, where are we to seek for its causes? Peritoneal lesions have been suggested. As a matter of fact, perihepatic and peritoneal lesions are always found post mortem, and may assist in the production of ascites. In addition to peritoneal inflammations directly associated with hepatic cirrhosis, Leudet has described chronic peritonitis in alcoholic patients, which appeared on its own account (the liver not being in fault), or which may appear at the same time as the cirrhotic lesions in the liver. This latent peritonitis betrays itself sooner or later by abundant ascites.

*Another Cause of Ascites.*—Cirrhosis of the liver is fairly frequently associated with **tuberculosis**. I do not merely allude to one of the forms of hypertrophic cirrhosis, which I shall describe in the following section. I refer to those cases in which we find post mortem old tuberculosis of the lung, which may be very limited, or even healthy lungs, the tubercular lesion affecting only the liver and the peritoneum. This lesion consists sometimes of a few miliary granulations or greyish patches that are hardly visible to the naked eye, and appear under the microscope in the form of tubercular follicles with giant cells and tubercle bacilli. These peritoneal lesions have their share in the production of ascites.

The recognition of these facts show the part of the peritoneum in the production of ascites in atrophic cirrhosis. The question, however, is not exhausted. I ask whether ascites in cirrhotic patients is not caused by lesions in the mesenteric and peritoneal tributaries of the portal vein? While the researches of Giraudeau and myself do not give an affirmative answer, they deserve notice.

In a patient who had died of atrophic cirrhosis without peritonitis we found in the stomach, intestine, and mesentery phlebitis of the small veins, which were at least 2 millimetres in diameter. At some points the external coat of the small veins was double or treble its normal size, while the other coats were atrophied or had disappeared. In the most diseased portions the periphlebitis of the tributaries of the portal vein gave rise to fibrous prolongations, which joined the tracts arising around the neighbouring veins, and enclosed the arteriole and the nerves in the vicinity. If new discoveries confirm these data, they may prove that the systematic venous inflammation which dominates the process of atrophic cirrhosis, affects the tributaries to the same extent as the terminals of the portal vein. They would also help to explain certain early symptoms (hæmorrhage from the œsophagus, stomach, or intestine, and intestinal discharges), which would not then be set down to blood stasis alone. They would have their share in the pathogenesis of ascites.

Let me add that **cold** may have some influence in the production of ascites in cirrhosis (Potain).

2. **Collateral Circulation.**—Whilst the circulation is impeded in the

interior of the cirrhotic liver, we find a **collateral circulation** outside the organ, which serves to return to the heart by a roundabout way the blood arrested in the portal system of the liver. This collateral circulation is thus established. In addition to the blood received from the portal vein, the liver also receives venous blood from various small veins. These veins, collected into five groups by Sappey, form the accessory portal system.

They arise in organs or regions other than the digestive system, and end in the liver or in the portal vein where it enters the liver, so that each accessory vein represents a small portal system. The fourth group arises in the diaphragm, passes through the suspensory ligament, and empties into the lobules of the liver, to which this ligament is adherent. This group anastomoses, on the one hand, in the liver, with the branches of the portal vein, and, on the other, with the diaphragmatic and subcutaneous thoracic veins. It may thus serve to establish a circulation between the liver and the veins of the thorax. The fifth group arises from the sub-umbilical portion of the anterior wall of the abdomen, and in part empties itself into the left branch of the portal vein. The small veins of this group unite the trunk of the portal vein with the deep epigastric and internal mammary veins, and with the cutaneous veins of the abdomen.

The accessory portal veins, which are but slightly developed in the normal condition, are much developed in obstruction of the portal vein. It is by them that the circulation is partly re-established. Let us suppose an obstacle caused by cirrhosis : the blood of the portal vein creates new routes for itself, and a portion of the blood flows back from the liver through the accessory veins of the fourth group into the mammary and intercostal veins, which carry it into the azygos veins. In the collateral circulation effected by this group the course of the blood is from below upwards. Another portion of the blood in the portal vein finds a new route by flowing back from the trunk of the portal vein into the veins of the fifth group, which lead it to the epigastric and subcutaneous abdominal veins, the former emptying into the iliac and the latter into the saphena veins. In the collateral circulation effected by this group the course of the blood in the veins of the abdominal walls is from above downwards. Let me finally mention the veins in the **adhesions** that are sometimes found between the peritoneum and the abdominal wall and the diaphragm.

The dilatation of the subcutaneous veins of the abdomen gives rise in some patients to a varicose plexus at the umbilicus, and a well-marked venous network between the xiphoid cartilage and the pubis, especially on the right side and towards the upper half of the abdomen. In certain cases a thrill is felt and a murmur is heard in the dilated veins. The congestion of the saphena vein from backward pressure plays some part in the œdema of the lower limbs.



This collateral circulation may retard, but does not prevent, the appearance of the ascites, and though these venous dilatations seem to be the result of a mechanical obstacle to the blood in its passage through the liver, there are nevertheless cases of atrophic cirrhosis in which a collateral circulation is absent. Furthermore, we do not always find the equilibrium between the ascites and the circulation necessitated by an entirely mechanical theory. It would seem, as a matter of fact, that if we are to rely on the mechanical production of ascites and of the collateral circulation, there ought to be more intimate bonds between these two symptoms. This is not the case, and close observation shows that each of them enjoys a certain independence.

3. **Atrophy of the Liver**, which depends less on the retractile power of the fibrous tissue than on the disappearance or atrophy of a large number of hepatic cells, is a constant symptom in advanced cirrhosis, but it is not always easy to discover the atrophy on account of the tympanites and ascites. It is sometimes possible to feel the lower edge of the liver, and to make certain that it has become blunted and irregular.

Besides the symptoms just described, others occur in the course of atrophic cirrhosis, and are of great value. The spleen becomes hypertrophied in half the number of cases (Frerichs), and sometimes atrophied. Some subjects suffer from **œdema** of the lower limbs, which, as I have already said, precedes or accompanies the development of ascites. This œdema sometimes develops early, and, as it becomes considerable, it may lead to the supposition, through lack of care, that there is a cardiac lesion.

4. Attacks of **hæmorrhage** are frequent, and epistaxis, hæmatemesis, melæna, purpura, hæmoptysis, or hæmorrhage from the pleura and peritoneum, may be seen. Hæmorrhage appears at all periods of the disease; it may, indeed, occur as a symptom of the **onset**. **Gastro-intestinal hæmorrhage** is the most terrible; the hæmatemesis may be fulminant. I had in hospital a cirrhotic patient who succumbed in a quarter of an hour from hæmatemesis, caused by rupture of the œsophageal varices. In other patients hæmatemesis occurs several times a day and on several successive days; it then ceases, or reappears some days or weeks later. I had in my wards a cirrhotic patient in whom ascites and copious hæmatemesis were the two principal symptoms. Immediately after evacuation of the ascitic liquid the hæmatemesis ceased; the fluid did not return, and the patient was able to leave the hospital.

**Nævi** are frequent in cirrhotic patients. I have seen in one of my patients at the Necker Hospital obstinate hæmorrhage, caused by an ulcerated nævus at the angle of the nostril. I had in the Hôtel-Dieu a cirrhotic patient, suffering with hæmorrhage from the larynx, as proved

by laryngoscopic examination. Another case of this nature has been published by Lubet-Barbon.

What are these hæmorrhages in cirrhotic patients due to? According to certain authors, the obstacle which the blood meets in the liver causes stasis of the circulation in the stomach or intestine, and hæmorrhage. It is certain that the mechanical influence of the obstruction in the portal system cannot fail to be recognized, for the hæmorrhagic erosions of the mucosa of the stomach, and the varices of the œsophagus and of the stomach are evidence to this effect. But this mechanical cause seems to me to play a part only in the pathogenesis of hæmorrhage in atrophic cirrhosis.

Hæmorrhage may appear so soon after the commencement of the disease that mechanical stasis of the blood cannot be the cause. Certain patients are seized with hæmatemesis or melæna before any other symptom, and as a **precursory sign** at an epoch when as yet neither collateral circulation, nor ascites, nor any of the usual indications of obstruction in the hepatic circulation are present; writers, in order to explain these hæmorrhages, invoke a blood dyscrasia due to the alteration in the hepatic functions. This dyscrasia, which is most marked in icterus gravis, and which cannot be denied in cases of advanced cirrhosis, seems to me to be scarcely admissible at the very commencement of the hepatic lesion. Other causes must therefore be sought, and the question may be put as to whether the radicles of the portal system—*i.e.*, the peripheral venules of the stomach and of the intestine—may not from the onset of the disease be the seat of changes that cause friability of these vessels.

**Icterus** is not a symptom of atrophic cirrhosis, and when it appears it is usually slight, and due to urobilin. Some patients with atrophic cirrhosis, after swallowing 5 ounces of syrup of sugar, suffer from **temporary glycosuria**. The sugar, absorbed in the intestine, passes into the general circulation and into the urine, without having been arrested in the liver. This experimental glycosuria is due either to obstruction in the portal system or to changes in the hepatic cells. Lowering of the quantity of urea is another sign of hepatic insufficiency. Hanot has noticed the existence of abdominal **lymphatic varices**.

**Course—Termination—Duration.**—In its typical form atrophic cirrhosis is an **apyretic** and **chronic** disease. In certain cases it runs its course without acute symptoms, and surprise is expressed when atrophic cirrhosis is discovered in patients who have died of some other disease. It usually lasts one or two years, but it may be checked by efficient treatment. Sometimes the patient dies of cachexia; at other times the end is hastened by hepatic adenoma, by complications, or by some intercurrent disease, such as peritonitis, pneumonia, erysipelas, infective endocarditis, suppurative cholecystitis, abscess of the liver, nephritis, phlebitis, hæmorrhage, or



tuberculosis affecting the liver, peritoneum, etc. (Rendu). Some patients succumb with profuse diarrhœa, algid condition, coma, and secondary infection due to the coli bacillus (Hanot). Finally, symptoms of **icterus gravis** may appear, and prove rapidly fatal. In the section on Icterus Gravis we shall see that the liver, already unable to defend itself, is invaded by the toxines and infections which produce secondary icterus gravis.

Atrophic cirrhosis is not absolutely fatal. In some cases it may be cured, but these fortunate cases are chiefly met with in the hypertrophic form of alcoholic cirrhosis, which I shall describe in the following section.

**Acute Atrophic Cirrhosis.**—Atrophic cirrhosis has not always the common and chronic form just described. Hanot has published four cases in which the cirrhosis ran a febrile and rapid course, lasting from two to six months. From the commencement, says Hanot, more or less sharp pains are noticed in the right hypochondrium. Ascites may appear before any abnormal development of the subcutaneous abdominal veins. Considerable œdema of the lower and upper limbs is soon seen. Jaundice, hæmorrhage from the mucosæ, and subcutaneous hæmatomata, although not as severe as in acute yellow atrophy, are more frequent and more pronounced than in ordinary atrophic cirrhosis. Death supervenes in the midst of the symptom-complex, called **acholia**.

Post mortem, the liver is found to be more or less atrophied, granular, hard, and consequently comparable with the liver in common atrophic cirrhosis. Histological examination, however, reveals essential differences. These differences are not seen in the distribution of the fibrous tissue, for it is **systematically** distributed as in ordinary cirrhosis. The differences are due to the lesions in the hepatic cells. In common cirrhosis the change in the protoplasm of the cells is slow, late, and incomplete, but in acute cirrhosis the cellular degeneration is rapid, general, and complete. A large number of cells are converted into fat globules, and it might be said that yellow atrophy has complicated cirrhosis. Moreover, as the important functions of the liver are rapidly abolished, the clinical picture of icterus gravis and of acholia supervenes. This proves once more that “in cases of cirrhosis, from the anatomo-pathological point of view, the diagnosis depends on the topography of the connective neo-formation, and the prognosis on the condition of the hepatic cell” (Hanot).

**Diagnosis.**—At first the diagnosis of atrophic cirrhosis of the liver is hardly possible through lack of signs. Later it is more easy, on account of the appearance of ascites, and of the complementary circulation, but yet it still presents some difficulties. Thus, **chronic tubercular peritonitis** has symptoms in common with cirrhosis—viz., digestive troubles, progressive loss of flesh, meteorism, and ascites; and as it is not always possible to prove atrophy of the liver, on the one hand, and, on the other hand, lesions of the

peritoneum, the data for a diagnosis must be sought elsewhere. In chronic tubercular peritonitis the fluid is less abundant and less easy to displace than in cirrhosis. The flanks are less spread out, the periumbilical region is less prominent, and the sensation of a fluid wave is less clear. The complementary circulation, which is only an initial stage in peritonitis, is often well developed in cirrhosis. Hæmorrhage, hypertrophy of the spleen, and jaundice belong rather to cirrhosis. Finally, the subject suffering from abdominal tuberculosis is, as a rule, affected with **pulmonary tuberculosis**.

The chronic peritonitis of **alcoholics** has also several signs in common with cirrhosis of the liver (ascites, dyspeptic troubles). The error is sometimes the more difficult to avoid, in that cirrhotic patients are most frequently alcoholics.

**Cancer of the peritoneum** may be accompanied by ascites and collateral circulation, but these symptoms are much less marked than in cirrhosis. Palpation reveals nodules or indurations, and the fluid, withdrawn by tapping, often has a hæmorrhagic tint.

The early appearance or the severity of **hæmatemesis** and of **melæna** is sometimes a cause of error. These symptoms are too frequently attributed to ulcers or cancerous lesions in the stomach and intestine, when they are really the precursors of hepatic cirrhosis. The same remark applies to **œdema** of the lower limbs, which occurs early, and must not be attributed to a cardiac lesion.

**Pylephlebitis** (inflammation of the portal vein) causes much ascites and well-marked collateral circulation, but the **acute course** of pylephlebitis and the accompanying jaundice are sufficient to clear up the diagnosis.

Finally, we must distinguish true atrophic cirrhosis from fibrous hepatitis associated with diseases of the kidneys and heart.

**Ætiology.**—Atrophic cirrhosis is a disease of adult age. It is frequent in France, and still more common amongst the people of the Northern countries, especially England. As regards its causes, one stands out prominently—viz., alcoholism in all its forms; and the disease is also called alcoholic cirrhosis. The mechanism of the lesion is readily understood, the poison being carried to the liver by the portal veins.

Alcoholism is sometimes produced by wine, and the patient, prior to the appearance of cirrhosis, has morning vomiting, trembling of the hands, and nightmare. At other times alcoholism is caused by spirituous liquors rich in aromatic essences. The two causes are often joined, and the better the individual stands drink, the more he takes, and the more surely will he suffer from cirrhosis. Children may suffer from alcoholic cirrhosis.

Numerous attempts have been made to produce alcoholic cirrhosis in animals, but the results obtained are not constant—a further proof that it is often difficult to draw conclusions from animals and apply them to man-



kind. Sabourin saturated guinea-pigs with increasing doses of alcohol for several weeks, and on examining the liver, found capillary phlebitis around the hepatic veins and centro-lobular steatosis. The results obtained by Strauss and Blocq are different. Prolonged alcoholic intoxication was produced in rabbits. The histological examination of the liver in these animals showed a systematic portal cirrhosis, though **no trace** of hepatic central cirrhosis was found. Laffite, in his experiments on rabbits, found, after examination of the liver, cellular and not vascular, lesions.

**Cirrhosis from Gastro-intestinal Auto-infection.**—Alcoholism has been assigned too much importance in the pathogenesis of cirrhosis. While alcohol produces fibrous changes, it is far from being the only cause. Many patients are not alcoholic, and we must therefore search elsewhere for the cause of the cirrhosis. This cause appears to exist in the poisons present throughout the entire length of the gastro-intestinal tract. In some circumstances they become the source of auto-infection, reacting directly on the liver.

The poisons of the digestive tube are of multiple origin. Some come from food—putrid meat, pork, fish, or shell-fish, bad water, etc. Under the influence of the microbes in the alimentary canal, the gastro-intestinal ferments give rise to a series of poisons—butyric, acetic, valerianic, lactic, and oxalic acid, etc., without counting indol, skatol, phenol, and the toxines manufactured by the microbic agents, notably by the *coli bacillus*.

Boix has carried out experiments with all these acids on rabbits, taking care that the acids were ingested in small doses and over long periods. By ingestion of butyric acid he was able to induce Laënnec's atrophic cirrhosis; by the ingestion of lactic and of valerianic acid he was also able to cause cirrhotic lesions. The ingestion of acetic acid produced even more marked fibrous lesions in the liver. Analogous results have been obtained with living cultures and with the toxines of the *Bacillus coli*. To sum up: "The very interesting researches of Boix," says Hanot, "prove that the organic acids of digestion may produce hepatic cirrhosis, some more easily than others. In the normal state the liver resists these daily poisons. If it grows feeble, or if it is already weak *ab ovo*, the toxic action takes place, and hepatic cirrhosis by auto-infection of a gastro-intestinal origin supervenes."

It is evident that the question differs somewhat in man, and many factors must be considered, such as heredity, disorganization of the liver by anterior or concomitant infections, and the tendency, which is perhaps the result of arthritism, to make fibrous tissue. Here, as elsewhere, the pathogenic problem is often complex, and yet, logically and experimentally, we cannot deny that gastro-intestino-hepatic intoxication plays a very important part in the determination of cirrhosis.

While the gastro-intestino-hepatic process does not always end in confirmed cirrhosis, it often causes congestive cirrhotic lesions in the liver, of which the following are the principal varieties :

In a first variety the liver is rather congested than cirrhotic, being enlarged, and tender on pressure. The patient experiences a feeling of heaviness in the right hypochondrium, and has slight jaundice. The size of the liver increases or diminishes according as the patient passes through the phases of more or less marked dyspepsia. "In 389 personal observations of dilatation of the stomach," says Bouchard, "I have found that swelling of the liver is seen in the proportion of 23 per cent., and, in order to prejudge nothing, I have given to this change the name of **enlarged liver**." This is an example of the **enlarged dyspeptic liver** (Boix), the word "dyspepsia" signifying by abbreviation the change in the liver resulting from dyspeptic troubles.

In this variety of enlarged dyspeptic liver, in adults as well as in children, the liver suffers from congestion, and a long interval elapses before the appearance of cirrhosis.

The cirrhotic liver of intestinal auto-infection is enlarged, hard, and not painful. Jaundice, ascites, collateral circulation, and splenomegaly are not seen. The liver reaches some inches below the false ribs, and measures 8 to 10 inches in the nipple line. It has a smooth surface, is remarkably hard and wooden. This ligneous hypertrophy of the liver persists for years without causing grave signs, and the organ continues to fulfil its functions sufficiently well. Sometimes acute attacks are noticed, and are characterized by dyspeptic troubles, pains in the hypochondrium, and the appearance of urobilin in the urine. In this variety of cirrhosis the liver remains enlarged, and need not necessarily end in atrophy (in experimental cases the liver becomes atrophied). In these cases we cannot find alcoholism, malaria, syphilis, diabetes, tuberculosis, or, indeed, any cause to explain this cirrhosis. And yet we have certainly to deal with cirrhosis, for in one case it has been possible to make the histological examination of a cirrhotic liver due to intestinal auto-infection. The lesions of bivenous cirrhosis were present: fibrosis of the porto-biliary spaces invading the hepatic lobule, the central vein of the lobule, and the portal capillaries. In some instances the lumen of the portal vein had completely disappeared.

In consequence of the relations between the **appendix** and the liver (see the chapter on the Appendicular Liver), I am quite prepared to add appendicitis to the intestinal intoxications which may cause cirrhosis of the liver. Cirrhosis of the liver from lead-poisoning has also been described.

**Treatment.**—The prognosis of cirrhosis of the liver is not so fatal as was supposed a few years ago, provided that efficient treatment is employed. Local treatment must not be neglected—*i.e.*, dry-cupping, the



actual cautery or blisters applied over the hepatic region. Milk diet, iodide of potassium, and hydrotherapy, form the general treatment.

Personally, I know of no treatment so efficacious as **milk diet**: 5 pints of milk daily, and milk foods, for six months. I further discuss this treatment under Curability of Cirrhosis of the Liver. I prescribe iodide of potassium, **but in very small doses** (3 grains daily), without intermission for a year. It is evident that the chances of success are best if the disease is treated as early as possible.

What is the best treatment for ascites? If the effusion is abundant, and the diaphragm is pushed up so as to interfere with the normal function of the lungs, paracentesis becomes necessary. The best spot for **paracentesis** is the middle of a line joining the umbilicus and the antero-superior iliac spine. I recommend a **small-size** trocar, previously sterilized. When the puncture has been made, a tube is attached to the cannula of the trocar, so as to siphon off the liquid in a **slow and continuous** manner. This operation, though quite simple, is sometimes followed by complications, some immediate, such as syncope, and others more remote, such as erysipelas, starting in the wound, and extending as far as the peritoneum. These complications can always be avoided if paracentesis is performed with a trocar of average size and with proper aseptic precautions. I admit that with a large trocar the operation is finished quicker, but to the detriment of the patient, because the rapid flow of the liquid more readily causes syncope, the wound remains open, and secondary infection, caused through the want of care, spreads more readily from the puncture to the peritoneum. The operation concluded, the dressings are applied. The patient must remain on his back for several hours, or on the side opposite to the puncture, the object being to prevent oozing from the wound.

Evacuation of the fluid affords the patient great relief, but the fluid usually reforms after a few days or weeks. The operation is again performed as soon as may be necessary. In some cases, however, after two or three tappings, the ascites does not reappear, and, indeed, restoration to health may be possible. The question of the definite disappearance of ascites and of the curability of cirrhosis will be referred to later.

**Surgical** treatment of cirrhosis of the liver has been employed (Talma and Morrison's operation). It has been thought that a good result would be obtained if a means were found to deflect the circulation of the portal vein by removing the obstacle met with in the cirrhotic liver. This hypothesis, however, leaves something to be desired. Eck proposed to establish direct communication between the portal vein and the inferior vena cava. This operation has not been adopted.

Talma proposed as a remedy for the obstacles in the portal circulation the establishing of venous anastomoses between the splanchnic portal veins

and the veins in the abdominal wall. In order to do this he fixed the great omentum to the wall of the abdomen. Morrison tried to obtain widespread adhesion. The results of the operation have been disputed : some say that they have been favourable, others say that they have been bad. The fact is that the question has still to be settled.

## VI. HYPERTROPHIC ALCOHOLIC CIRRHOSIS—REGENERATION OF THE LIVER.

Hypertrophic alcoholic cirrhosis does not form a clearly defined morbid entity. Many intermediate forms are found between the atrophied cirrhotic organ and the hypertrophic one ; and yet I cannot see how the same factor (alcohol) can produce in one case the atrophic liver and the fatal cirrhosis of Laënnec, and in another case the enlarged liver of curable hypertrophic cirrhosis. I have, therefore, doubts as to the pathogenesis. Clinical medicine, however, teaches us that the enlarged alcoholic liver remains enlarged after the ascites is cured, and experiments teach us that the hypertoxic urine, which causes convulsions in the case of atrophic cirrhosis, is of normal toxicity in the case of hypertrophic cirrhosis. While the existence of initial hypertrophy in atrophic cirrhosis is not improbable, it may be considered as exceptional. When the liver is hypertrophied in alcoholic patients, suspected of cirrhosis, we generally see one of the following results : the organ either contracts to its normal size or remains enlarged. In the latter case the cirrhosis is hypertrophic (Hanot and Gilbert). The initial symptoms as well as the ætiology do not differ in the slightest from common cirrhosis. Later, in the stationary stage, hypertrophic, like atrophic, cirrhosis may be lacking in such cardinal symptoms as collateral circulation or ascites. It may be rich in symptoms. "Dyspeptic troubles, alteration in the colour of the fæces, physiological and chemical changes in the urine, the yellow tint of the skin, the appearance of varicose veins and of vascular nævi, hæmorrhages from diverse tracts (especially epistaxis and gastrorrhagia), ascites, dilatation of the subcutaneous abdominal veins, hæmorrhoids, splenomegaly, loss of strength and of flesh, belong to the hypertrophic as well as to the atrophic forms of alcoholic cirrhosis" (Hanot and Gilbert). The dilatation of the subcutaneous abdominal veins is sometimes slight or absent. It is only in exceptional cases that it shows great development.

The liver extends below the costal margin by the width of several fingers. It is firm ; its edge is rounded, and the surface may be studded with nodules. The diagnosis must be made from syphilitic, malarial, and fatty liver.

The recognition of hypertrophic alcoholic cirrhosis is especially interesting from the point of view of the **prognosis**. We know that in the curability



of cirrhosis has recently been under discussion and that two-thirds of the cases quoted as improved or cured belong to the form under description. The ascites diminishes; the subcutaneous and hæmorrhoidal vessels become small. The liver may contract, but as a rule incompletely. Millard has quoted a case in which the spleen contracted. It must be remembered that the curability may be only transient, and that ascites may reappear as a result of alcoholic excess. Finally, we see cases in which cirrhosis with an enlarged liver may cause death, after the manner of atrophic cirrhosis.

The **anatomical** characters are as follows: The weight of the liver may be 5 or 6 pounds; the edges are rounded, and the colour is of a rosy yellow. The surface, after removal of Glisson's capsule, presents uneven nodules, less numerous, however, than in atrophic cirrhosis. The surface of the section is smoother than in Laënnec's cirrhosis, but is divided into yellowish granules by reddish fibrous rings. "The cirrhotic rings occupy the whole of the portal spaces and the majority of the veins of the hepatic system." The sclerotic tissue is furrowed with capillary angiectases and newly-formed bile-ducts.

Hypertrophic alcoholic cirrhosis, like atrophic cirrhosis, is, then, an annular and perivenous fibrosis. Where are we to look for the cause of the enlargement of the liver? Perhaps in the numerous capillary angiectases which furrow the fibrous rings, or perhaps in the hypertrophy of the hepatic cells which tend to the concentric orientation seen in nodular hepatitis.

The first condition in treatment is the suppression of alcohol. Strict milk diet, continued for a long time, iodides in small doses, diuretics, and hydrotherapy, may bring about a cure.

**Regeneration of the Liver.**—Hypertrophic cirrhosis is of the same nature as atrophic cirrhosis. Both diseases present the same topography of the connective neo-formation. Both have the same ætiology, which often is alcoholism, and both have the same symptomatology (ascites, dilatation of the subcutaneous abdominal veins, splenomegaly, etc.), and their course alone is different. Hypertrophic cirrhosis, as proved by examples, has a natural tendency to recover. What is the cause of this relative benignity? Attempts have been made to explain it. Relying on experiments, and comparing the facts observed in certain affections of the liver, authors who have studied the question admit that hypertrophic alcoholic cirrhosis is an example of compensatory hyperplasia, and that we have to deal with a phenomenon of regeneration, recently made clear. It will be well to give some details of this view, which is destined, I think, to play a great part in the conception and the classification of cirrhosis.

Experiments prove that the liver possesses in a high degree the faculty of regeneration. Ponfick, van Merster, Ploeck, and Kretz, show that it is possible, without danger to the life of the animal (dog, rabbit, rat), to remove

half, or even three-quarters, of the liver. The animal regains its appetite, and gradually recovers. The remainder of the liver becomes double or treble its size in three or four weeks. At the end of twenty-six days, on an average, the hepatic tissue is regenerated to such an extent that the newly-formed tissue may weigh more than the normal liver. This regeneration results from the hypertrophy of the organ, and especially from the hypertrophy of the hepatic cells. The lobules attain a size three or four times larger than normal, and may be recognized with the naked eye. The cellular proliferation commences in the periphery of the lobule, and advances progressively towards the centre. This proliferation is regular, and the newly-formed parts retain the primary anatomical type. Histological examination proves that the process is one of true hyperplasia, and the percentages of urea seem well to justify the name of compensatory. After ablation of the liver, the quantity of urea is lowered. This diminution is proportional to the amount of liver removed. Some time after the operation (ten to fifteen days) the amount of urea rises, and reaches the normal figure.

These experimental results have been applied to human pathology. Hydatid cysts of the liver furnished the first example of regeneration (Durug, Hanot, Chauffard, Kahn). As a matter of fact, these parasitic affections almost realize the conditions of Ponfick's experiments. A portion of the hepatic parenchyma—most frequently the right lobe—is destroyed by the tumour. In all observed cases there was vicarious hypertrophy of the left lobe. In one of these cases this lobe alone weighed more than the entire normal liver. The increase in size of the lobules is apparent to the naked eye. Under the microscope the hepatic trabeculæ are hypertrophied and composed of cells larger than normal, and containing two, three, or even four nuclei, in which karyokinesis can be distinguished. It is a remarkable fact that the columns of the hepatic cells do not preserve their normal relation, and here, as in experimental cases, the lobule no longer exists. The rows in some cases are sinuous, and arranged without regular order; in other cases—and these are the more common—the rows have a tendency to the concentric arrangement observed in nodular hyperplasia.

This process of regeneration which exists in hydatid cysts of the liver, and which I have described in Hydatid Cysts of the Spleen, has been found to be well marked in hypertrophic cirrhosis. It supplies us with the key to the different evolution of the atrophic and hypertrophic forms of alcoholic cirrhosis. In the former most of the cells are destroyed or are on the way to fatty degeneration; in the latter, not only do the cells show no change, but we find in places foci of hyperplasia, cells with karyokinetic proliferation and concentric nodules, comparable in every way with those seen in the case of hydatid cysts. Here again the idea of compensation in hypertrophic



alcoholic cirrhosis is verified by clinical evidence. We have seen, as a matter of fact, that the urinary toxicity, instead of being increased, as in the atrophic form, remains normal. Furthermore, researches upon urobilinuria and alimentary glycosuria show the perfect condition of the cell. In alcoholic cirrhosis this regeneration may be diffuse, and give the microscopic appearance just described. The hypertrophy affects the liver as a whole, or is confined either to one lobe or to a part of a lobe. All these varieties of one and the same pathological condition differ in their microscopic aspect, but they are blended in the condition of the living and regenerated cell that is peculiar to them. The condition of the cell must serve to classify cirrhotoses, for it commands the evolution, while the prognosis is closely related to it. The proximate causes of this reaction by the organism are still obscure in certain cases. The regenerative process has been noticed particularly in young arthritic individuals. We shall meet with this compensatory hyperplasia when we study other kinds of cirrhosis, such as hypertrophic biliary cirrhosis, in which, as far as Hanot is concerned, it explains the course of events. It manifests itself especially by the formation of concentric nodules. "Similarly, it is admissible that the lesion described by Kelsch and Kiener and by Sabourin under the name of hyperplastic nodular hepatitis in certain cases of malarial and tubercular cirrhosis with enlargement of the liver, is only the expression of a process of more or less developed, though often insufficient, regeneration."

## VII. CURABILITY OF CIRRHOTIC ASCITES AND CIRRHOSIS.

Cirrhosis and cirrhotic ascites were considered to be incurable, and every individual with cirrhosis of the liver was doomed in almost the same manner as an individual suffering from cancer. These opinions must be modified. In 1886 Troisier made an interesting communication to the Société Médicale des Hôpitaux on the curability of cirrhotic ascites, and mentioned with regard to this subject the work of Lendet and Ribeton. The following examples give an idea of this process :

Troisier : A man was admitted to hospital for ascites, œdema of the lower limbs, and collateral circulation. The quantity of fluid in the peritoneum was estimated at about 12 pints. The liver seemed to be enlarged, and the spleen was hypertrophied. The diagnosis pointed to alcoholic cirrhosis, and absolute milk diet was prescribed. Under the influence of this treatment the urine rose to 5 pints, the ascites disappeared in a few weeks, as did the œdema of the lower limbs. Since then the ascites has not returned, and the patient is in excellent health.

Descoust : A patient was suffering from considerable ascites, with œdema of the legs, and jaundice. The urine was scanty, but did not contain albumin ; the heart was healthy. The man had for a long time been addicted to drink, and the diagnosis of cirrhosis with ascites was made. Ascites, œdema of the lower limbs, and jaundice had appeared four years before, but had disappeared as long as the patient abstained from

alcohol. Vulpian, who saw the patient at the time of his second attack, gave a very grave prognosis. Soon after this consultation the patient left for the country. No news was received from him, and it was thought that he was dead. Great was the surprise of Descoust to see him walk into his consulting-room, completely recovered.

Séailles : A blacksmith, of alcoholic habits, was taken ill with ascites and œdema of the legs. The liver was enlarged and extended beyond the false ribs by 2 inches. The case was one of alcoholic cirrhosis, with enlarged liver. The abdomen was tapped, and 18 pints of a citrine liquid were removed. The reproduction of the liquid was so rapid that, between January 28, 1885, and November 5 of the same year, eighteen tapplings were necessary, and gave a total of 360 pints. Each tapping was followed by relief, but the cachectic condition became more pronounced and the patient began to waste. From the commencement of treatment absolute milk diet had been prescribed. After the eighteenth tapping the ascites again recurred ; and then the urine became abundant, the circumference of the abdomen fell from 45 to 40 inches, and the ascites disappeared, although the liver remained hypertrophied. In a few weeks he regained his health, and went back to his trade as a blacksmith ; his appetite was good, but, as regards drink, he replaced wine by milk.

Millard : This was a case of a man of alcoholic habits. After a prodromal phase, lasting a year and a half (diarrhœa, tympanites, yellow complexion, and emaciation), œdema of the lower limbs, and soon afterwards ascites, appeared. A doctor tapped him, and drew off 16 pints of clear fluid. The effusion rapidly reappeared. Millard found considerable ascites ; the liver was enlarged, and the spleen was hypertrophied. An exclusive milk diet was prescribed. From that time the ascites diminished progressively, and the liver and spleen decreased in size. The improvement was maintained, and health was regained in a few months. The ascites completely disappeared, the liver and spleen resumed their normal size, and the man was cured of ascites and of alcoholic cirrhosis.

Kahn : A woman, of alcoholic habits, noticed a gradual enlargement of the abdomen. Soon afterwards she suffered from slight jaundice, œdema of the lower limbs, and difficulty in breathing. She went into Hanot's ward. Considerable ascites and a well-marked collateral circulation were found ; the lower limbs were œdematous ; the face was œdematous ; the stools were almost clay-coloured ; and she experienced dyspnœa on the least exertion. The urine contained neither albumin nor sugar, though urobilin and red pigment were found. Paracentesis was performed, and 30 pints of yellowish fluid were removed. The patient was placed on a strict milk diet. After tapping, the size of the liver was more easily appreciated, the organ being much enlarged. The diagnosis of hypertrophic alcoholic cirrhosis was made. The liquid was rapidly reproduced, and a second tapping was necessary in three weeks' time, 34 pints being withdrawn. Other tapplings were performed on the following dates : April 3, 42 pints ; April 20, 41 pints ; May 6, 41 pints ; May 25, 40 pints ; June 26, 37 pints ; August 16, 34 pints ; October 10, ninth and last tapping, withdrawal of 36 pints. From this date the ascites did not return, the liver gradually diminished in size, the general condition became excellent, and the patient finally recovered. The salient feature is that she was seen three years later in good health.

At the Hôtel-Dieu I had two patients whose history I have given in a clinical lecture\* on "The Curability of Ascites and Cirrhosis." *Résumé* : A woman was admitted into my wards for ascites estimated at about 24 pints. The patient told us that the trouble began seven months before, in August, 1897. She noticed that it caused her pain to put on her corset, and that her stomach was more swollen than usual. As regards digestive troubles, she had occasional vomiting. In November slight œdema of the lower limbs made its appearance, and quickly increased, so as to make walking

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\* *Clinique Médicale de l'Hôtel-Dieu, 1897, 19<sup>me</sup> leçon.*



impossible. Micturition became difficult. She was admitted in this condition to a surgical ward on February 2, 1898, the diagnosis being ascites, following a cystic lesion of the ovary.

On February 5 an incision was made in the median line below the umbilicus, and about 30 pints of serous liquid withdrawn. The ovaries were examined, but were healthy, so that the ascites was not due to an ovarian lesion. The peritoneum was carefully examined, but was healthy, so that the ascitic form of tuberculosis was out of the question. The diagnosis of cirrhotic ascites was, therefore, made, and, as soon as the abdominal wound had healed, the patient was sent over to the medical wards, under Chauffard. For some time after the evacuation of the fluid the patient felt better, and breathed more freely, and the legs were less œdematous. She was placed on a milk diet, and oxymel scillæ was prescribed. On February 15 (ten days after laparotomy) ascites was again present; the œdema of the legs had increased, and the urine was less abundant. On March 2, 27 pints of yellowish liquid were withdrawn. The œdema of the legs diminished rapidly and the patient left the Cochin Hospital on March 6 for home. To her great despair, the œdema reappeared, and the belly again swelled, so that she was compelled to keep to her bed.

On March 15 she was admitted to my wards, complaining, as before, of the size of her belly, of œdema of the legs, and of difficulty in micturition. On examination I found the recent cicatrix of the median laparotomy. The ascitic liquid was very abundant and free in the peritoneal cavity. The size of the belly and the displacement of the intestinal coils made the measurement of the liver impossible. Palpation revealed neither tumour nor induration. A well-marked collateral circulation was evident in the right flank and the epigastrium. The urine contained neither albumin, sugar, nor urobilin; examination for alimentary glycosuria remained negative. The existence of hæmorrhoids was noticed.

The previous operation simplified the diagnosis greatly, and I knew that neither cystic degeneration nor peritoneal tuberculosis had been found, so that I could only agree with the previous diagnosis of cirrhotic ascites. Moreover, the patient had "a hepatic past," which must always be taken into account. A few years previously she had had jaundice. Again, she had managed a wine-shop, which in itself is an invitation to drink. My diagnosis was, therefore, profuse ascites, hepatic cirrhosis, relative integrity of the hepatic cells; bladder troubles caused by the amount of the ascites. The patient was placed on a strict milk diet. Iodide of potassium was given only for a short time.

On March 27 tapping became necessary. Thirty pints of albuminous yellow liquid were withdrawn; the lower edge of the liver was then found to be smooth. Percussion gave a vertical dullness of 5 inches. The cirrhosis was therefore not atrophic, but had, rather, a hypertrophic tendency. During the next few weeks the ascites slowly reappeared, and the œdema of the legs persisted. On May 16 a fresh puncture was made, and 26 pints of yellowish liquid were withdrawn. The patient continued her milk diet, and felt much better. During the next few months the situation completely changed: the œdema of the legs became insignificant, and the increase in quantity of the peritoneal fluid was so slow that in two months and a half the belly contained only a few pints. Nevertheless, on August 2 I tapped again, 15 pints of fluid being withdrawn. From that date not the slightest peritoneal effusion was noticed; the œdema of the legs disappeared completely, and the woman felt in perfect health; but I kept her in bed on a milk diet. On October 15 I allowed the patient to get up, and I added eggs and vegetables to her milk diet. She left the hospital on November 1, promising to continue her diet. She came back to see us on several occasions, and we found her in the best of health. She ate almost all kinds of food, but she drank only milk. There has not been a trace of œdema in the legs, the ascites has never reappeared, and the liver has gone back to its normal size. Summary: In this case of ascitic cirrhosis

134 pints of fluid had been withdrawn in six months. After the last tapping the cure remained permanent.

I saw a similar case at the Hôtel-Dieu. A wine-broker was admitted into my wards for ascites. He told me that the belly had commenced to swell three months previously, and that it became enormous in six weeks. The appearance of the ascites was preceded by precirrhotic symptoms—loss of appetite, irregularity of the bowels, appearance of hæmorrhoids, tympanites, epistaxis, wasting, and loss of strength. On examination I found that he had an earthy complexion, and that his conjunctivæ were yellowish. The belly was much enlarged, and measured 50 inches around the umbilicus, which was very prominent. The flanks were bulging, and the skin was covered with dilated veins, indicating a collateral circulation. Dullness was complete in the flanks and hypogastrium, and the resonance only appeared to start from the umbilicus. The sensation of fluid was very clear. I estimated the quantity of fluid in the peritoneum at about 20 pints. In spite of the distension of the abdomen the enlargement of the liver was perceptible; on dipping it was found to extend an inch below the false ribs. The hypertrophy was likewise shown by the vertical dullness, measuring 6 inches. The spleen was enlarged. The urine was of normal quantity, and contained urobilin in small amount. The legs were not cedematous. The heart was normal.

The man was an alcoholic, drinking some 12 pints of wine a day. He had followed the wine trade for twenty-five years. Chronic alcoholism showed itself by such symptoms as dyspeptic troubles, nightmare, pains and cramps in the legs, and tremors of the hands and tongue. The diagnosis of ascites, associated with hypertrophic alcoholic cirrhosis, was obvious. The large size of the liver was not antagonistic to this diagnosis; on the contrary, an enlarged liver is frequently characteristic of alcoholic cirrhosis.

In spite of the ascites, I postponed tapping, and ordered complete rest and milk diet, to which a tablespoonful of Trousseau's diuretic wine was added daily. The urine became more abundant, and the general condition improved. After a month's treatment the belly was more supple, and grew smaller. Strict treatment was continued, and gradually cured the ascites and the cirrhosis. Whilst the size of the belly diminished, the urine, which amounted to 3 pints daily, no longer contained urobilin.

After the disappearance of the fluid the hypertrophied liver could be more easily felt. Improvement ended in cure, and when the patient left the hospital, the ascites had completely disappeared, and the general condition was excellent. His food consisted of white meats, bread, and vegetables; the wine was cut off, and replaced by 4 pints of milk. I saw the patient after he had left the hospital: the liver had regained its normal size; the cure was maintained.

These cases prove that cirrhotic ascites is curable. Recovery has occurred after five tapplings (my first patient), after eighteen tapplings (Séailles), after nine tapplings (Kahn), and one tapping (Millard), and cure sometimes results without tapping (Troisier's case and my second patient). This cure of ascites is not spontaneous, but is due to an exclusive milk diet, to diuretic medicines, and to the absolute suppression of alcohol. Two factors contribute to the cure of ascites—diuresis, on the one hand, and, on the other hand, total suppression of alcohol.

Cirrhotic ascites is curable. Is cirrhosis of the liver also capable of cure? Cirrhosis of the liver may be cured. I am of opinion that the more it resembles the type described by Hanot and Gilbert as "alcoholic and hypertrophic cirrhosis" (the type described in the preceding section), the



more easily will it be curable ; and the farther it is removed from this type, the less curable it will be. Let me explain : In cirrhosis with an atrophic tendency the hepatic lobules undergo such destruction that the disease is incurable ; but there are cases of alcoholic cirrhosis with compensatory hyperplasia. By this hypertrophy the losses sustained through the cirrhotic process are more than compensated by the new formation of liver substance.

This fact was proved in one of my patients who recovered from cirrhosis and ascites, finally dying of erysipelas. The examination of his liver was of exceptional interest. The liver weighed over 4 pounds. The histological examination showed the existence of bivenous cirrhosis ; the portal lesions were of especial interest. The intrahepatic capillaries were enormously developed, both in the fibrous tissue and in the lobules, the portal circulation being completely re-established.

Furthermore, the numerous centres of cellular regeneration found explained the disappearance of the signs of hepatic insufficiency. The remedy, therefore, exists side by side with the disease ; the regenerative process supplants the degeneration, and the liver, instead of becoming atrophied, may finally be much larger than normal. The curability of ascites and of hepatic cirrhosis by suitable treatment is more likely when the cirrhosis approaches the type of alcoholic cirrhosis with an enlarged liver. Clinically this type differs slightly from Laënnec's cirrhosis. The course is less rapid ; there is less cachexia, and the liver is enlarged.

If we refer to the preceding cases that prove the curability of ascites and of cirrhosis, we see that hypertrophy of the liver was present. In the case of Troisier's patient the liver seemed to be increased in size. In one of Letulle's patients the liver was very much enlarged, and on percussion showed dullness of 6 inches in the nipple line. In Séailles's patient the liver was larger than normal, and reached below the false ribs. In Millard's three cases the liver was very much enlarged, and in two of my patients it was increased in size. The curability refers not only to the ascites, but also to the cirrhosis, provided the treatment is continued for a long time. Nevertheless, I repeat, all kinds of cirrhosis are not apt to be cured, but recovery is the more probable, the more the cirrhosis approaches the hypertrophic alcoholic type.

The **treatment** is very simple. It consists in suppressing alcohol in any form (Millard). The patient must be placed on **absolute milk diet** and water (Vittel or Évian water), with or without lactose. Rest in bed is essential. If the ascites is considerable, the fluid must be removed, and if it reforms rapidly, tapping must be done as often as necessary. Improvement and cure may sometimes be obtained. On the other hand, recovery is sometimes slow : after the fifth tapping in my first patient ; after the ninth tapping in Hanot's patient ; or after the eighteenth tapping in Séailles's patient.

## VIII. HYPERTROPHIC CIRRHOSES.

**Hypertrophic biliary cirrhosis** does not sum up the entire history of hypertrophic cirrheses. Besides hypertrophic biliary cirrhosis there are other varieties of hypertrophied liver, which I shall now mention.

1. First of all, we find malarial hypertrophic cirrhosis, described by Kelsch and Kiener, who found in the liver of patients dying from pernicious fever "that not only the portal vessels and the capillaries of the islets contained white pigmented corpuscles, but that the endothelial cells of these vessels contained also black pigment. In patients who died from intermittent fevers of long duration or from malarial cachexia these authors have seen different forms of cirrhosis, generally with **hypertrophy** of the liver, the lobules being affected by nodular parenchymatous hepatitis (regeneration of the liver) with adenomatous growths, as well as with more or less marked pigmentation of the new connective tissue and of the hepatic cells" (Cornil and Ranvier).

2. There is a hypertrophic alcoholic bivenous cirrhosis, described in one of the preceding sections.

3. There is a bivenous hypertrophic cirrhosis due to auto-intoxication of gastro-intestinal origin.

4. Fatty degeneration of the liver, with increase in size of the organ, is sometimes accompanied by a cirrhotic lesion, which allows us to describe a fatty hypertrophic cirrhosis. This variety is especially seen in alcoholics, or in patients who are alcoholic and tubercular at the same time. This variety will be described under Tuberculosis of the Liver.

5. Amyloid degeneration of the liver, with or without previous syphilis, will be discussed later. It is sometimes associated with cirrhosis, which may be hypertrophic or atrophic.

6. We find in some patients with diabetes cirrhosis which is almost always hypertrophic, and presents special characters. It will be dealt with in the next section.

## IX. PIGMENTARY HYPERTROPHIC CIRRHOSIS—PIGMENTARY CACHEXIA.

The disease, with which I am going to deal in this section, has received diverse names. It has been called pigmentary hypertrophic cirrhosis, which is not absolutely true, because the pigmented liver may not be hypertrophied. It has also been called bronzed diabetes, which does not answer to the sum-total of the cases, because diabetes and bronzed skin may be absent in pigmentary cirrhosis. The more vague name of "pigmentary cachexia" would be more in accordance with the actual facts.

I had in my wards a patient suffering from this pigmentary cachexia. On admission I was struck with the extreme pallor of his face, while the skin of the forehead,



the orbits, and the cheeks were of a bistre colour, somewhat resembling the bronzed tint of Addison's disease. He could hardly stand erect, because he was so weak. I examined him, and was confronted by a most difficult diagnosis. On the one hand the patient was covered with large purpuric ecchymoses, and on the other the liver was enormous. The urine contained neither sugar nor albumin. The analysis of the blood showed that there was no increase in the number of the leucocytes. The clotting was normal. The enormous liver and the bistre tint of a portion of the face rather pointed to bronzed hypertrophic cirrhosis, and I made this diagnosis with reserve. It was, however, correct. A few days later the patient died.

The post-mortem examination revealed some interesting features. The liver was enlarged, but it was heavier in proportion, because it weighed 110 ounces, instead of 50 ounces. It was hard and creaked under the scalpel, even more than an ordinary cirrhotic liver. It seemed as though the knife were crushing grains of sand. Finally, and this was the particular point which arrested my attention, the liver had a special tint; **it was the colour of rust.** Granules of cirrhosis were observed in the sections, which were of a rusty colour. These granules and the intermediary tissue were coloured with two different tints of rust. These macroscopic characters already showed that the liver was affected with the curious lesion called "pigmentary cirrhosis."

The microscopic and chemical examinations fully confirmed the diagnosis of pigmentary cirrhosis. The sections showed large bands of fibrous tissue, forming irregular rings, dividing the lobules and surrounding the hepatic veins, as well as the portal spaces. In short, the lesion had the aspect of a bivenous cirrhosis. The hepatic lobules were let into the cirrhotic tissue, and in places the cells were affected with vesiculo-fatty degeneration. The characteristic feature of the lesion, however, was the presence of irregular polyspherical granules of a golden-yellow colour in the bands of the fibrous tissue; in the interior of the hepatic cells granules of the same colour, though much smaller, were found. In the cells they were almost regularly divided, but they formed tracts in the fibrous bands, and were united into compact masses in some places.

These granules had very peculiar micro-chemical characteristics, and in spite of staining with many reagents **they preserved their yellow colour** in the midst of tissues coloured pink, red, or blue. Even in very thin sections, cleared with balsam, they appeared bright yellow, while the tissues were so transparent as to be almost invisible. Acids or alkalis did not affect the yellow colour. They were, therefore, rebellious to all colouring and decolouring organic materials. On the other hand, with sulphide of ammonia they assumed the black tint of sulphide of iron, and with ferrocyanide of potassium and hydrochloric acid they gave the beautiful blue colour of ferrocyanide of iron (Prussian blue). The granules were, therefore, formed of an iron pigment.

Summary: The liver was affected with hypertrophic cirrhosis, and was infiltrated with pigment, giving the reactions of iron. There was, therefore, no doubt that it was a case of the pigment called **ochre pigment** and of the lesion known as **pigmentary hypertrophic cirrhosis.**

The lesion and the disease that it causes have for some years been the object of much investigation that tends to modify former conceptions. A recent discussion at the Société Médicale des Hôpitaux showed great difference of opinion with regard to this subject. In this disease it is the rule that the liver is not the only organ infiltrated with the pigment; the pancreas, the salivary glands, the suprarenal capsules, the myocardium, and the lymphatic glands corresponding to the diseased organs are often infiltrated with pigment. The more active the cells the more apt are they to be loaded with iron pigment.

**Description.**—This disease was first described by Trousseau. In a man with diabetes brought on by overwork and privation, Trousseau was struck with the bronze-like colour of the face and the blackish colour of

the penis. "At the post-mortem examination," says Trousseau, "we found no lesion in the suprarenal capsules. The liver, however, was twice its normal size, and the whole surface of the organ was granular, of a uniform yellowish-grey colour, and of considerable density. It resisted pressure, and did not break down between the fingers. It creaked under the scalpel, and the cut surface, instead of being smooth, was granular. There was evident cirrhosis, but it was hypertrophic."

Hanot and Chauffard thus sum up the symptomatology of the disease: (1) Signs of diabetes (glycosuria, polyuria, polyphagia, polydipsia, impotence, boils, gingivitis, etc.); (2) signs of cirrhosis (ascites, collateral circulation, digestive troubles, increase in size of the liver, urobilinuria, jaundice, hæmorrhage, etc.); (3) bronzed colour of the skin, especially of the face, the genital organs, and the flexures of the limbs. The pigmentary patches are rare in the mucosæ, unlike the pigmentation in Addison's disease.

Pigmentary hypertrophic cirrhosis is hardly ever seen, except in feeble patients suffering from cachexia, tuberculosis, alcoholism, malaria, cancer, etc. In those individuals who are already diseased the signs of cirrhosis and the bronzed tint develop insidiously, and the examination of the urine reveals the presence of glucose. The loss of flesh is rapid, diarrhœa appears, and fever is frequent, the temperature varying from 98° to 100° F. The patient becomes cachectic, and death generally supervenes a few months after the appearance of the bronzed tint and of the diabetes. The fatal termination is frequently hastened by such infectious complications as moist gangrene, extensive erysipelas, pneumonia, broncho-pneumonia, or miliary tuberculosis.

Such is the common clinical picture. Nevertheless, all the symptoms are not constant. Thus, diabetes may be absent, as in the case of my patient; the bronzed tint of the skin may likewise be wanting. The name of "bronzed diabetes" is not, therefore, applicable to all cases of pigmentary cachexia. Hypertrophy of the liver is not constant, and the organ may be atrophied. The liver may, indeed, be infiltrated with ochre pigment, while the cirrhosis is very slight. Ascites is absent, and no collateral circulation develops. Accordingly, masked forms are seen, and bronzed cirrhosis is often but a chance find at the autopsy. If care were taken to submit all livers to the sulphide of ammonia and the ferrocyanide tests, it would be found that pigment giving the reactions of iron was relatively frequent.

**Pathogenesis.**—The first writers on this subject believed that bronzed cirrhosis was always the consequence of diabetes, and explained the formation of the pigment by the defective action of the liver in diabetes. In Marie's opinion, the diabetes is secondary, and the primary phenomenon is the change in the blood-corpuscles and the conversion of their hæmoglobin into iron pigment. This pigment accumulates afterwards in the



parenchymata; in the liver it produces cirrhosis; in the pancreas the changes induce diabetes. Ausscher and Lopicque have shown experimentally that, if a dog's blood is injected in the peritoneum of another dog, and the animal is killed a few days later, the liver will be found infiltrated with iron pigment. I was able to lay hold of the formation of this pigment at the expense of the hæmoglobin in the following case: A woman in one of my wards died during an attack of hæmoglobinuria, and Widal found at the post-mortem examination iron pigment in the cells of the convoluted tubules.

In my case of pigmentary cirrhosis, diabetes, alcoholism, malaria, and tuberculosis were absent, but the patient had had for some months recurrent purpura, with subcutaneous hæmorrhages. The ochre pigment was also seen around the hæmorrhagic foci, but in patients who are in good health it is quickly absorbed. And the same fact obtains in dogs (used for experiment—Ausscher and Lopicque) if several months are allowed to elapse before the animal is killed. It might be said that previous visceral changes are essential if the pigment is to remain fixed in the organs. This explains the fact that the disease is hardly ever seen, except in individuals rendered cachectic by tuberculosis, alcoholism, cancer, or malaria. The treatment of pigmentary cachexia is purely symptomatic, for we know of no means capable of preventing the formation or of hastening the absorption of the iron pigment.

## X. HYPERTROPHIC BILIARY CIRRHOSIS—HANOT'S DISEASE.

We have seen in alcoholic cirrhosis that the morbid process first attacks the portal and hepatic veins, and then the hepatic cells. In hypertrophic biliary cirrhosis the process commences in the hepatic cells and the biliary canaliculi, the reaction of the vital elements preceding or accompanying the lesions of the connective tissue.

Every cirrhosis, however, in which the biliary apparatus is affected, does not deserve a place in the category of so-called **biliary** cirrhosis. Jaundice may appear in atrophic cirrhosis, with neo-formation of biliary canaliculi, and is an integral part of biliary cirrhosis due to gall-stones. Jaundice may appear in alcoholic, fatty, pigmentary, or malarial hypertrophic cirrhosis, proving that there are **several varieties of hypertrophic cirrhosis with jaundice**.

There is, however, a type of hypertrophic biliary cirrhosis, which was described by Hanot in 1876. "Chronic jaundice; considerable hypertrophy of the liver, often also of the spleen; absence of ascites and of collateral circulation in the abdominal wall; symptoms of icterus gravis, as the most common termination," are the cardinal features ascribed by Hanot to the disease that rightly bears his name. For my description I shall select a typical case of hypertrophic biliary cirrhosis.

**Anatomical Pathology—Autopsy.**—The size of the liver is considerable. The weight may exceed 100 ounces. The shape is not altered; the edge remains sharp, and the surface is smooth, or at most is but slightly granular or nodular—features very different from atrophic cirrhosis, in which the liver is atrophied and hobnailed.

Externally the hypertrophied liver is of an olive-grey colour, especially in the projecting parts. On section, it is firm and resistant, but it does not creak under the scalpel, as in atrophic cirrhosis. It is of a dark brown or greenish colour, according to the amount of bile present. The surface of the section is overrun by wide bands of connective tissue, and studded at intervals with projecting granulations, which cannot be enucleated, and are therefore very different from the granulations of the atrophic liver. There is no amyloid degeneration. Glisson's capsule is often inflamed, thickened, and adherent to the neighbouring organs (perihepatitis).

The bloodvessels and the large bile-ducts of the hilum are normal. The gall-bladder shows no lesion. It may be small, and contain only a little bile, but no calculi. The glands of the hilum are generally not swollen. The hypertrophy of the liver is due to several causes : to abundant connective tissue that is less fibrous and retractile than that of atrophic cirrhosis ; to the integrity of a large number of hepatic cells ; to the frequent hypertrophy of the cells ; and to the formation of biliary canaliculi.

**Histological Examination.**—1. *Portal Spaces.*—In specimens stained with picrocarmine under a low magnifying power the cirrhotic tissue appears in the form of patches, stars, or of irregular islets, whence the name “insular cirrhosis.” These islets are caused by the tracts which penetrate into the fissures between the hepatic lobules. The tracts incompletely surround an islet or a group of islets, and skirt the lobules, so that the latter assume the form of an indented oval. The connective bands generally penetrate the lobule, and bend back or terminate in a swollen end, without reaching as far as the central vein of the lobule.

The fibrous tissue is not dense ; it has few fibres and little elasticity. It has been compared with the neuroglia of disseminated sclerosis (Brissaud), and does not resemble the fibrous tissue of venous cirrhosis.

In the new connective tissue blocking the portal spaces sections of the biliary canals, portal veins, and capillaries are seen. The striking fact is the size and number of the biliary canals, of which several may be counted in each portal space. The number of embryonic cells, with which the canals are surrounded, shows that they are a centre of fibrous formation. They may be recognized by the thickness of their walls, by their large connective sheath, by their deep colour, and by their lumen, which is lined by one or two rows of deeply stained cubical cells. The thickness and the staining of the biliary canaliculi distinguish them from the portal veins, the walls of which, though thicker than in the normal condition, are only half as thick as the walls of the biliary canals. This is the essential difference from atrophic cirrhosis, which presents inverse lesions—that is to say, the portal veins are thicker and more deeply stained than the biliary canals. The portal veins, dilated and gorged with blood in atrophic cirrhosis, are often



empty in hypertrophic biliary cirrhosis. In a word, the dominant factor in hypertrophic biliary cirrhosis is angiocholitis and periangiocholitis. The dominating factor in atrophic cirrhosis is phlebitis and bivenous periphlebitis.

2. *Hepatic Lobules*.—We have just seen that the hepatic lobules are indented and furrowed by the connective tissue arising as islets in the portal spaces. Sometimes the intralobular connective tissue is a direct outgrowth of the perilobular tissue ; sometimes it appears to be independent of it ; but whatever source it may arise from, the cirrhosis is extra- and intralobular.

Several authors admit that intralobular cirrhosis arises, not at the expense of the connective tissue of the lobule, but at the expense of the epithelial elements (epithelial cirrhosis). The parenchyma of the cells is said to undergo change, and to give birth to new tissue (parenchymatous inflammation). The cirrhosis would then be both **interstitial** and **parenchymatous**, and would come under the heading of Epithelial Cirrhosis. Such is not the present-day opinion. "The doctrine of epithelial cirrhosis has had its day" (Letulle). At any rate, in hypertrophic biliary cirrhosis the central vein of the lobule is never cirrhotic. According as the lobule is more or less deformed, the topography of the hepatic lobule is modified, the hepatic vein loses its central position, and may even disappear. When the lobules are much involved, the cells are dissociated and deformed, but the degeneration and the atrophy of the cells so frequent in atrophic cirrhosis are exceptional in hypertrophic cirrhosis. One of the most prominent characters of this hypertrophic cirrhosis, says Hanot, is the integrity of most of the hepatic cells. In certain lobules, even those which are attacked by fibrosis, the cells preserve their columnar disposition, and are not crowded together, as in atrophic cirrhosis, but each cellular ray is separated from its neighbour by an empty space, and these spaces radiate in little columns. The cells preserve their normal size and shape, although some are hypertrophied. They stain with carmine, and are rarely infiltrated with fat or bile pigment.

3. *Biliary Network*.—The biliary canals visible to the naked eye undergo no notable modification. The process primarily attacks the canaliculi of the portal spaces, and causes angiocholitis. Furthermore, in the hyperplastic connective tissue we see a large number of much finer, elongated canaliculi, which ramify and form meshes by their anastomoses. These dilated canaliculi sometimes form biliary angiomata, which may become cystic (Sabourin). Small biliary abscesses have also been seen following suppurative angiocholitis. Some canaliculi are formed by a wall lined with cubical epithelium ; others have a flattened epithelium. In any case they are not pseudo-, but true, canaliculi, because the smallest of them has a

lumen. In some specimens they may be seen penetrating the hepatic lobules in the spaces between the little columns of cells.

The marked development of the biliary canaliculi is met with in acute just as in chronic hepatitis (Cornil), but in no case is it as marked as in hypertrophic cirrhosis. What is the origin of these biliary canaliculi? Are they completely moulded, or are they, while scarcely visible in the normal condition, produced by the inflammation of the tissues around them? This network of canaliculi is not found in a healthy liver, but it can be produced experimentally. In their experiments on biliary cirrhosis caused by the ligature of the common bile-duct, Charcot and Gombault found the development of a rich network of canaliculi with cubical epithelium, and in several specimens they were able to prove the union of the extralobular with the intralobular canaliculi. They thought that the intralobular canaliculi were but the transformation of those normally present in the lobule. How, then, is the presence of cubical epithelium in the new canaliculi to be explained? Kiener and Kelsch have answered this question, admitting that the intralobular biliary canaliculi and their cubical epithelium are formed at the expense of the hepatic cells, "the hepatic cells returning to the embryonic stage after having been previously the seat of nutritive irritation." This opinion was adopted by several authors (Charcot); the present tendency is to reject it. The progressive atrophy of the epithelia, says Letulle, already recognized by histologists, but incorrectly interpreted, served as a basis for the doctrine of the possible return of the epithelia to the embryonic condition; but well-observed cases have thrown it in the shade (Brault). "The frequency of biliary canaliculi in all hepatic affections, and their direct relations with the biliary canals of the portal spaces and with the intralobular canaliculi, would rather lead us to consider them as old canaliculi brought out either by the retreat of the hepatic lobules or by the inflammation of the connective tissue in the midst of which they are found.

4. *Bloodvessels*.—In describing the lesions of the portal spaces I said that phlebitis and periphlebitis may be met with, but the phlebitis is only slight, and does not form the principal lesion, as in atrophic cirrhosis. The integrity of the portal circulation assuring the function of the hepatic cell explains why hypertrophic cirrhosis may last for years without compromising life. Moreover, the newly-formed fibrous tissue contains a vascular network between the portal vessels and the capillaries of the affected lobule. This network is said to be not of new formation. "It would rather represent that portion of the hepatic capillary network in the meshes of which connective tissue has been substituted for the glandular cells" (Ackermann).

In any case, wide communications exist between the portal and central veins, and injections into the portal vein do not meet with the same obstruction as in atrophic cirrhosis.



5. *Process*.—I have discussed the different changes in hypertrophic biliary cirrhosis. But how does the process commence? It begins in the biliary canaliculi of the portal spaces (biliary cirrhosis), whereas the process in Laënnec's cirrhosis commences in the portal and the hepatic veins (bivenous cirrhosis). As the inflammatory signs are very marked in the immediate neighbourhood of the biliary canals in the portal spaces, Hanot has rightly supposed that the lesion is at first confined to these canals (angiocholitis and periangiocholitis). This inflammatory process is systematic, commencing in the interlobular ducts almost at the same time throughout the whole liver, and spreading in the manner already described.

Perhaps, too, the process commences in the hepatic cells, the hypertrophy and overactivity leading to a kind of biliary diabetes. This polycholia would then cause enlargement of the intralobular biliary canaliculi, stagnation of the bile in the extralobular canaliculi, and consecutive angiocholitis (Schachmann).

The theory that the process in hypertrophic cirrhosis commences in the inflammation of the biliary canaliculi is confirmed by the following facts: The persistent obliteration of the common bile-duct by a calculus or by cancer of the pancreas, and the experimental ligature of this canal in animals causes fibrous lesions, closely resembling those of hypertrophic cirrhosis. Evidently there are differences; thus, experimental biliary cirrhosis is not hypertrophic, or, at least, the hypertrophy is transient. Furthermore, it is accompanied by rapid change in the hepatic cells and by lesions of the large biliary canals, which is not the case in hypertrophic cirrhosis; but it allows us to witness, as it were, in the very act, the course and formation of the cirrhotic tissue, which commences in the biliary vessels, and then spreads to the connective tissue. This theory has been questioned on the ground that the formation of the biliary network which is so well marked in hypertrophic cirrhosis also exists in acute atrophy of the liver and in all forms of cirrhosis. This biliary network is found "in the midst of the pathological fibrous tissue of the gland, no matter what is the origin or the nature of the morbid process." This biliary network, however, though it has been found in several affections of the liver, in no wise diminishes the value of the initial localization of hypertrophic biliary cirrhosis in the canaliculi of the portal spaces.

6. The accessory lesions of hypertrophic cirrhosis are hypertrophy of the spleen, which is rarely absent. The spleen may weigh three or four times its normal weight. I would, further, mention perihepatitis and dilatation of the right side of the heart, with tricuspid insufficiency (Potain).

**Symptoms.**—Confirmed hypertrophic biliary cirrhosis is characterized by three essential symptoms: enlargement of the liver, persistent jaundice,

and hypertrophy of the spleen. The troubles of the onset are variable. Some cases begin with flatulence, eructations, loss of appetite, a feeling of discomfort in the epigastrium, and a sensation of weight in the right hypochondrium. The disease may begin with congestion of the liver, accompanied by fever. These congestive attacks are followed by jaundice, and simulate catarrhal icterus. When they are accompanied by pain, they simulate hepatic colic. These various modes of commencement are only met with in certain cases. As a rule, jaundice is the first symptom, fever, pain, and dyspeptic troubles being absent. No matter what is the early character of the disease, jaundice is the chief symptom, and must first demand our attention.

The jaundice is due to catarrh of the biliary canaliculi and to the resulting retention of the bile. It may be slight or severe, varying from a pale yellow to the olive tint generally seen at an advanced stage of the disease. It lasts for months and years without ever completely disappearing, because the catarrh of the bile-ducts is permanent. The retention of the bile causes the urine to be more or less charged with pigment.

As the catarrh only obliterates a portion of the bile-ducts, sufficient bile continues to flow into the intestine. Accordingly, the faecal matter is not colourless, as it is in ordinary catarrhal jaundice, where the common bile-duct is for the time being obliterated. In twenty-six cases of hypertrophic biliary cirrhosis absence of colour in the faecal matter has been noticed only twice, and, even when it exists, it is incomplete or transitory. In order to be complete, obstruction of the biliary canals or an arrest in the secretion of bile would be necessary. The attacks of jaundice are in some cases preceded by hepatic pain and fever. The jaundice is sometimes accompanied by pruritus and xanthelasma.

The examination of the urine reveals more than the presence of biliary pigments; the urea is less than in normal urine. This diminution of the urea is not due to the diminution of the nitrogenous foods, for patients continue to eat well, but to the fact that the liver does not act properly. The urine contains neither albumin nor sugar. The patient can take a certain quantity of sugar without glycosuria—a proof that the hepatic cells are but little affected, and that they continue to convert the sugar in the food.

Enlargement of the liver is a constant symptom, and may come on gradually or in successive attacks. It may be subject to oscillations, but it is persistent, and has no tendency to terminate in atrophy. The liver finally becomes enormous, but it retains its form, and its edge remains sharp. It is hard and smooth to the touch, reaches up into the thoracic cavity beyond the fifth intercostal space, and descends into the abdomen as far as the umbilical region, and its left lobe projects into the hypochon-



drium close to the spleen. This hypertrophy causes bulging of the lower intercostal spaces.

Examination of the liver does not cause any pain. In some cases, however, the hepatic region is sensitive on account of the perihepatitis that often accompanies cirrhosis.

The **spleen** is almost always much hypertrophied, and its dimensions may be the easier appreciated, as it attains twice or three times its normal size. The hypertrophy of the liver and of the spleen explains the bulging and the deformity of the supra-umbilical region.

The absence of ascites and of a collateral circulation are negative symptoms of great value. The absence of ascites is not surprising, because the portal venous system is intact. When ascites exists, it usually occurs at an advanced period of the disease. In atrophic cirrhosis the ascites may reach its full development at an early date.

**Duration—Termination.**—Hypertrophic cirrhosis is a chronic and fatal disease, lasting from three to ten years. Patients may, for years, present the cardinal symptoms of this affection—viz., hypertrophy of the liver and spleen with chronic jaundice—without being otherwise incommoded. Many preserve their appetite and strength indefinitely. Sometimes, however, especially in alcoholic or overworked persons, acute phases appear, and are characterized by fever, with hepatic pain and increased jaundice. After a time, loss of flesh, emaciation, and cachexia (dryness of the skin, anorexia, diarrhoea, and cutaneous eruptions) supervene, and end in death. In other cases, death is hastened by a cholera-like attack, due, no doubt, to secondary infection by the coli bacillus. Pulmonary complications sometimes hasten the end. Finally, in some cases, the liver being without defence and the door being open to toxins and micro-organisms, the hepatic cell is destroyed, and symptoms of icterus gravis suddenly appear. They include multiple hæmorrhages, nervous troubles, and coma, which rapidly prove fatal.

**Diagnosis.**—As long as the liver is not hypertrophied and permanent jaundice does not develop, the diagnosis is impossible, since the disease rests on these two fundamental symptoms. The pains associated with jaundice simulate hepatic colic, and the attacks of jaundice, with dyspeptic troubles, resemble catarrhal jaundice. This last error is the more likely, in that catarrhal jaundice may last two months or more, the liver being hypertrophied. It is true that in catarrhal jaundice decoloration of the faecal matter is the rule. It is the exception in hypertrophic cirrhosis.

Later, when the liver has increased in size and the jaundice has become permanent, the diagnosis is even more difficult.

Primary cancer of the liver resembles hypertrophic cirrhosis in the enlargement of the liver, but it differs from the latter in the absence of

jaundice. Secondary cancer is often accompanied by jaundice, but the tumour is uneven and nodular. In cancer of the liver, the spleen is not hypertrophied, and in a few months the disease reaches the cachectic stage. Melanotic cancer presents this peculiarity—that the hypertrophied liver is neither deformed nor nodular; but the other distinctive signs persist, and jaundice is generally absent (Straus).

In leucocythæmia, the liver is often enlarged, and the spleen is hypertrophied, but jaundice is wanting, and examination of the blood is conclusive.

Patients suffering from malarial cachexia may have enlarged livers and spleens, but in these diseases the jaundice is much less pronounced. The actual troubles have been preceded by attacks of malaria, and quinine has an influence that is not seen in hypertrophic cirrhosis.

Hypertrophic cirrhosis has great analogy with hydatid cysts of the liver, and the proof is that exploratory punctures have several times been made, either through error or through an incomplete diagnosis. The hydatid cyst has not on palpation the fibrous hardness of the hypertrophied liver. The spleen is normal, and jaundice is exceptional.

The amyloid liver may be as large as the hypertrophied cirrhotic liver. It is not accompanied by jaundice, and it is related to causes (syphilis, chronic suppuration) favouring the deposit of lardacein in the system.

In bronzed diabetes, marked hypertrophy of the liver may be observed, but the skin of the patient is rather bronzed than jaundiced. The spleen is not enlarged, and the presence of sugar in the urine and the concomitant symptoms give the differential diagnosis.

The diagnosis is sometimes difficult between hypertrophic cirrhosis and a syphilitic liver. Syphilitic hepatitis runs the following course: It causes but slight pain, and is characterized by vague malaise and digestive troubles. It is accompanied by diarrhoea and albuminuria, but hardly ever brings on jaundice. Locally hypertrophy of the liver and of the spleen, without ascites or with moderate ascites, is generally seen. In exceptional cases palpation shows that the surface is uneven. In all cases we must look for symptoms or marks of syphilis; in doubtful cases treatment must be exhibited without delay.

The difference between hypertrophic and atrophic cirrhosis is such that an error of diagnosis is impossible. In the former the liver is bulky; the spleen is much enlarged; jaundice is constant; and we find neither ascites nor collateral circulation. In the latter the liver is small; jaundice is scarcely ever present; and ascites and the complementary circulation are the rule. Between these well-defined types, however, there are mixed forms, presenting some difficulties in diagnosis.

**Splenomegalic Form.**—Hanot's hypertrophic cirrhosis may present varieties differing slightly from the common type. In certain cases the



spleen assumes unusual proportions. In the classical cases the hypertrophy of the liver greatly exceeds that of the spleen. On examining the patient, it is the size of the liver that first attracts attention. The right hypochondrium bulges more than the left. The deformity of the abdomen is rather hepatic than splenic. Post-mortem examination shows that the liver weighs 5 or 6 pounds, while the spleen weighs on an average 2 pounds.

In some cases the spleen is so large that it attracts quite as much attention as the liver. The left hypochondrium bulges as much as the right one, and the spleen descends into the abdomen, forming a tumour of considerable size. To obtain an idea of the size to which the spleen may attain, it will be sufficient to quote a few cases in which the weights of the two organs have been noted :

Liver, 70 ounces ; spleen, 60 ounces (Landrieux and Milian).

Liver, 75     ,,     spleen, 60     ,,     (Guillain).

Liver, 60     ,,     spleen, 70     ,,     (Smith).

Gilbert and Fournier have described this form under the name of "hyper-splenomegalic biliary cirrhosis." I do not think that there is any need for this variety of hypertrophic biliary cirrhosis, because the other symptoms, the anatomical characters, the ætiological conditions, the evolution and the termination, are common to these varieties of biliary cirrhosis. It is, nevertheless, important to recognize these forms with great splenic enlargement in order not to direct the diagnosis unduly towards splenomegaly of a different nature, when the case is really one of Hanot's disease. We find, in opposition to the cases in which the spleen is very much enlarged, cases in which its size is scarcely altered.

**Ætiology—Treatment.**—Hypertrophic cirrhosis is a disease of adult age. Alcoholism can only be blamed in a moderate degree. The hypothesis of infection is possible, but has not been proved.

The treatment is practically the same as in atrophic cirrhosis : purgatives for the dyspeptic troubles, diuretics, tonics, Vichy water, Carlsbad water, Tharasp water, milk diet, iodide of potassium, and hydrotherapy.

## XI. MIXED CIRRHOSIS.

**Description.**—The difference between atrophic cirrhosis and hypertrophic biliary cirrhosis is so great, especially when extreme types are chosen, that certain authors consider them to be absolutely distinct species.

In order to span the distance that seems to separate these two forms of cirrhosis, it will suffice to mention Charcot's propositions :

**Atrophic cirrhosis is annular, multilobular, extralobular, and of venous origin.**

**Hypertrophic cirrhosis** is insular, monolobular, both extra- and intralobular, and of biliary origin.

While the various terms of these propositions may not be absolutely true, it is none the less certain that the process in these two forms of cirrhosis is different. In atrophic cirrhosis the lesion commences in the portal and hepatic veins (bivenous cirrhosis). In hypertrophic cirrhosis the lesion commences in the biliary canals and the hepatic cells (biliary or visceral cirrhosis). In the former the fibrous tissue is indurated and retractile, like scar tissue, and causes atrophy of the organ, and the hepatic cells are frequently affected or destroyed. In the latter the cirrhotic tissue remains in an incomplete fibroid state, and has not the same importance, while the hepatic cells are for the most part intact or hypertrophied.

This difference in appearance, however, does not mean that atrophic cirrhosis and hypertrophic cirrhosis are absolutely opposite. It is true they form two distinct varieties when dealing with extreme types, but on some occasions we find ourselves face to face with mixed or intermediate forms which serve to bridge the extreme types.

To these mixed or intermediate cases I have given the name of **mixed cirrhosis**. Clinically and anatomically they belong partly to atrophic and partly to hypertrophic cirrhosis. Guiter has collected several cases, and a fair number of cases have been since observed.

Clinically, mixed cirrheses are apt to mislead us. We have been so accustomed to the hard-and-fast division of atrophic venous cirrhosis and hypertrophic biliary cirrhosis that, when icterus appears during the course of cirrhosis thought to be atrophic, or ascites appears in a cirrhosis thought to be hypertrophic, the diagnosis becomes doubtful, and we are loath to admit that a case of cirrhosis has broken the barriers imposed by too limited a classification.

And yet cases of mixed cirrhosis are not rare. They show at the same time the symptoms and the lesions of atrophic and of hypertrophic cirrhosis. To enable the reader to form a better judgment I will give a résumé of three cases :

**CASE 1.**—An alcoholic, non-syphilitic patient was taken ill with cirrhosis. He had ascites and complementary abdominal circulation, as in atrophic cirrhosis, and icterus, as in hypertrophic cirrhosis. At the post-mortem examination the liver was small and granular, and the histological examination showed lesions of atrophic (perilobular fibrous sclerosis), and also of hypertrophic cirrhosis (rich biliary network, invasion of the lobule by embryonic cells).

**CASE 2.**—An alcoholic, non-syphilitic patient was taken ill with cirrhosis. The liver was enlarged, and the jaundice was persistent, as in hypertrophic cirrhosis; well-developed collateral circulation and considerable ascites, as in atrophic cirrhosis, were found.

**CASE 3.**—An alcoholic patient was seized with cirrhosis. The disease commenced with persistent jaundice, as in hypertrophic cirrhosis. Considerable ascites then



supervened, as in atrophic cirrhosis. At the post-mortem examination a slightly atrophied liver was found, and the microscopic examination showed an extra- and intra-lobular fibrosis and a marked biliary network at the same time.

Cases of mixed cirrhosis will become more numerous, according as they are sought for. The conclusions which I think we may draw from a study of these cases are that venous atrophic cirrhosis and hypertrophic biliary cirrhosis form varieties that are the more distinct, the more extreme the types described are, and that it is greatly to the credit of the Paris school to have shed light on the chaos of chronic hepatitis. The spirit of systematization and of classification must, however, not be pushed too far. Clinical medicine accommodates itself but ill with too clearly defined a selection of morbid species, and the lesion is here, as always, in agreement with the clinical conditions. Between the extreme types described in the preceding sections there is room for intermediate forms, and it seems to me that the name "mixed cirrhosis" should be applied to them.

## XII. TUBERCULOSIS OF THE LIVER.

Writers in the first half of the last century considered tuberculosis of the liver as an exception. In the opinion of Andral and Cruveilhier, fatty degeneration was the usual lesion of this organ in phthisical patients.

Thaon, in 1872, stated that, in eight cases out of ten, careful observations would reveal nodules in the liver of tubercular patients; but, later, Julius Arnold went so far as to state that the nodules were found in all cases of tuberculosis. The truth is that, while the hepatic tubercle is frequent in all varieties of tuberculosis, it is the rule in children, and during the course of acute abdominal tuberculosis. The nodule is its most frequent form, but the lesion may run its normal course, and give rise to caseous masses, cavities (Sergent, Jacobson), and abscesses.

Steatosis and tubercles are not the only lesions seen in the liver of phthisical patients; fibrosis may also occur in it, as in the lung. The history of tubercular cirrhosis of the liver has been elucidated during the past few years, and furnishes an interesting contribution to the general history of cirrhosis of infectious origin.

In addition to the fatty hypertrophic cirrhosis of Hutinel and Sabourin (1881), the works of Hanot, Lauth, and Gilbert have taught us that there are two other varieties of tubercular cirrhosis—the one furrowed and comparable with the condition found in syphilitic patients, and the other closely resembling common alcoholic cirrhosis.

In phthisical patients with cachexia amyloid degeneration of the liver may be found, and in phthisical patients with diseased hearts the so-called cardiac liver may be noticed.

These changes are not peculiar to tubercular patients, and have been described elsewhere. The experimenter, as well as the pathologist and the clinical physician, has derived benefit from the study of tuberculosis of the liver. It must not be forgotten that the experimental hepatic tubercle has furnished most valuable enlightenment on the much-disputed histogenesis of the tubercle.

**Pathological Anatomy.**—The tubercular nodule is the most characteristic lesion. The liver is congested, and the nodules are widely disseminated in the lobules and in the porto-biliary spaces. They form little

round grey and semitransparent spots, that often require a lens in order to be seen. According to Brissaud and Toupet, the topography of the tubercle, which varies in each case, is subject to certain laws. In the same liver the nodule is said to be always systematized in the subdivisions of the same order of Glisson's capsule. The sublobular portal space is said to be the most frequent seat of the lesion. In time the tubercles may increase in size and become caseous. The cavity is an exceptional lesion in the liver of tubercular patients. When it exists, it is always peribiliary, and is the result of tubercular angiocholitis. The walls of the biliary canal in which it is inserted are studded with fine granules. By virtue of this localization of the hepatic cavities around the biliary canals, Chauffard supposes that other associated germs of intestinal origin assist Koch's bacillus. It is very difficult to stain Koch's bacilli in tuberculosis of the liver, no doubt because of the special qualities of the chemical action in the liver.

Fat is found in almost every tubercular liver. Fatty degeneration of the liver may be either systematic or general. Around the hepatic tubercles there may often be seen a small zone of fatty degeneration, which forms, "as it were, a layer concentric to the central epithelioid and embryonic zones" (Hanot and Lauth).

Under the name of nodular fatty evolution in tubercular patients Sabourin has described a lesion, arising systematically around the porto-biliary spaces as a centre, and sparing the parenchyma around the central veins. Whether the steatosis is local or general, the fat always infiltrates the cellular elements. The nuclei still retain their affinity for taking stains. According to Hanot and Lauth, it is simple infiltration, and not degeneration. The process of steatosis and fibrosis may combine so as to produce the lesion known as fatty hypertrophic cirrhosis, the ætiology of which, according to many writers, depends on both alcoholism and tuberculosis.

The liver is enlarged, its edges are thickened, and its weight may be 100 ounces. It is of a yellow-ochre colour, and the cut surface is smooth and fatty. Small rosy patches in the porto-biliary spaces are formed of new connective tissue. Under the microscope we find porto-biliary cirrhosis, which is both insular and diffuse. The connective tissue thus developed around the blood and biliary vessels has characters differentiating it from other cirrhoses. It does not bring about fibrosis. It is studded with tubercular follicles, which sometimes penetrate the adjacent parts of the lobule, and it sends into the lobule fine pencilled bands that separate and envelop the hepatic cells, so as to form a true monocellular cirrhosis. Here, again, the hepatic cells are surcharged with fat, and are not in a condition of degeneration.

Amongst the other types of tubercular cirrhosis the most interesting is the variety described by Hanot and Lanta. The liver is lobulated and



furrowed by fibrous bands, that cause it to resemble the liver produced by tight-lacing. The cirrhosis is porto-biliary, with numerous biliary canaliculi. There is a periportal steatosis, with fine intercellular connective radiations, and numerous tubercular granulations disseminated in the hepatic parenchyma.

There is a rare form of hepatic tuberculosis, characterized by nodular hepatitis without fatty degeneration, and analogous to that described by Kelsch and Kiener in the malarial liver. This hepatitis is either isolated or associated with hepatic cirrhosis. The various lesions, which I have separated for purposes of description, are often isolated or at times differently associated in the livers of tubercular patients.

**Experiments and Pathogenesis.**—The entrance-gate of the tubercle bacillus into the liver of human beings is very variable. The bacillus may enter by the portal veins, or by the arterial vessels, by the peritoneum, and by the biliary passages. In the foetus it may, in exceptional cases, follow the umbilical vein (Sabouraud). The hepatic tubercle has been reproduced experimentally in animals by varying the afferent channel. The injection of tubercular matter into the peritoneum of a guinea-pig causes death in two to six weeks. The liver and spleen are filled with tubercles, and the retroperitoneal and subcutaneous glands are swollen and caseous (Straus and Gamaleia). In man tuberculosis of the liver is often combined in like manner with tubercular peritonitis.

The inoculation of a pure culture of tuberculosis into the mesenteric veins of a rabbit (Gilbert and Lion) causes the death of the animal in three to five weeks. The liver and the spleen are the only organs crammed with tubercular nodules. The tubercles appear on the seventh day around the periportal capillaries. Their evolution follows the multiplication of the bacilli in the vessels.

A virulent culture of human tuberculosis injected into the general circulation by the marginal vein in a rabbit's ear produces rapid miliary tuberculosis (Koch, Straus and Gamaleia). The bacilli in this case reach the liver by the hepatic artery, and so cause tuberculosis.

Cornil and Yersin, using the same method, only found acute septicæmia, with swelling of the liver and the spleen, which were crammed with bacilli, though no tubercles were present. The reason was that they had employed the cultures from avian and not human tuberculosis. Hanot and Gilbert were able to observe tubercular fibrosis of the liver in a guinea-pig. The fibrosis was perilobular, and the fibrous bands penetrated the lobule. They only saw in this a tubercular lesion, giving rise to fibrosis. In a guinea-pig inoculated with avian tuberculosis the same writers were able to produce a tubercular furrowed liver.

Finally, Pillet has recently shown that cultures of human tuberculosis

inoculated in a guinea-pig or in a dog may cause large degenerative lesions in the liver from coagulation necrosis. The hepatic cells swell, their protoplasm liquefies, and their nucleus loses its affinity for colouring matter.

Experimental tubercular hepatitis has contributed, as we have said, to the elucidation of the histogenesis of the tubercle in general. Cornil and Yersin made intravenous injections of avian cultures in rabbits, and were able to observe the course of the lesion in the liver. About the fifth or sixth day after inoculation the bacilli simply cause small fibrinous clots in the capillaries, where they end near the portal spaces. A zone of leucocytes soon envelops the fibrinous thrombus within the vessel. The leucocytes, no doubt under the influence of the substances secreted by the bacilli, are converted into epithelioid or giant cells. The process has not time to proceed farther, as death supervenes about the end of the third week. Metchnikoff, and Gilbert and Girode consider that the principal part in the formation of the nodules and of the giant cells is played by the endothelial cells of the vessels and by the large mononuclear leucocytes. One of the proofs of the activity of these elements is the presence of glycogen in the protoplasm.

The hepatic cells do not, as Baumgarten maintained, take part in the initial constitution of the tubercle. The hepatic tubercle may consist in a simple agglomeration of round cells, like the infective nodules of typhoid fever or smallpox. Experiments as well as pathological anatomy prove, therefore, that the tubercle bacillus produces in the liver the fatty degeneration of the cell and the fibrosis, as well as the tubercle. How does it produce these different lesions? Does it do so by direct action, or by means of the toxins which it secretes? According to Hanot and Lauth, the bacilli act through their secretions, which are said to be sclerogenous for the connective tissue and fat-producing for the hepatic cell. The experiments carried out with Koch's tuberculin have not confirmed this alluring hypothesis (Chauffard).

It is, perhaps, more probable to consider, with Hanot and Gilbert, that the difference of the lesions is due to an abnormal individual resistance against the tubercle bacillus, or to an infection of the organism by bacilli which, in the very extensive scale of virulence that the bacillus of Koch must possess, occupy no very high place as regard species.

Koch's bacillus is, perhaps, not the only factor at work. Alcoholism has been considered as a contributory cause in the first researches published on fatty hypertrophic cirrhosis. Whilst Sabourin wrongly adopted alcohol as the only cause, Hutinel explained the fibrosis by alcoholism, and the fatty condition of the liver by tuberculosis. According to Hanot, alcoholism plays only a secondary part, and recent observations made by Hutinel on young children free from the suspicion of alcoholism are in favour of this opinion.



The researches of Hanot and Létienne have proved that biliary infections play a secondary part.

**Symptoms.**—Clinically, hepatic tuberculosis is, as a rule, silent in its progress, whether we have to deal with tubercular granules, peribiliary cavities, or nodular foci. The symptomatology in these cases points to the general disease—tuberculosis—rather than to the hepatic localization. If, however, we make a practice of examining the liver in tubercular patients, certain signs will show that the organ is affected. The slightly hypertrophied liver, extending two or three fingers' breadth below the edge of the false ribs, may be somewhat painful on pressure. The spleen is also somewhat enlarged. Slight jaundice, some decoloration of the fæces, scanty and brick-coloured urine, urobilinuria, and alimentary glycosuria, show that the liver is diseased. As for the anatomical type, it is sometimes impossible to decide without taking these symptoms into account, and confusion between a fatty and an amyloid liver is still possible. Tubercular cirrhosis, however, has a much clearer clinical individuality, which should be well known, so that errors of diagnosis may be avoided.

During the past few years two types have been described—fatty hypertrophic cirrhosis of the Hutinel-Sabourin type and tubercular cirrhosis of the Hanot-Lauth type.

In fatty hypertrophic cirrhosis a premonitory period and a stationary stage may be distinguished. In the premonitory period the patient is an alcoholic who shows all the symptoms of gastro-intestinal alcoholism—viz., phlegm, anorexia, vomiting, slight swelling of the liver, and scanty urine, loaded with urates. On these symptoms there are grafted those of pulmonary tuberculosis, which most frequently become predominant. After some months, a chill, a fresh outburst of tuberculosis, or some alcoholic excess, causes the latent cirrhosis to throw off its mask. The digestive troubles, then, become marked, the anorexia is complete, the skin assumes a yellowish tint, and the urine becomes scanty, with but little urea, though, on the other hand, urobilin, sugar, and sometimes albumin, are present.

The fæcal matter is sometimes colourless; the lower limbs become œdematous; attacks of hæmorrhage (especially epistaxis and hæmatemesis) may appear; and the patient falls into a state of profound asthenia. To these symptoms of auto-intoxication there are added a subcontinuous febrile condition and an aggravation of the pulmonary tuberculosis, which may prevent the lesion being recognized. Ascites is, in general, negligible, and the subcutaneous veins are not much dilated. The key to the diagnosis lies in the examination of the liver, which is considerably increased in size, and extends below the false ribs by four or five fingers' breadth. It is hard, smooth, and painful on the slightest pressure. The spleen is likewise enlarged. This stationary stage, characterized by painful hyper-

trophy of the liver, with hepatic insufficiency, is generally complete in five or six weeks.

Although alcoholism is frequent in the premonitory period, it is not indispensable to the development of fatty hypertrophic cirrhosis, as shown by the recent observations of Laure and Honorat, and of Hutinel, in children. Tubercular cirrhosis of the Hanot-Lauth type, which anatomically is allied to alcoholic cirrhosis by the topography of the fibrosis, may show itself clinically by the cardinal symptom of Laënnec's cirrhosis—ascites. The abdominal effusion and the collateral circulation may be sufficiently marked to make the diagnosis between the two affections most difficult.

The appearance of abdominal pains, tenderness of the liver on pressure, marked jaundice, and rapid cachexia, are especially observed in tubercular cirrhosis. Advanced signs of pulmonary tuberculosis or of tubercular peritonitis are most important in diagnosis. Tubercular peritonitis often completes this form of cirrhosis, and in a number of cases, clinically labelled as tubercular peritonitis of an ascitic form, the effusion is due rather to a cirrhotic lesion than to peritonitis. Clinical medicine has, therefore, gained by the careful anatomical study of the liver in tubercular patients during recent years, and we see that the methodical examination of this organ in such patients will furnish valuable indications as to prognosis and diagnosis.

### XIII. CANCER OF THE LIVER AND BILE-DUCTS.

**Pathological Anatomy—Ætiology.**—Cancer of the liver may be primary or secondary. Primary cancer is very rare, since it forms only the eighth part of the cancerous tumours of this organ, but secondary cancer is frequent. It may, indeed, be said that secondary growths are most common in the liver. Cancer of the stomach, intestines, rectum, lungs, uterus, bones, skin, or choroid, may lead to secondary growths in the liver, including columnar-celled epithelioma, encephaloid, scirrhus, hæmatoid, colloid, and melanotic carcinoma, according to the rule that a secondary cancer is always of the same kind as the primary growth. The encephaloid form and the cylindrical epithelioma are the most frequent.

1. *Secondary Cancer.*—I have just said that columnar-celled epithelioma is one of the most common malignant tumours of the liver. The reason is that this variety has as its seat of origin the stomach, the intestine, and the bile-ducts, the mucosæ of which are provided with columnar epithelium, and has, as a means of transport, the portal veins leading from these organs to the liver. Histologically, the columnar epithelioma differs from encephaloid and colloid carcinoma by reason of its tube-like or irregular cavities, lined with columnar cells, and seated in the midst of a fibrous, embryonic, or mucous stroma; but to the naked eye "this epithelioma presents abso-



lutely the same disposition, the same dissemination, and the same aspect as encephaloid carcinoma."

These secondary cancers, instead of forming one single mass, like massive primary cancer, form islets that are more or less spherical, mammillated, varying in size from a millet-seed to the head of a foetus, and disseminated throughout the entire organ. On scraping the cut section, the islets yield a milky juice, and their yellowish-white colour stands out against the brown-red background of the parenchyma. The nodules which protrude on the surface of the liver are often umbilicated, from the degeneration and softening of their central portion. The softening may be so complete as to convert the cancerous tumour into a cyst, which, in its turn, becomes the seat of hæmorrhages. These cancers acquire enormous proportions, some weighing as much as 20 pounds. Their development is sometimes very rapid, and they may arise from a cancerous ulcer of the stomach hardly as large as a florin. Melanotic sarcoma also grows to a large size. It is usually secondary to a primary growth in the choroid. It is not nodular, like the encephaloid cancer, and the hepatic tissue is infiltrated with black pigment.

A study of the cancerous lesions under the microscope will show that all the elements of the hepatic lobule (cells, vessels, biliary canals, and connective tissue) are invaded by the cancer. If we study the hepatic zone next to the cancer, which serves, so to say, as the transition tissue, we find that the hepatic cells are deformed, spindle-shaped, hypertrophied, and swollen by accumulations of protoplasm. The interlobular connective tissue loses its fibrous appearance, and becomes infiltrated with embryonic nuclei. The ramifications of the portal vein and of the hepatic artery which surround the lobule become thickened, and their walls are infiltrated with cancer cells. The endothelium of the vessels is, in its turn, invaded, and cancerous vegetations project into the lumen of the vessel. The biliary canaliculi are invaded in a similar manner; their calibre is increased, and their walls are infiltrated with cancer cells. The same process may attack the larger branches of the portal vein, causing ulceration of the walls. "It would be incorrect to say that the growth near the vein has perforated the wall so as to open the vein, for it is the carcinomatous nodule in the walls of the vein that has become ulcerated" (Cornil and Ranvier). The question is: Which tissue of the liver is first attacked by the cancer? Is it the glandular (Rokitansky, Lancereaux), or the connective tissue (Virchow, Vulpian)? Both opinions are admissible, though the epithelial origin now seems to be proved. It is, however, certain that the portal vessels and the biliary ducts play a considerable part in the transportation and dissemination of cancer.

2. *Primary Cancer*.—Primary cancer is much rarer than secondary cancer. In some cases it has the nodular form of secondary cancer, but it most frequently deserves the name of massive cancer, because it forms

a uniform mass. When the growth does not reach the surface of the organ, the cancer is said to be almond-like. The liver in massive cancer is not nodular and deformed externally, as in secondary cancer, and its surface and form remain normal; but the hypertrophy, especially in the right lobe, is such that it may weigh from 15 to 18 pounds.

On section, the liver is transformed into a soft or lardaceous mass, yielding a small amount of cancerous juice on scraping. The surface of the section is greyish or yellowish, and the centre of the cancer is hardly ever softened, whilst softening is frequent in the nodules of secondary cancer. In some cases a few nodules are found around the main growth.

The extrahepatic bile-ducts and the large arterial and venous trunks are healthy. Perihepatitis, which is frequent in secondary cancer, with its superficial nodules, is rare in massive cancer. Here, as in secondary cancer, cancerous degeneration is found in the glands of the hilum as well as in the gastro-hepatic, peripancreatic, prevertebral, and mediastinal glands, into which the lymphatics of the liver empty. Specific emboli may follow the course of the hepatic veins and sow cancer in the lungs.

At first sight massive cancer has most frequently the look of encephaloid cancer, with or without hæmorrhagic foci. Histological examination shows that it is a variety of epithelial cancer. Primary cancer of the liver assumes two principal forms—alveolar epithelioma and trabecular epithelioma. These two forms may combine and take on the trabeculo-alveolar form, but in the great majority of cases the alveolar epithelioma is the type of massive or nodular primary cancer, whilst the trabecular epithelioma is the type of primary cancer with cirrhosis.

3. *Adenoma and Cirrhosis*.—When describing alcoholic venous cirrhosis of the liver, I said that discrete or confluent and small or large adenomatous growths may be met with in cirrhotic livers. These growths do not project in a section of the liver; they have a putty-like consistency and a yellowish-grey colour, sometimes tinted by hæmorrhage. A stream of water may enucleate them from the surrounding capsule.

According to some writers, the association of cirrhosis with adenoma of the liver may be explained in the following manner: Interstitial hepatitis opens the scene, and produces cellular irritation and parenchymatous hepatitis, which may end in the formation of epithelial tumours or adenomata. From this point of view the adenoma would so far be a benign tumour, like adenomata of the breast. The adenoma, however, may, according to some writers, be transformed into carcinoma—that is to say, into a malignant tumour. The proofs of this transformation are said to be drawn, not from the structure of the tumour, for the structure of a tumour does not suffice to indicate its malignancy; they are drawn from the general invasion of the glands of the hilum and “the neoformation



of a tissue made up of hepatic cells in the interior of the portal vessels—an indication that the adenoma becomes infective.” Adenoma of the liver is said to have this character in common with certain adenomata of the breast, which, after being harmless for a long time, assume the characters of malignant tumours. According to Brissaud, the series of transformations is even more complete. The venous cirrhosis is the cause of the adenoma, and the adenoma is transformed into carcinoma—in other words, cirrhosis, adenoma, and carcinoma are successive links in the same pathological chain.

Gilbert interprets the association of adenoma and of cancer in another manner. According to him, there is no question of transformation, for adenoma and cancer are one. “Adenoma, as described by Kelsch and Kiener and by Sabourin, forms, according to my opinion, a particular variety of hepatic carcinosis, and cannot therefore undergo cancerous transformation.”

This is also the opinion of Hanot: “Adenoma is a variety of epithelioma. It is a trabecular epithelioma, also called adeno-carcinoma, and it might be called cylindroma, tubular epithelioma, or acinous epithelioma.” As I have previously remarked, in the great majority of cases alveolar epithelioma is the type of massive or nodular primary cancer, whilst trabecular epithelioma is the type of primary cancer with cirrhosis. Hanot and Gilbert write: “The relations of trabecular epithelioma and of cirrhosis have been differently understood by writers. Lancereaux supposes that the cirrhosis results from development of the neoplastic nodules in the liver. Sabourin, on the other hand, places cirrhosis first in chronological order, and looks upon adenoma as a complication of cirrhosis. We cannot accept either of these explanations. Lancereaux’s explanation, which might be maintained if it were always a question of the coexistence of neoplastic nodules disseminated throughout the liver, together with cirrhosis, cannot account for cases in which only a few neoplastic nodules coexist with cirrhosis. Sabourin’s explanation falls to the ground when confronted with the fact that adenoma may exist without cirrhosis. We admit, with Kelsch and Kiener, the simultaneous development of cirrhosis and adenoma, and we believe that these two processes result from the action of the same irritating agent on the connective tissue and on the hepatic epithelium, that the cirrhosis may evolve by itself, and also that, in exceptional cases, trabecular epithelioma may develop separately.” This simultaneous process of cirrhosis and adenoma was very evident in one of my cases. The invasion of the lymphatics and of the glands is rapid in alveolar epithelioma, whereas trabecular epithelioma invades the veins and respects the lymphatics.

The study of adenoma and cirrhosis has, recently, been undertaken by Rénon and his pupils. They have given the lesion the name of **hepatoma**. The hepatoma is a tumour which, originally, has multiple foci; it is characterized by exuberant proliferation of the elements of the hepatic

parenchyma following the embryonic type, with a tendency to extend locally, among the vessels. This tumour is neither an adenoma, nor a cancer.

4. *Ascites and Jaundice*.—The compression of the portal vessels by the cancerous tumour causes ascites; the compression of the bile-ducts in the hilum of the liver causes jaundice.

Adhesions are established between the cancerous liver and the neighbouring organs, and are frequently invaded by the growth.

The causes of hepatic cancer are as obscure as the causes of cancer in general. Hereditary predisposition plays a great part, and though cancer of the liver is a disease of advanced age, it is, nevertheless, seen rather often in adults, and even in children.

**Symptoms.**—Secondary cancer of the liver sometimes shows itself as a late epiphenomenon in advanced stages of cancer of the stomach, intestine, or rectum, and the patient succumbs before the hepatic lesion has given rise to special symptoms. Sometimes, on the contrary, cancer of the stomach, gall-bladder, or intestine, has only shown indefinite symptoms, when the hepatic cancer declares itself, and spreads with such rapidity that it claims the entire attention. Finally, there are cases—rare, it is true—in which hepatic cancer is primary. In these various conditions what symptoms and signs indicate cancer of the liver?

Let us deal first with secondary cancer. It often commences with a latent stage, and some patients have advanced cancer (hypertrophy and nodules in the liver, cachexia) without having experienced manifest symptoms. Amongst the symptoms digestive troubles appear first, and we find dryness of the mouth, loss of appetite, anorexia, ballooning of the belly, and foetid stools. So far there is nothing significant. The patient complains of heaviness or dull pain in the right hypochondrium. Acute pains are absent, unless perihepatitis is present. Jaundice is frequent, and has been noticed in thirty-nine cases out of ninety-one (Frerichs). It varies from a slight tinge to deep and permanent coloration. It is due to multiple causes: catarrh or compression of the bile-ducts. Ascites is frequent, but not as abundant as in cirrhosis. It is due to divers causes: cancerous masses obstructing the large trunks of the portal vein, partial peritonitis near the liver, or the cancerous nodules which invade the peritoneum and the omentum. In the latter case the ascitic liquid is often blood-stained.

Melæna is fairly common when the cancer attacks the gall-bladder and bile-ducts (Hanot). Dilatation of the subcutaneous abdominal veins is often noticed.

The liver is enlarged in four-fifths of the cases (Lebert), and may weigh 15 or 16 pounds. It reaches below the false ribs, and stands out in relief under the abdominal wall, extending below the umbilical region, and encroaching on the left flank. When the growth is formed of large nodules,



as is usually the case, the upper surface of the liver is hard, uneven, and nodular. Its sharp edge, instead of being smooth and thin, grows soft and irregular. In some cases the growth of the cancer is so rapid that considerable increase is noticed from one week to another. The spleen is never hypertrophied. This negative sign is most valuable.

As the disease progresses the general symptoms become more marked. Emaciation, loss of strength, anæmic colour of the skin, gradual diminution of the red blood-corpuscles, and peripheral œdema, are the signs of cancerous cachexia, and the patient dies—in a couple of months if the course of the cancer is acute, and in six or eight months if the course is chronic. Death is due to cachexia or to complications, such as subacute peritonitis, cerebral troubles and multiple hæmorrhages, icterus gravis, lowering of the temperature, or rupture of a blood-cyst found in the interior of a cancerous nodule.

Primary cancer of the liver in its massive form presents a somewhat different picture. Anorexia, nausea, and meteorism open the scene, as in secondary cancer. Pain in the right hypochondrium is common, but ascites, dilatation of the subcutaneous abdominal veins, and jaundice are almost always absent. The chief sign is hypertrophy of the liver, which is usually considerable; but the liver is smooth and hard, with a sharp edge, that shows neither the indentations nor the deformities found in secondary cancer. Another very important symptom is acholia, or hypocholia. The liver makes but little bile, because many cells are replaced by the neoplasm. The fæcal matter is almost colourless, and very offensive. The intestinal coils are distended by gas. The same cause—*i.e.*, the changes in the liver producing acholia—opposes the production of jaundice. “Jaundice in these conditions is almost unrealizable, even though the excretory bile-ducts are blocked” (Gilbert). Though the acholia (acholia by secretion) may not be absolutely special to massive cancer of the liver, it has none the less a great value. It realizes the type of acholia from secretory trouble, in which decoloration of the fæcal matter coincides with the absence of jaundice, and is very different from false acholia or acholia caused by excretory trouble, such as obstruction of the common bile-duct, in which case decoloration of the fæcal matter is accompanied by an icteric tint of the skin and of the urine. The urine is scanty, and never contains albumin. it is very poor in urea, on account of the lesions in the liver and of the inanition (Robin).

Cachectic œdema, obliterating phlebitis and diarrhœa, are observed in all forms of cancer. During the course of hepatic cancer fever is at times a symptom of great importance. It may be remittent (Monneret) or intermittent, with evening exacerbations. It is due to perihepatitis or to true attacks of hepatitis. Febrile cancer ends very rapidly in death.

**Diagnosis.**—The diagnosis of cancer of the liver is especially difficult when the cancer is primary and massive, because it has several signs in common with the amyloid liver, with certain forms of hypertrophic cirrhosis, and with hydatid cysts.

Amyloid degeneration of the liver causes smooth hypertrophy of the liver, with absence of jaundice and ascites, just like massive cancer; but it differs from cancer in the hypertrophy of the spleen, in the presence of albuminuria and in the pathological conditions (scrofula or prolonged suppuration) which cause lardaceous disease.

Hypertrophic biliary cirrhosis, like massive cancer, causes enormous smooth hypertrophy of the liver, without ascites and without dilatation of the abdominal veins; but it differs from cancer by its slow course, polycholia, and early persistent jaundice.

Hydatid cysts of the liver, like massive cancer, cause a large smooth tumour, with absence of ascites and icterus; but they differ from cancer in the following points: In hydatid cyst the liver is not uniformly enlarged, but is swollen at one point, and gives a feeling of resistance or almost of fluctuation, and not a feeling of wood-like hardness. The cyst is for a long time compatible with good health. The diagnosis is very difficult in a case of alveolar cyst, and tapping is often the only means of diagnosis.

The diagnosis is sometimes difficult between cancer and syphilis of the liver, because we find in either case an enlarged lobulated liver, jaundice, ascites, œdema, and cachexia. The only distinctive sign is that in cancer the complications are rapid, whereas they are slow in syphilis.

The **prognosis** is absolutely fatal, and the **treatment** is purely palliative. It consists in maintaining the patient's strength and in treating the symptoms as they arise.

### Cancer of the Biliary Passages.

Cancer of the gall-bladder and bile-ducts is almost always primary.

Primary cancer of the gall-bladder may be columnar-celled, encephaloid, colloid, or scirrhus. The mucous surface of the bladder is villous at the points invaded by the new growth. The cavity of the bladder is generally enlarged, and the walls, especially the tunica muscosa, are hypertrophied.

The bile contained in the bladder is sometimes colourless, sometimes brown and thick. Gall-stones are usually found (fourteen times in fifteen cases), and it is probable that the appearance of the cancer precedes the formation of the calculi.

The tumour commences in the bladder, and then invades the cystic and common ducts, narrowing their calibre. The neighbouring lymphatic glands are attacked, and the duodenum, the colon, and the stomach are the seat of adhesions, often infiltrated with growth. The liver is almost always



invaded, and in certain cases the primary growth in the gall-bladder is small, while the liver is crammed with secondary growths.

The cancer may primarily invade the large bile-ducts, and Claisse has compiled the following statistics: Primary cancer of the large bile-ducts is often situated at the duodenal end of the common duct. The lesion may, however, occur in other spots. In Claisse's two cases the cancerous nodule attacked the hepatic duct and the middle portion of the common duct. In Griffon and Artigue's case the cancerous nodule was not far from the end of the common duct, but did not involve the ampulla of Vater. Hebb, Birsch-Hirschfeld, and Kraus have noted the localization of cancer in the middle portion of the common duct. In Rabé's case the cancer was seated in the middle portion of the common duct, and the gall-bladder was involved. In the two cases of Naunyn and Schuppel the cancer was situated in the hepatic duct or at the union of the hepatic and cystic ducts. Such was the case in one of Jourdan's patients. In Debove's case the cancer was situated between the cystic duct and Vater's ampulla. In my own case the cancer was found at the union of the cystic and common ducts.

Cancer of the common bile-duct generally causes permanent jaundice, and the diagnosis is very difficult, as we shall see in Section XXI. When the infection involves the liver, the symptoms are blended with those of hepatic cancer. Melæna almost always points to the presence of cancer of the biliary passages (Hanot).

#### XIV. SYPHILIS.

In this section I shall describe secondary and tertiary troubles and hereditary syphilis.

##### 1. Secondary Troubles—Jaundice.

**Benign Jaundice.**—Jaundice appears within a few months of infection. The colour of the skin is sometimes light, sometimes deep, yellow, and the fæces may or may not be colourless. The jaundice may appear as an isolated symptom, but it more often is accompanied by digestive troubles, diarrhoea, and especially by general symptoms—fever, lassitude, malaise, headache—which belong to this stage of syphilis. The pathogenesis of the condition is imperfectly known. It is probably due to catarrh of the bile-ducts, analogous to the secondary syphilitic catarrh of the mucosæ of the pharynx, larynx, or bronchi, or to lesions of the parenchyma without alteration of the bile ducts.

**Icterus Gravis.**—Icterus gravis may appear during the secondary stage of syphilis. Contrary to the opinion of Mauriac, who believed in simple coincidences, the observations of Lebert, Gubler, Lacombe, Senator, Roque and Devic, and Talamon, show that icterus gravis is a direct manifestation

of the syphilitic infection. It is chiefly seen in women. In Talamon's case the histological examination showed acute diffuse hepatitis, characterized by embryonic infiltration of the whole connective tissue of the organ, with more or less profound destruction of the hepatic cells. The lesions are those of acute yellow atrophy, without any specific character. Early hepatic syphilis, then, may be compared with early renal syphilis, in which the anatomical lesions are those of ordinary toxic nephritis. Between the benign and malignant varieties of early syphilitic icterus there are intermediate forms, as one of Senator's cases seems to show.

As it is important not to confound syphilitic icterus with common catarrhal icterus, we must always look for some syphilitic manifestation on the mucosæ or the skin. The spots of the macular roseola may be hidden by the jaundice, but the papular, squamous, or lenticular syphilides are perfectly recognizable. The syphilides of the throat, mouth, vulva, or anus, the crusts of the scalp, and adenitis in the groin, are signs which may help us in the diagnosis, and must be carefully sought after. The pathogenic cause of the jaundice being recognized, it is necessary to order immediate treatment with mercury.

## 2. Tertiary Troubles—Syphilitic Liver.

**Pathological Anatomy.**—In hepatic syphilis, perihepatitis is more pronounced than in any other form of chronic hepatitis. It forms solid and resistant adhesions between the liver and the diaphragm. The false membranes may surround the liver, compressing the veins and ducts in the hilum, and causing ascites and jaundice.

**Syphilis of the liver** shows itself in the form of fibrosis or of gummata. The two kinds of lesions are generally associated (sclero-gummatous hepatitis), though one of them may predominate. We can, then, describe (1) fibrous hepatitis; (2) gummatous nodular hepatitis.

1. **Diffuse fibrous hepatitis** has some characters in common with ordinary cirrhosis. The liver is hypertrophied or atrophied, nodular, dark brown, and lobulated rather than granular, whilst it is granular rather than lobulated in atrophic cirrhosis. In typical cases the lesion has the following characters: the edges of the liver are anfractuous and irregular; the surface is nodular, and furrowed by deep fibrous cicatrices, whence the name of **tight-laced liver**. The fibrous tissue, which forms bands, encloses small indurated or caseous gummata. Adhesions are formed between the liver and the neighbouring organs (kidney, colon, or diaphragm). In some cases atrophy of one lobe and hypertrophy of the other are noticed. Under the microscope the syphilitic liver shows a mixture of common and of hypertrophic cirrhosis. The cirrhotic tissue surrounds the lobules, and penetrates into them. The hepatic arterioles are affected by endarteritis, and are



doubtless the origin of the sclero-gummatous process. These arterial lesions are almost peculiar to syphilitic cirrhosis, and portal phlebitis is of secondary import.

2. **Gummatous nodular hepatitis** is characterized by the presence of gummata on the surface or in the parenchyma of the liver. The tumours rarely exceed the size of a pea or of a small nut. They are generally spherical, and often grouped in islets, each enclosing several gummata. During the first period of their growth the gummata are greyish and resistant, but later their centre becomes opaque and softens. The fibrous tissue then invades the little tumour, and the gummata contract, and those present on the surface of the liver under Glisson's capsule often terminate in star-shaped cicatrices with puckered edges. On making a section of a gumma three distinct zones are seen. The central part is opaque and caseous, and often divided into islets. Around this central mass is a fibrous covering, composed of connective tissue, that varies in appearance according to the age of the gumma. The most external zone is formed of fibrous tissue, which infiltrates between the neighbouring hepatic lobules.

To these lesions must be added amyloid degeneration, which attacks the liver as a secondary lesion in the same manner as it attacks the spleen and the kidneys. Hanot found hypertrophic hepatitis with splenomegaly and chronic jaundice in three cases—an anatomical syndrome which belongs to hypertrophic cirrhosis. In the lobule he found dilated capillaries, the walls of which were infiltrated with nuclei, and filled with white corpuscles; displaced rows with atrophied, hypertrophied, or disaggregated cells; numerous embryonic nodules in the portal spaces—lesions, in short, common to every infected liver. Hanot in his specimens, however, did not find telangiectatic cirrhosis, catarrhal angiocholitis, or newly-formed biliary canaliculi.

**Description.**—The tertiary lesions are generally late, but they may appear as early as the third or fourth year after infection. Their onset is usually obscure, and is not accompanied by acute pain, fever, or marked symptoms. Only slight digestive troubles may be found. Later, the symptoms are more evident. The appetite is bad, the digestion is at fault, the loss of flesh progresses, and diarrhoea, œdema of the feet and legs, and absolute loss of strength appear. The patient has a yellowish colour, but true jaundice is rarely seen. Ascites and collateral circulation are often absent, or only appear at a late period. In some cases hepatic syphilis is most insidious, and ascites is the first symptom. It is due to several causes—either compression of the portal vein by the glands in the hilum or by adhesions, or to compression of the hepatic veins at their entrance into the vena cava (Barth).

Examination of the liver furnishes indefinite information. The liver

may be hypertrophied or atrophied. It is nodular and uneven. Hanot has applied the name of "syphilitic hypertrophic hepatitis with chronic jaundice" to a special form of tertiary hepatic syphilis, which he considers a morbid entity. As it is accompanied by splenomegaly, it somewhat resembles certain forms of malarial hepatitis and Hanot's disease. It is distinguished from the latter by the syphilitic ætiology, by the absence of paroxysmal attacks, and by the more rapid march of events when treatment has been taken in hand too late. Furthermore, the spleen and the liver are never as large as in Hanot's disease. Finally, leucocytosis, which is the rule in the latter affection, has not been noted in the former.

Hepatic syphilis runs a very slow course. It may take years. As long as the liver performs its functions sufficiently, and as long as the other viscera are free, there is no danger; but the risks arise when amyloid or fatty degeneration causes hepatic insufficiency, or when the kidneys are attacked, and urinary insufficiency results.

### 3. Hereditary Syphilis.

Hereditary syphilis of the liver in the new-born child (early hereditary) and that at a more advanced age (late hereditary) must be described separately.

**Description.**—Syphilis of the liver during intra-uterine life hampers the venous circulation. The pressure in the umbilical vein increases, and extra-fœtal ascites or hydramnios is produced. The size of the mother's abdomen may be enormous (48 inches at the umbilicus). The movements of the fœtus cannot be felt distinctly, and the uterus forms a huge sac, in which a fluid thrill may be felt. At the same time, especially in acute hydramnios, where the abdominal girth has not been able to increase gradually, compression of the abdominal organs and of the ureters, increasing dyspnœa, vomiting, cyanosis, and lumbo-abdominal pains show themselves (Chauffard). The fœtus dies in the proportion of 23 per cent. (Bar). The accouchement is often premature, and takes place under the worst conditions.

In the new-born child syphilis is but the continuation of the intra-uterine disease. In some cases the child is born with syphilitic cachexia (extreme wasting, palmar and plantar pemphigus), and death supervenes in a few hours. In other cases the new-born child appears to be in good health, and syphilis of the liver, like all the other forms of early hereditary syphilis, appears during the first months of life. Syphilis of the liver is generally accompanied by coryza, mucous and cutaneous syphilides, fissures of the umbilicus, anus, or lips, eruptions on the skin, etc. The hepatic or hepato-splenic lesion may be so pronounced that a spleno-hepatic form of heredo-syphilis has been described. The skin is bistre-coloured or yellowish; the digestive troubles are constant; the loss of flesh is rapid; and the liver is enlarged, smooth, and tender on pressure. The belly is enlarged and



streaked with veins. Tympanites is sometimes accompanied by ascites. The spleen is enlarged and painful. The prognosis is very grave, but recovery may occur.

The **diagnosis** of hepatic syphilis is easier in the new-born child than in the adult, because the customary symptoms of early heredo-syphilis are usually present.

**Pathological Anatomy.**—The description of the syphilitic liver in the adult differs from that applicable in the foetus or in the new-born child. This anatomical difference is the result of variation in the mode of infection. In the adult the lesion is disseminated in the liver, following the hepatic arterioles, which are attacked with arteritis and peri-arteritis, and ending by a more or less slow process in sclero-gummatous tissue. In the foetus, on the contrary, the liver immediately receives a portion of the blood from the placenta, and is infected as a whole. "It sustains the first attacks of the infection, and thus reacts more surely to congenital syphilis" (Chauffard). We find in a syphilitic child stillborn or dying soon after its birth from hepatic syphilis the following lesions: The liver has preserved its form and its smooth appearance, because there is as yet no fibrosis. The organ is larger than in the normal condition, hard, and elastic. The habitual red-brown tint has given place to a grey-yellowish colour, like flint, and on section of the parenchyma we see little whitish granulations (microscopic gummata) like grains of semolina (Gubler). The lesions are usually found in the left lobe and the sharp edge of the liver.

This syphilitic hepatitis is a young cirrhosis. The embryonic tissue is not yet fibrous, but, histologically, diffuse and nodular lesions are noticed. Hutinel and Hudelo have shown that this periportal cirrhosis at its commencement follows the vessels, and with them penetrates the hepatic lobules, where it becomes diffuse. The perihepatitis is not as marked as in the adult, and the ascites is often blood-stained.

**Late Hereditary Syphilis.**—I must specially mention the late form of hereditary syphilis. The gummatous, amyloid, and cirrhotic lesions in the liver may be the result of hereditary syphilis appearing in childhood and adolescence or adult life. Fournier has collected twenty-five cases. When the lesion is cirrhotic, the liver is generally enlarged, indurated, and deformed. Jaundice is rare, but ascites is constant, and sometimes it is the symptom which gives the alarm. This syphilitic cirrhosis is often associated with hypertrophy of the spleen and with renal lesions accompanied by albuminuria.

We can understand the difficulties of diagnosis in syphilitic cirrhosis if a proper clue is not given by the antecedents and by the reminders. We may look upon the case as one of common cirrhosis—a disease usually incurable—when we are really face to face with syphilitic mischief, which

often recovers. Too much attention cannot, therefore, be given to the pathogenic diagnosis, and too much care cannot be devoted to the tracing of acquired or hereditary syphilis, which may assist in finding the cause of the disease.

The course of the disease may help in diagnosis. Syphilitic cirrhosis most resembles Laënnec's atrophic cirrhosis. Ascites, œdema of the lower limbs, meteorism, and collateral circulation exist in both cases. In syphilitic cirrhosis the liver is more enlarged, and the ascites and the other symptoms develop, as a rule, much later.

In hereditary syphilis it is very necessary to recognize the stigmata which may help in diagnosis. They include dental changes, diffuse keratitis, natiform cranium, bosses on the forehead, nasal deformities with or without ozæna, swelling and incurvation of the tibia, cicatrices on the skin, sarcocele, and glandular enlargement.

The **treatment** is that of tertiary syphilis. Injections of biniodide of mercury, with or without iodide of potassium, must be employed as soon as the cause of the disease is known or suspected.

## XV. HYDATID CYSTS OF THE LIVER.

**Evolution of the Hydatids.**—The mature cyst is formed of an envelope containing clear watery liquid, in which float daughter cysts, echinococci, and hooklets. We must now study the life-history of the hydatid in detail.

There is a little ribbon-like worm, called *Tænia nana* (dwarf), or *Tænia echinococcus*, measuring 4 millimetres in length, reaching its complete development in the intestine of the dog. This little worm is composed of a head like that of the echinococcus and of three segments. The head is armed with a double row of strong hooklets and with four suckers. The last of these segments contains a branching ovary, a lateral genital orifice, and several thousand embryos, or eggs. These segments are excreted in the dog's dejecta, and break up, liberating the eggs. The eggs attach themselves to vegetables or plants, and may be swallowed by man or by a herbivorous animal, with the following result: The egg introduced into the digestive passages has a very thick wall, which softens, setting free the embryo. The hexacanthic embryo, provided with sharp spicules, perforates the tissues, and is probably carried by the blood of the portal vein to the liver, in which it is usually arrested. It loses its hooklets, and secretes from its posterior portion an envelope, in which it becomes encysted, forming the hydatid pocket, composed of an internal layer, which is but the transformation of the embryo (germinative membrane), and of an external layer, which is the product of the secretion. A clear and transparent liquid accumulates in the cavity.



At a later stage the cyst surrounds itself with a new wall or pericystic layer. This is a borrowed wall, which forms no part of the cyst, and which is due to the irritation of the neighbouring hepatic connective tissue. Let us next consider the cyst at this advanced period of its evolution.

As I have said, the pericystic layer does not form an integral portion of the cyst, but is a connective fibroid membrane, produced by the irritant action of the parasitic vesicle. This membrane may be  $\frac{1}{4}$  inch in thickness. It is covered with a nutrient network of vessels derived from the hepatic artery and the portal vein. It is in this layer that the phenomena of calcification and suppuration take place. This fibrous envelope is intimately connected with the parenchyma of the liver, but it can be separated from the cyst. The isolated cyst appears in the form of a soft whitish and trembling spheroidal mass. I have already explained the formation of the two membranes which make up the wall of the cyst, and I shall now follow them in their growth.

The external membrane of the cyst has a thickness of from 1 to 3 millimetres, and looks like half-cooked albumin. It is opaline, semitransparent, and formed of several stratified, non-vascular, amorphous layers. These layers have been compared to the leaves of unequal thickness formed by the edges of an album. When cut, they curl up like elastic membranes, though the microscope shows no trace of any figured element. As this formation is absolutely peculiar to hydatid cysts, it is impossible to make a mistake, and the diagnosis is fixed when a shred is found in a pathological liquid. Above this membrane we find a granular, raspberry-like, **germinative** membrane (Giraldès), which gives birth to vesicles and to echinococci.

The vesicles begin as sessile buds, which become pedunculated and filled with liquid. They enlarge, become detached, and fall into the interior of the cyst (daughter vesicles). The daughter vesicles in the primary cyst are more or less bulky, and some of them contain a third or fourth generation. Their structure is identical with the parent cyst. As long as the germinative membrane of the hydatid cyst produces vesicles and not echinococci, the cyst is said to be acephalocystic. When the germinative vesicle produces echinococci, the cyst is said to be fertile. This fertility may be shown by the iodophilic reaction. With iodized gum (Loeper) the germinative membrane, the heads of the echinococci, and the hydatids are seen to be filled with glycogen when they are alive; they have none when they are dead.

The **echinococcus** (ἐχῖνος, hedgehog; κόκκος, grain) at first resembles a whitish granulation, which protrudes on the internal surface of the cyst, and becomes pedunculated. It is formed of a head with four suckers and a double crown of twenty to thirty hooklets. The head is separated from the body by a neck. The body is rounded, and ends in a process, fixing

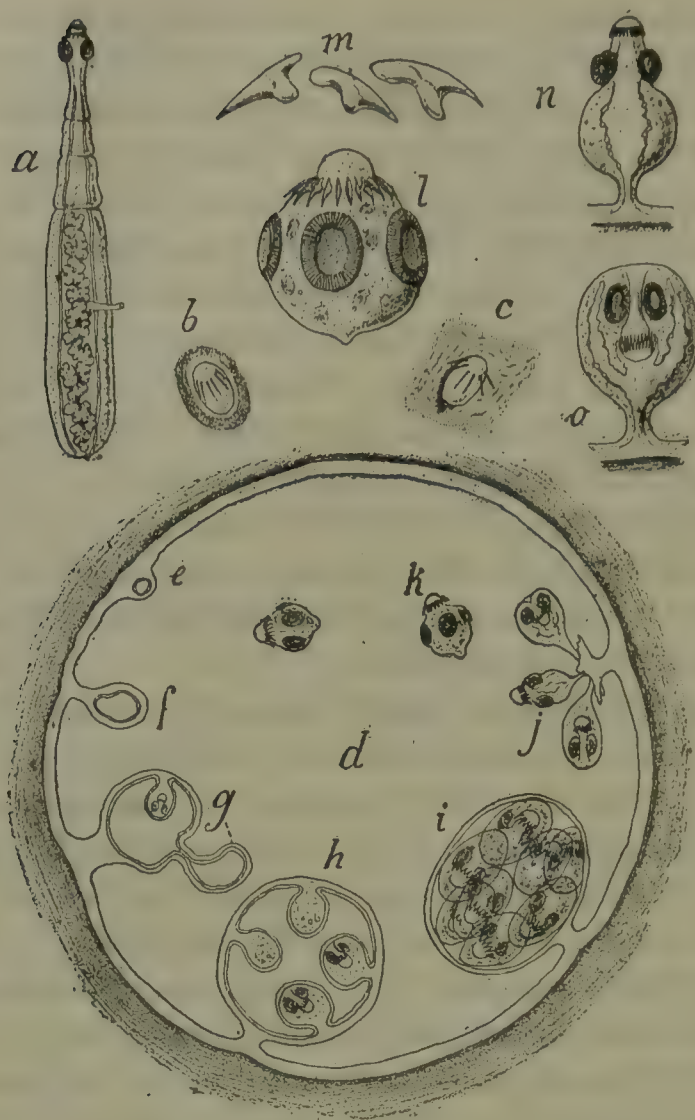


FIG. 49.

(a) *Adult Tænia nana*, or *echinococcus*. The head is provided with a double crown of hooklets and four suckers. The last segment contains an ovary, with a lateral genital pore.

(b) *Egg*; the granular shell encloses the *hexacanthic embryo*.

(c) The ingested *hexacanthic embryo*, perforating the tissues by means of its hooklets.

(d) *Hydatid cyst*, formed by the distension of the embryo, fixed and encysted in the liver.

(e) Under the pericystic stratified membrane, the true wall, or proligerous membrane, gives birth to sessile buds, which form secondary vesicles, or daughter cysts.

(f) Vesicle, without echinococcus, such as the acephalocystic cyst produces.

(g) Vesicle, with an echinococcus and a granddaughter cyst.

(h) Hydatid, producing scolex, each of which has the head of an echinococcus.

(i) Hydatid full of piled-up echinococci.

(j) Freeing of the echinococci by the bursting of a hydatid daughter cyst.

(k) Free scolex in the mother hydatid.

(l) Scolex of echinococcus, with its four suckers and its crown of hooklets.

(m) Separate hooklets.

(n) The echinococcus, still pedunculated and adherent to the fertile wall; the tube is evaginated.

(o) The same, with the tube invaginated.



the animal to the germinative membrane. When this process breaks, the animal falls into the liquid of the cyst. The liquid is transparent, like spring-water, whence the name of these cysts (*ὑδαρίς*, from *ὑδωρ*, water). The quantity of the liquid varies from a few ounces to several pints. Under the microscope hooklets are visible. They contain chloride of sodium, but no albumin. The presence of albumin in the liquid indicates, according to Gubler, the death of the hydatids.

The presence of glycogen indicates activity on the part of the hydatid. "The iodine reaction in the scolices of the aspirated fluid in the internal layer of the young hydatids, or in the germinal membrane of the cyst, shows that the hydatid is active, even though the fluid is turbid and the vesicles are withered" (Loeper).

**Pathological Anatomy.**—The pathological anatomy of the cysts in the liver is in part concerned with the evolution of the cyst and with its various modes of termination, such as perforation, rupture, fistulæ, and divers transformations. The pathological anatomy will, therefore, be given during the course of the description.

I must, however, mention hypertrophy of the liver due to compensatory hyperplasia. I have already discussed this question under the Enlarged Cirrhotic Liver of Alcoholic Patients, and the same question, slightly modified, presents itself in dealing with hydatid cysts of the liver. Several writers have studied this vicarious hypertrophy which attacks the cystic lobe, but more especially the unaffected lobe, and I know of no more complete example than the one related by Chauffard.

In this case the patient died, and the liver, which contained a hydatid cyst, weighed 200 ounces before and 90 ounces after evacuation of the cyst—that is to say, about 35 ounces more than a normal liver. The left lobe weighed 40 ounces. The compensatory hyperplasia had, indeed, outstripped the object, and it was really hepatic tissue of good quality which had thus been regenerated. In spite of the presence of an enormous hydatid cyst, the normal action of the liver was more than assured. As a matter of fact, says Chauffard, in all this hyperplastic tissue there was an absence of cirrhotic lesions, the hepatic cells being well preserved, and their protoplasm, with the nucleus, having a normal aspect. The trabecular ordination was, however, modified, and the hepatic parenchyma recalled a foetal liver at the seventh month of intra-uterine life. "There was the image of an almost full cellular parenchyma." The hepatic cells were grouped in small radial columns, let into the connective tissue around the portal spaces, "like the radiating veins of a palm-leaf around their pedicle." Such is the compensatory hyperplasia found in a liver containing a hydatid cyst; it may be likened to the hyperplasia in the enlarged alcoholic liver, in the malarial liver, and in the tubercular liver, where it forms foci of nodular hyperplasia.

**Symptoms.**—During the first period of its evolution, sometimes for months, as long as the cyst has not reached a fair size, the parasite does not reveal its presence by any marked symptom, and even when it has become large, some patients only complain of enlargement of the abdomen. And

yet before the cyst provokes serious troubles, we may find symptoms which are frequently unrecognized, but which I consider very important, because they give the diagnosis at a period when the hydatid tumour is but slightly apparent. These symptoms are pain in the right shoulder, the appearance of urticaria, distaste for fatty food, and the development of pleurisy on the right side.

Pain in the right shoulder, so frequent in some affections of the liver, is almost constant in the case of hydatid cyst, especially when the cyst approaches the upper surface of the liver.

Urticaria is a curious symptom. I am not speaking of urticaria after aspiratory puncture, but I allude to nettle-like eruptions appearing during the evolution of the hydatid cyst, of which I have been able to collect five cases.\*

I have several times noticed another early symptom—distaste for fatty substances. In one patient this distaste was present for two years, and was so great that he had repeatedly ordered less butter and fat to be used in the kitchen. I have quoted similar cases elsewhere.† A woman suffering from a hydatid cyst had from the first a distaste for fatty foods. After meals she suffered from regurgitations, and, without any attack of nausea, her mouth became filled with the fatty portions of her food. In another patient this regurgitation of fatty matter was so marked at the outset of the disease that she rejected the fat immediately after meals. The expectoration had on paper the look of spots of oil, and she compared her saliva to what is commonly called “the eyes on the soup.” The symptom existed for several weeks, to the exclusion of any other digestive trouble, and then disappeared with the progress of the disease.

**Secondary pleurisy**, either dry or with effusion, which sometimes accompanies tumours of the abdominal organs (kidneys, spleen, or liver), is equally likely in hydatid cysts of the liver. It has been noticed by several writers, and I have collected six cases. When the cyst is large, and especially when it is seated on the convex surface of the liver, we can understand how the inflammatory process spreads gradually from the peritoneum to the pleura; but there are cases in which the cyst is deeply seated and of small size, when pleurisy appears as a forerunner.

Boulay and I saw a patient suffering from a cyst of the liver, in whom the other symptoms had been preceded by three slight attacks of pleurisy on the right side.

It is not impossible for the inflammatory process to be carried to the pleura by the lymphatics (Schweiger-Seidel, Recklinghausen), since the serous cavities of the pleura and of the peritoneum communicate by means of lymphatic vessels and spaces. Further, if the inflammatory process in some cases is able to spread from the peritoneum to the pleura, the converse is true, as cases of pleurisy of the diaphragm, followed by peritonitis, show.

\* Dieulafoy, “Les Kystes hydatiques et leur Traitement” (*Gaz. Hebdom.*, 1877, No. 30).

† Dieulafoy, “Traité de l’Aspiration,” p. 67.



The development of pleurisy seems sometimes to hasten the growth of the cyst (Verneuil). A patient has a small cyst. In the meantime he gets pleurisy, and the cyst (*pars minoris resistentiæ*) undergoes an increase in size.

Epistaxis, dragging and heaviness in the pit of the stomach and in the hypochondrium, are symptoms belonging to the early stage in the growth of the hydatid cyst.

When the cyst becomes large, it follows two principal directions. It sometimes projects on the convex surface of the liver, and pushes up the diaphragm, but does not depress the liver much. It protrudes into the thoracic cavity, and simulates an effusion into the right pleura. Sometimes it forms an abdominal tumour. The enlargement of the organ is not uniform, as it is in hypertrophy. We find a more or less limited tumour, which often protrudes into the epigastric region, or in other cases raises the false ribs and dilates the last intercostal spaces on the right side. Sometimes the tumour appears in the right hypochondrium, assumes an elongated or bilobed form, encroaches on the linea alba, after the manner of a pedunculated tumour, and reaches the left hypochondrium. The plates in Frerichs' work show, even if the situation of the tumour only be considered, how cysts of the liver have been mistaken for tumours of the peritoneum, spleen, or ovary. Palpation and percussion give the limits of the tumour, which is smooth, uniform, and resistant, and which sometimes gives the sensation of fluctuation. Unless there is some complication (perihepatitis), the hydatid cyst develops without pain.

The **hydatid thrill** (Boinet), which is said to be due to the elasticity of the walls of the cyst, or to collision of the hydatids when percussion is performed, is very rare. Ascites and jaundice are generally absent, unless there are complications, such as compression of the portal vein or of the bile-ducts, or the development of catarrhal jaundice.

The veins of the abdomen are slightly dilated, and the collateral circulation is only seen in compression of the portal vein.

**Course—Duration—Termination.**—Left to itself, the hydatid cyst of the liver may grow during a period of two to six years without causing serious mischief, but at a more or less distant date cachexia appears. In this case the general condition of the patient becomes bad, the appetite is lost, digestion is painful, the loss of flesh is considerable, and there are attacks of hæmorrhage, epistaxis, or metrorrhagia. This hydatid cachexia may appear early, as I have several times observed. It may be said that the patients become intoxicated by the hydatids.

The dyspnœa makes constant progress if the cyst pushes up the diaphragm and interferes with the function of the lungs. Some patients experience pain in the abdomen, the loins, or the thorax. They are unable

to find a comfortable position in bed, and suffer from distressing insomnia. In many cases the cyst suppurates, and causes perforation. Even the general symptoms have become marked. The causes of suppuration in hydatid cysts have been elucidated by Chauffard and Vidal. Their conclusions are as follows :

The liquid in the hydatid is aseptic, but it is a medium favourable to the growth of pyogenic microbes. The absolute impermeability of the hydatid membrane to micro-organisms protects the cyst from infection, the membrane filtering the organisms. The hydatid membrane, however, allows soluble substances, such as sublimate, iodide of potassium, and pyocyanine, to dialyze into the vesicle. The agents of suppuration can, therefore, only penetrate into the interior of the cyst by effraction when a path has been opened by pericystic lesions. It is through this vascular pericystic pocket that the pyogenic invasion occurs. Pericystitis causes necrobiosis of the subjacent hydatid membrane, and the enemy enters the fortress. The cyst becomes infected. The pyogenic agents may get into the pericystic pocket through the biliary passages (ascending angiocholitis) or through the bloodvessels and the lymphatics. The pyogenic agents are probably those of ordinary suppuration, but it is remarkable that in some cases the pus is sterile.

### Gaseous Cysts—Pyopneumohydatid.

In some cases gas forms in the suppurating cyst. It is generated *in situ*, and is due to the suppuration. The following case occurred at the Hôtel-Dieu :

A woman had complained for some time of acute pain in the right hypochondrium. In the right diaphragmatic region there was an oblong bulging, with œdema and collateral circulation. Percussion gave a tympanitic note. Below the bulging, the liver dullness descended as low as the umbilicus. The evening temperature was 104° F., and the dyspnœa was severe.

At the base of the chest, on the right side and behind, percussion gave a tympanitic note, with *bruit d'airain*, and auscultation yielded metallic breathing. No succussion splash. What was the diagnosis ? The signs found were those of a gas-containing cavity, occupying, behind and on the right, the base of the thorax, and in front the hepato-gastric plane. Pneumothorax appeared to be the lesion, but it was necessary to ascertain the relation of the collection to the diaphragm. Was the collection above or below the diaphragm ?

The patient did not cough, and there was no sputum. Tubercular pneumothorax was not admissible, more especially because it would not explain the bulging in the gastro-hepatic region. The mischief was below the diaphragm.

There was no history of gastric or duodenal ulcer, which might have perforated and given rise to subphrenic pyopneumothorax. Subphrenic empyema consecutive to appendicitis was out of the question, because the patient had shown no symptoms of appendicitis, and, as I have shown under Appendicular Pleurisy, appendicitis, which often causes putrid effusion with gas in the thorax, does not provoke the formation of gas in subphrenic abscesses. The patient finally said that, some twenty years before, Le Dentu had punctured a hydatid cyst, and drawn off turbid fluid.



Mauclore opened the abdomen, and exposed the hydatid cyst in the liver. The tumour was situated in the upper part of the right lobe; it had pushed down the liver, and had extended towards the thorax. As soon as the thickened fibrous wall was opened freely, foul-smelling gas, pus, and fragments of dead hydatids escaped. Some pus was removed in sterile pipettes for examination.

The autopsy showed that the cyst had no connection with the intestinal tract; the gas must, therefore, have formed *in situ* from fermentation in the pus. Bacteriological examination of the pus showed the reason. The formation of the gas was due to a streptococcus and various anaerobic germs.

Hallé and Bacaloglu found aerobic and anaerobic organisms in the pus from a suppurating hydatid cyst of the liver. The pus contained the streptococcus and the *Bacillus coli* in small numbers, and a great abundance of anaerobic microbes (*Staphylococcus parvulus* and *Bacillus fragilis*). Gas was not present, the suppuration being foetid, but not putrid.

In a similar case to the one I have described, Gilbert and Weil found the *Bacillus coli* as the only organism present. In Lippmann's case the pus contained various anaerobic micro-organisms. Dévé has described two cases of gas formation in suppurating hydatids of the liver.

Let us next consider the results of suppuration in the cyst, which may be due to injury or some intercurrent disease.

**1. Opening of the Cyst into the Air-Passages.**—Cysts of the convex face of the liver have a special tendency to open into the air-passages. The walls of the cyst become inflamed, the hydatids die, the liquid becomes purulent, and adhesions are formed between the cyst and the diaphragm, between the diaphragm and the pleura.

This inflammatory process is generally ushered in by rigors, fever, pains in the region of the liver, and on auscultation the rub of dry pleurisy is sometimes heard. The rupture of the cyst may take place under various conditions. If the layers of the pleura are adherent (this is most often the case), the cyst opens into the bronchi, and the pus is coughed up. The vomica is preceded and accompanied by sharp pain, attacks of coughing, attempts at vomiting, and fits of suffocation. The patient brings up streams of sero-purulent, foetid liquid, which is sometimes mixed with blood, and in which shreds of hydatid membranes are found. The presence of bile in the vomica is of evil omen. During the following days the liquid continues to come out by the broncho-hepatic fistula, and in the end a cure may be effected, though it is far more common for the patients to be carried off by some complication (pulmonary gangrene, hæmoptysis), or to succumb from the results of interminable suppuration. Auscultation of the thoracic region generally yields the signs of partial pneumothorax. When the adhesions have not united the two layers of the pleura, the cyst empties itself into the pleural cavity, and causes grave mischief.

In some cases the hydatid cyst empties into the bronchi through a narrow

fistula. The pus is then brought up in small quantities. It is usually foul-smelling and blood-stained. The narrowness of the opening allows neither hydatids nor membranes to pass. The diagnosis is, then, difficult, and the prognosis is grave.

**2. Opening of the Cyst into the Digestive Tract.**—Here, as in the preceding form, the opening of the cyst is preceded by an inflammatory process, which causes suppuration in the cyst on the one hand, and adhesions between the cyst and the neighbouring organs on the other. This process may be latent, though it is more often accompanied by fever, pain, and symptoms of peritonitis. Rupture of the cyst into the intestine generally takes place in the colon, and the pain is exceedingly severe at the moment of perforation. If the opening of communication is large enough, the liquid and the membranes are emptied into the intestine, and are passed *per anum*. The hepatic tumour grows smaller, the discharge continues for some time, and a cure may take place (twenty-seven times in thirty-two cases—Letourneur). If the orifice is insufficient, a kind of chronic diarrhoea is the result, and the indefinite suppuration wears out the patient. Rupture of the cyst into the stomach is extremely rare, and is generally followed by death. Rupture into the duodenum is unfavourable. The cyst is got rid of both by the stomach and by the intestine.

**3. Opening of the Cyst into the Peritoneum.**—Rupture of the cyst into the peritoneum is not always spontaneous. It is sometimes the result of traumatism, and causes fatal peritonitis if the cyst is infected. If the liquid of the cyst is aseptic, peritonitis may be absent, and recovery has been noted in eleven out of thirteen cases (Finsen). The mixture of bile with the discharged hydatid liquid is not serious, provided the bile is not infected. An eruption of urticaria has often been noticed after rupture of the cyst. To this curious symptom I shall refer later. In some cases the vesicles discharged into the peritoneum have continued to live and to proliferate.

**4. Relation of the Cyst to the Bile-Ducts.**—Ulceration of the bile-ducts close to the cyst and passage of the bile into the cyst may be favourable, because the bile may kill the hydatids and bring about recovery. The cyst may open into the gall-bladder or into the common, hepatic, cystic, or intra-hepatic ducts. Rupture of the cyst into the common duct is serious. If the hydatid is not large, it may pass through the duct, causing hepatic colic, and cure is possible. If the membranes block the duct and give rise to chronic obstructive jaundice, dilatation of the ducts on the proximal side of the obstruction and acute purulent angiocholitis follow.

5. I shall quote, as quite an exception, the opening of the cyst into the pericardium or the vena cava, ulceration of the abdominal wall, and opening of the cyst in the neighbourhood of the umbilicus, or in an intercostal space.



**6. Spontaneous Cure.**—Some cysts (33 per cent.) recover spontaneously, without reaching a large size—for example, those discovered by chance post mortem. The cure is brought about by the death of the echinococci. When the hydatid dies (no matter what is the cause of death), the liquid loses its transparency, becomes albuminous, and is absorbed. The hooklets, salts of lime, epithelial cells, and crystals of hæmatoidin of a biliary origin form a small degenerated caseous mass. The pocket of the cyst becomes fibrous, contracted, and infiltrated with calcareous salts, and the cyst thus transformed becomes inoffensive.

**Diagnosis.**—A smooth, painless, resistant bulging, which develops slowly in the liver without fever, jaundice, and ascites, can only be a hydatid cyst. The diagnosis is not always so simple. Some pedunculated hydatid cysts protrude into the left hypochondrium, and simulate a cyst of the spleen. Others descend into the iliac fossa, and simulate a cyst of the ovary. Others protrude towards the diaphragm, push it back, encroach on the thoracic cavity, and at first sight simulate effusion into the pleura. These possible causes of error must be remembered in order to avoid mistakes. The enlarged liver of leucocythæmia and of malaria is characterized by general and uniform swelling, which does not resemble the more or less limited tumour of the cyst. Besides, the ætiological conditions and the concomitant symptoms would suffice to remove all doubts. **Hypertrophic cirrhosis**, with uniform enlargement of the liver and chronic icterus, cannot be taken for a hydatid cyst; but the opposite mistake has been made because certain cysts of the liver, accompanied by jaundice, may closely resemble hypertrophic cirrhosis. There is, however, the difference that in hypertrophic cirrhosis the spleen is likewise hypertrophied, and the jaundice is contemporaneous with the onset of the disease.

**Secondary cancer of the liver** is distinguished by its rapid growth, and by the indurated bosses of the tumour. Primary massive cancer does not cause bosses, but offers a ligneous hardness to the touch, and the general malnutrition of the patient is not to be compared with the excellent health of an individual with a hydatid cyst. Cysts of the **kidneys** may project into the hypochondrium so as to simulate a cyst of the liver. In some very rare cases the hydatid cyst, opening into the peritoneum, has been mistaken for ascites.

The diagnosis between pleural effusion and a cyst on the convex face of the liver is often very difficult, and a cyst has often been punctured under the impression that the pleural cavity was being penetrated. Percussion, auscultation, the form and limits of the dullness, give imperfect information. The history of the illness must be investigated, and the deformity of the thorax must be carefully studied. As a matter of fact, cysts of the liver do not generally simulate large effusions into the pleura (4 or 5 pints), in

which the dullness reaches to the upper level of the thorax. They simulate, rather, the average effusions of 2 or 3 pints, which cause dullness, with its upper limit at the spine of the scapula. These average effusions do not cause enlargement of the hypochondrium or bulging and enlargement of the intercostal spaces, such as are common in hydatid cysts.

In hydatid cysts of the liver or of the spleen, and also in all tumours of the hypochondrium, I would advise the following mode of exploration: The patient is first examined lying on a couch; he, then, stands stripped, in the upright position, with the arms extended. Examination from the front and the back and comparison of the healthy and diseased sides show any deformity on the diseased side.

**Eosinophilia.**—In cases where the diagnosis of hydatid cyst is doubtful, do we find signs of irritation produced by the echinococcic toxine on examination of the blood? Loeper has published some interesting work on this point. Leucocytosis is generally slight or absent. The number of polynuclear cells is practically normal, but the eosinophiles are usually present in increased proportions. This observation shows that the hydatid has an affinity for the eosinophile. If we examine the liver, we find in the connective tissue and in the intercellular spaces many eosinophile cells. The number is most marked in the neighbourhood of the cyst, and the eosinophiles abound in the fibrous shell, through which they pass, insinuating themselves in the interstices of the cyst-wall, and falling into the cavity.

Eosinophilia is the most marked blood reaction. In many cases it indicates activity on the part of the cyst, because it disappears after operation. As it is not constant, it cannot be said to have an absolute value. On the other hand, eosinophilia, even when abundant, does not always necessitate the diagnosis of hydatid cyst. Certain tumours of the spleen, certain lesions of the pleura or of the lung may be accompanied by an increase in the number of eosinophiles. The examination of the blood affords, therefore, a **presumption**, but not a **certainty**, in the diagnosis of the hydatid.

**Reaction by Fixation.**—Weinberg and Faure have recently (1909) applied Bordet and Gengou's reaction—fixation of the complement—to the diagnosis of hydatid cysts. By making use of the hydatid fluid of the sheep, desiccated and diluted in alcohol, these authors obtained results which are very important in diagnosis. The technique of the reaction is carried out after the method described in the chapter on typhoid fever. This reaction disappears rapidly after the surgical extirpation of the cyst. Its persistence indicates that one or more cysts remain in the subject. Let it be added, nevertheless, that the reaction by fixation is no more absolutely constant than the eosinophilia.



When suppuration occurs in the cyst, we find more or less marked polynuclear leucocytosis.

The prognosis of hydatid cysts of the liver is very serious, on account of mishaps of rupture of the cyst, infection, or opening into the thorax, peritoneum, or bile-ducts. We shall see, nevertheless, that the treatment may remove these grave dangers, and make the prognosis favourable.

**Treatment.**—Since 1870\* the treatment of cysts of the liver by aspiration has been much practised, and though this operation does not succeed in all cases, experience has proved that in some unilocular, non-suppurating cysts puncture by means of the aspirator is harmless, and is sometimes followed by cure.

**Description of the Operation.**—The choice of the aspirator is a matter of indifference. **The important point is the choice of the needle.** I use a No. 2 needle, which measures only  $1\frac{1}{2}$  millimetres in diameter, and I put aside the trocar, which offers no advantages. The needle having been sterilized, and all aseptic precautions having been taken, the preliminary vacuum is made in the aspirator. The patient is placed on his back, and the operator introduces the needle at the most prominent part of the tumour. The corresponding cock of the aspirator is then opened, and the liquid from the cyst flows into the bottle, and as the cyst is emptied care is taken to push the needle a little deeper, because the level of the liquid in the tumour is being gradually lowered. If during the course of the operation the flow of the liquid stops suddenly, or if a piece of hydatid membrane is thought to obliterate the needle, the needle is left *in situ*, and a new puncture is made. Care must, however, be taken not to knead the tumour, so as to favour the outflow of the liquid. Care must also be taken not to percuss the tumour or to make the patient sit up, in order to ascertain the diminution or the disappearance of the liquid. These manœuvres are wrong. They may favour the issue of a few drops of liquid, and become the cause of complications. These details are of importance.

After the cyst is emptied, the needle is withdrawn, care being taken to leave it during the withdrawal in communication with the aspirator, as by this means the needle cannot leave a single drop of liquid in the peritoneum. After the operation, a bandage, previously applied, is gradually tightened. The patient must remain still on his back for some hours. He may get up the day after the operation.

The operation thus performed is merely a harmless needle-prick. In some cases of unilocular non-suppurating cysts cure is obtained by one aspiration. It will be asked what becomes of the pocket of the cyst. It is probable that, in part, it undergoes fatty degeneration, as in spontaneous

\* Dieulafoy, *Gaz. des Hôp.*, 1870, and "Traité de l'Aspiration," 1873, p. 51.

cure. This happy termination is the more frequent, the earlier the cyst is attacked. Later, the cystic pocket, as it grows older, becomes vascular and thickened, and the chances of success diminish.

**After-Effects of the Operation—Urticaria.**—The after-effects are so slight that recovery is practically immediate. Certain urgent symptoms may, however, show themselves, and it is necessary to remember them. We see patients who a few hours after the operation suffer from dyspnoea, nausea, and sometimes hiccough, vomiting, or fever, and we naturally fear the onset of acute peritonitis. Itching, however, soon supervenes in different parts of the body, and an attack of urticaria appears.

When I published my first remarks on urticaria after puncture of cysts of the liver, the fact had passed unnoticed in France, but cases soon multiplied;\* I have collected about fifty. It is only right to say that this fact did not pass unnoticed in Denmark, and Finsen, when reporting several examples of urticaria following **rupture** of hydatid cysts into the peritoneum, points out that puncture of the cyst may lead to the same result. The urticaria shows itself in somewhat different forms. Sometimes it is not accompanied by any other symptom save slight itching that lasts a day or two. At other times the urticaria is preceded and accompanied by the general symptoms above mentioned. In some cases the urticaria invades the mucous membranes of the mouth or of the pharynx. The fever, nausea, and vomiting generally become severe, and last two or three days. Sometimes the urticaria affects certain portions of the body. I have seen a case in which it was limited to the right side.

These symptoms closely resemble those of mussel-poisoning. In both cases it is a question of intoxication. The hydatid fluid is toxic, and may contain substances that belong to the class of ptomaines analogous to the mytilotoxin of poisonous mussels. According to Viron, the hydatid liquid contains an albuminoid substance something like toxalbumin.

Aseptic hydatid liquid, when injected under the skin with a Pravaz syringe, may produce urticaria. The toxicity of the hydatid liquid is thus clearly established, and explains the nettle-like eruptions which may supervene during the growth of the cyst, and which I have pointed out as revealing signs. It perhaps explains the early hydatid cachexia of certain patients in whom the general symptoms are more pronounced than the local troubles. In some cases puncture of a hydatid cyst of the liver has caused fatal complications.

\* Dieulafoy, *Gaz. des Hôp.*, 1870; "Traité de l'Aspiration des Liquides Morbides," 1873. Hayem et Ferrand, *Soc. Méd. des Hôp.*, 1874. Bussard, *Gaz. des Hôp.*, 1875. Feytaud, *Th. de Paris*, 1875. Raynaud, Verneuil, Legroux, *Soc. Anat.*, 1875. Archambault, *Union Méd.*, 1876. Lereboullet, *Gaz. Hebd.*, 1876. Bradbury, *British Medical Journal*, 1874. Neisser, "Die Echinococcenkrankheit," Berlin, 1877.



Moissenet : A man suffering from hydatid cyst of the liver was operated upon, the puncture being made with a capillary trocar and the discharge of the liquid being stopped at 30 ounces. Five minutes later, says Moissenet, the patient was seized with syncope. Two hours afterwards there was a severe rigor, with pallor of the face, pinched nose, hollow eyes, and hiccough, nausea, copious green vomiting, and yet there was no pain on pressing the belly. The symptoms became worse and worse : the pulse stood at 125 ; the extremities grew cold ; the facies became more marked ; and the patient succumbed during the night. The post-mortem revealed a hydatid cyst, as large as an adult head, containing clear liquid and daughter vesicles.

Martineau : Aspiratory puncture was performed on a patient suffering from hydatid cyst of the liver. A colourless liquid flowed from the cannula ; after a few grammes had passed, the discharge ceased, and did not appear again, in spite of a change of place of the cannula. On withdrawal Martineau found that it was obstructed by hydatid debris. A few minutes afterwards the patient was taken ill with intense dyspnoea and vomiting. Respiration stopped, the pulse became imperceptible, and the patient fell into a state of syncope, dying in twenty-four hours. At the post-mortem examination mucus obstructed the bronchi. Old pleural adhesions, pericarditis, and old mitral endocarditis were found, as well as two hydatid cysts in the liver, one of which showed the puncture. There was nothing in the peritoneum.

Bryant : In a man suffering from hydatid cyst of the liver a trocar, not larger than a silver probe, was inserted to a depth of about  $3\frac{1}{2}$  inches to the right of the median line. Nine ounces of a clear non-albuminous liquid were withdrawn to diminish the tension. A few seconds afterwards the patient became livid, lost consciousness, and after vomiting two or three times, had an epileptiform fit. At the same moment the pulse stopped. Artificial respiration, the galvanic battery, and nitrite of amyl were tried, but the patient died in five minutes. The post-mortem examination was performed by Hilton Fagge. The peritoneum contained  $\frac{1}{2}$  pint of blood-tinged liquid. A hydatid cyst as large as a man's head occupied the posterior portion of the right lobe of the liver. There were no other visceral changes.

Chauffard : In order to confirm the diagnosis of a hydatid cyst of the liver an aspiratory puncture was made. A little liquid had just been withdrawn, when the patient was taken with sudden itching, loss of consciousness, and an epileptiform fit, with foaming at the mouth, and emission of urine and faecal matter. A few moments afterwards there was another epileptiform fit, with clonic movements, restlessness, and expectoration of whitish froth. In a few minutes the patient fell back exhausted. The skin had a cyanotic tint, the pulse grew weaker and weaker, collapse was imminent, the face was bathed in sweat, and at last fatal asphyxia supervened. The post-mortem examination revealed neither peritonitis nor fluid in the peritoneum. It was evidently a case of superacute hydatid intoxication.

We must try to explain these fatal complications occurring after puncture of hydatid cysts in the liver. One fact is certain—namely, that the passage of a few drops of hydatid liquid into the peritoneal cavity may cause benign or fatal intoxication. The most benign complication is urticaria. The grave and fatal complications have just been described. They all are due to the passage of the toxic liquid into the peritoneum. The treatment of hydatid cyst by puncture is not to be feared. Mishaps result from small punctures, which withdraw only a very small quantity of the liquid, because the cystic liquid under pressure makes a way for itself through the little opening left by the needle, is absorbed by the peritoneum, and gives rise to intoxication. As a general rule small punctures, and also incomplete or

exploratory punctures, must be avoided. This mode of investigation must never be employed to clear up the diagnosis of cysts of the liver. No matter how fine the aspiratory needle, we must not forget that it may leave an open channel for liquid which under pressure is ready to pass into the peritoneum.

**Summary.**—I have gradually given up the treatment of the cyst by aspiratory puncture for laparotomy. In cysts of the anterior surface of the liver median or lateral laparotomy should be performed. The postero-inferior cysts should be attacked by the lumbar incision. In subphrenic cysts the inferior edge of the thorax should be resected (Lannelongue). Finally, if the cyst reaches up into the thoracic cavity, it should be attacked through the pleura, with or without costal resection (Segond).

## XVI. ALVEOLAR HYDATID CYST OF THE LIVER.

**Pathological Anatomy.**—The alveolar hydatid cyst, also called multilocular echinococcus, is so rare that Carrière was only able to collect eighteen cases in his interesting thesis. The subsequent cases have been collected by Vierordt in 1886, and by Posselt in 1899. In this variety the hydatids are not collected in one mother vesicle, but are scattered through the parenchyma of the liver, forming more or less large groups. At the post-mortem examination the liver is found to be uneven and nodular. Over the hydatid tumours adhesive peritonitis is often found. The largest group is generally seated in the posterior part of the right lobe. A section shows that this cystic mass is composed of a thick shell and of a cavity containing purulent fluid and caseous débris that represent the parts of the cyst in a condition of retrogression.

The shell or envelope of the tumour is composed of a fibrous stroma, forming a number of pockets of various dimensions. These pockets contain little gelatinous and colloid bodies which are really hydatids. The vesicles of a multilocular hydatid have the same structure as the unilocular hydatid already described. Many of them are sterile, but some of them contain echinococci. These vesicles are in considerable quantity, and are of variable size. Some are not visible to the naked eye; others are as large as a hazel-nut. When the hydatids are confluent, the alveoli communicate with one another, and the tumour has the appearance of certain cancers. The hydatid bulges into the interior of the vessels, finally causing obliteration. The branches of the portal vein and of the hepatic artery (Ranvier), the bile-ducts (Frerichs), and the lymphatics (Virchow) are thus obliterated.

Many hypotheses have been formulated to explain the special disposition of the multilocular echinococcus. While some think that it is a case



of exogenous proliferation of the mother hydatid, which is said to give birth to daughter hydatids and to echinococci from its external wall instead of producing them from its internal one, others hold that they arise from a *tænia* different from the *Tænia echinococcus*.

**Ætiology.**—The alveolar echinococcus rarely develops before the twentieth year. Close contact with domestic animals, and especially with the ox (Posselt), is the chief cause of the affection. It is found in Southern Germany (Bavaria, Würtemberg), in the North of Switzerland, and has just invaded the Tyrol (Posselt). France (Carrière's patient was a Bavarian) has now been invaded. Railliet and Morot pointed out cases in animals in 1898. So far only two cases are known in man—viz., that of Bruyant (alveolar cyst of the liver), and that of Rénon (alveolar cyst of the lung and pleura).

**Symptoms.**—The alveolar hydatid cyst develops slowly, and its onset passes unnoticed for some while. Later the symptoms are peculiar, in that no one is pathognomonic. Thus, the liver presents nodules and bosses, as in **cancer**. Ascites is often present (seven cases out of thirteen, Frerichs) as in **atrophic cirrhosis**; but the cyst is more often accompanied by chronic jaundice (fifteen cases out of eighteen, Carrière), as in **hypertrophic cirrhosis**. The general health remains good; the appetite is excellent; the fæces are colourless from the onset of the jaundice; and the spleen is often hypertrophied. This diversity in the symptoms makes the difficulty in diagnosis obvious.

The course of the disease is very slow. Hæmorrhage and œdema are frequent at an advanced period. Death occurs in eight to eleven years after the onset of the disease.

## XVII. FATTY AND AMYLOID DEGENERATION OF THE LIVER.

1. **Fatty Degeneration.**—In the physiological condition, especially after meals, the liver contains a considerable quantity of fat. The hepatic cell can, indeed, manufacture fat from proteids, as is proved by the fat found in dogs, fed on an exclusive meat diet. The fat derived from digestion is stored in part in the hepatic cell, especially in the cells at the periphery of the lobule, where it awaits its ulterior elaboration. This accumulation of fat in the liver is even more abundant during pregnancy and lactation, on account of the nutrition of the foetus and the secretion of the milk; but in this case the fat is found in the central cells, close to the hepatic vein. In these different cases the fat contained in the liver is derived from outside and infiltrates the hepatic cell for the time being by pushing aside its protoplasm, but the cell loses neither its autonomy nor its functions—in a word,

there is fatty infiltration, but no degeneration. Not only does the liver elaborate fat, but it is also an organ for the excretion of fatty matter. In pathological conditions, there is fatty degeneration—that is to say, the fat is formed at the expense of the protoplasm of the cell. In consequence of chemical processes not fully understood, the hepatic cell loses its structure and properties, and becomes transformed into fatty tissue. It should be noted that these two varieties—infiltration and degeneration—often occur together.

**Steatosis** of the liver is produced by acute and chronic intoxications, phosphorus-poisoning being the most typical. Alcohol is a fat-producing poison. Arsenic and morphia (morphia habit) give the same result. Steatosis is sometimes caused by the organisms of the infectious diseases and their toxins: puerperal fever (Widal), typhoid fever (Legry), cholera (Hanot and Gilbert), and pneumonic infection (Pilliet). The toxins, no doubt, play the most important part in steatosis of the liver, associated with the infectious diseases. This hypothesis is proved in experiments where only toxins are present—viz., injection of cultures of the diphtheria bacillus (Roux and Yersin).

I have just reviewed the different intoxications which may cause steatosis of the liver, but there are circumstances in which it is associated with other pre-existing lesions (alcoholism, tuberculosis, and syphilis). Finally, in some cases the fatty degeneration is the consequence of an anterior process, such as cirrhosis, cardiac liver, or chronic jaundice.

Post mortem the following characteristics are met with: the liver is fatty, enlarged, and of a dead-leaf colour. Its tissue is soft, and on section fatty droplets exude. The proportion of the fatty elements is notably modified, and the liver contains 20 to 25 per cent. instead of 4 to 5 per cent. The bile is light in colour, on account of the changes in the protoplasm, which probably helps to make the bile pigment. The histological examination shows that the hepatic cell has lost its polyhedral form, becoming spheroidal. The nucleus of the cell is pushed aside to the periphery, the protoplasm has more or less disappeared, and the cell seems to be converted into a fatty mass.

The **symptoms** of hepatic steatosis are so obscure that the diagnosis can only be made from the ætiology. The liver is enlarged, and is not painful. Ascites, jaundice, and collateral circulation are absent. When the steatosis takes a rapid course, the symptoms of icterus gravis may result.

**2. Amyloid Degeneration.**—The amyloid liver is also called lardaceous, or waxy. The name “amyloid” is bad. It was employed by Virchow, who, relying on the iodine reaction, supposed the presence of an amyloid substance. The substance that infiltrates the cell, is of an albuminous nature, though it differs in several respects from true albumin.



The **causes** of amyloid degeneration are those of fatty degeneration. It is always a secondary process, and we may note, as having a special action in its production, chronic tuberculosis, especially of the bones, prolonged suppuration, syphilis, malaria, and leucocythæmia.

The amyloid liver is enlarged. Glisson's capsule is smooth, vitreous, and but rarely thickened. On section, the liver is fairly resistant, lardaceous, and, as it were, infiltrated by a highly refracting colloid substance. It is easy to see the amyloid infiltration by means of tincture of iodine. If the surface is moistened with tincture of iodine, the stained part assumes a mahogany-red tint, which turns into blue and violet. If the test is made with methyl-aniline violet, the affected parts are stained red-violet and the healthy parts blue-violet (Cornil). Under the microscope we see that the degeneration affects the cell and the vessels, especially the hepatic artery. The biliary passages remain intact. Amyloid degeneration of the liver is often associated with other lesions (steatosis, syphilis).

The symptoms of the amyloid liver are as obscure as those of the fatty liver, and the disease might be latent in its course were it not for the enormous enlargement of the organ. Furthermore, amyloid degeneration of the liver is often associated with amyloid disease of the spleen, kidneys, and intestine, in which case other symptoms appear (diarrhœa, albuminuria).

The treatment must be based on the ætiology of the amyloid degeneration. Tonics, constitutional remedies, and a long sojourn at the seaside should be advised for tubercular patients and those worn out by long suppuration. Iodide of potassium and mercurial preparations should be prescribed for syphilitics.

## XVIII. ABSCESS OF THE LIVER.

**Acute hepatitis**, which often terminates in suppuration, and gives rise to the tropical abscess, is rare in our climate, but common in hot countries. This form of suppurative hepatitis will be the principal subject of this section. Aside from suppuration caused by hepatitis in warm countries, other forms of suppuration leading to abscess may occur. The study of these forms must only figure in this section as an indication, and I shall merely sketch the principal varieties before discussing acute hepatitis.

1. **Metastatic Abscesses of the Liver.**—These abscesses arise from any cause of purulent infection, such as traumatism, head injuries, surgical operations, smallpox, puerperal infection, or medical septicæmia (endocarditis, infective aortitis, or suppurative pneumonia). The abscesses are miliary and commence with an ecchymotic tint of the hepatic lobule. The bloodvessels of the lobule are congested with red and white corpuscles.

The latter, having passed out of the bloodvessels, infiltrate the lobule; the hepatic cell becomes granular and atrophied, and the small abscess is formed. These little islets of suppuration grow larger, unite with the neighbouring islets, and attain the size of a pea or a hazel-nut. The branches of the portal vein around them show secondary periphlebitis, phlebitis, and thrombosis. The phlebitis, in its turn, causes extension of the abscesses to the neighbouring parts.

How is the formation of these abscesses to be explained? The old theory of capillary embolism (Virchow) has been replaced by that of microbic infection. The pyogenic microbes enter the general venous system and are carried to the liver by the hepatic artery. These pyogenic agents, which are almost always the streptococci and the staphylococci, are arrested in the radiate capillaries of the hepatic lobules, where the circulation has become sluggish. Their presence and their toxine produce lesions in the vascular endothelium, obliteration of the capillaries, invasion of leucocytes, formation of a clot, suppuration, and miliary abscess.

Hepatic pyæmia is ushered in by rigors, marked rise in temperature, and profuse sweating. The liver becomes enlarged and painful, the skin is yellowish and earthy, and the urine contains bile pigment.

2. **Pylephlebitis.**—The abscesses do not begin in the general venous system, but in the portal system. Purulent inflammation of the portal vein (pylephlebitis) may follow ulceration of the intestine (dysentery), or an abscess in the spleen and the phlebitis extends as far as the bloodvessels of the liver. Various micro-organisms, especially the coli bacillus, must be in evidence. At several points in the liver little abscesses are situated along the course of the inflamed veins. The vein is attacked by phlebitis and periphlebitis, and surrounded by embryonic tissue. In certain places the walls of the vein are destroyed. Such is the course of the abscesses caused by pylephlebitis, but in many cases pylephlebitis is not necessary, and the arrival of the pathogenic microbes in the liver may cause suppurative inflammation of the vein, which produces inflammation of the liver tissue and abscesses in the surrounding region. Abscesses of the liver consequent on appendicitis (appendicular liver) will be discussed later.

3. Can embolism of the portal vein caused by thrombosis of the mesenteric vein or of the other portal tributaries cause abscesses of the liver? It gives rise to an infarct of the embolized region, with consequent anæmia and granulo-fatty degeneration; but this infarct is not followed by suppuration. An exception must be made with regard to portal embolisms loaded with micro-organisms, such as the coli bacillus (septic embolism).

Widal has shown that the pyæmic abscesses of the liver in puerperal infection begin round the hepatic veins. The process commences with



endophlebitis, leading to periphlebitis and abscess. The periportal tissue is generally free.

4. **Biliary Abscesses.**—Several causes (gall-stones, obstruction of the common duct, etc.) may lead to inflammation and enlargement of the bile-ducts. The dilated ducts are at times as large as the finger and filled with muco-pus. At first sight the condition might be taken for abscesses, but on closer inspection it will be seen that it is caused by dilatations of the bile-ducts, which are often surrounded by new connective tissue. In certain cases, however, the walls of the ducts are destroyed, and the angiocholitis is followed by abscess of the liver. This question is discussed under Angiocholitis and Gall-stones. We have seen how the secondary infections begin and cause these biliary abscesses.

### **Acute Suppurative Hepatitis—Large Abscesses of the Liver.**

**Ætiology.**—Acute suppurative hepatitis is rare in France, and very frequent in hot countries (India, Algeria, Senegal, Bengal, Cochin-China, Martinique). Dysenteric infection is the chief cause. Several conditions may present themselves : In the first series, dysentery precedes the abscess ; in the second series dysentery and abscess march side by side. Finally, in the third series, the hepatitis or tropical abscess, as Murchison called it, appears to be independent of dysentery. This independence is most frequently only apparent. In any case, it may be said that the amœba of dysentery produces hepatitis, abscess of the liver, and dysentery, separately or simultaneously.

Abscess of the liver is found in countries where dysentery is found, and both diseases have the same origin. There are, however, cases of suppurative hepatitis in hot countries, and of abscess of the liver which have an origin apparently independent of dysentery. The intimate nature of these various abscesses is unknown to us, but it may be said that they have nothing in common with malaria.

Tropical dysentery is not the only form which may cause abscesses of the liver. Boinet has described large abscesses of the liver of dysenteric origin in France.

What does bacteriology teach us ? In non-dysenteric abscesses bacteriology has revealed the presence and also the absence of staphylococci. As far as dysenteric abscesses are concerned, they are chiefly due to amœbic dysentery. The pus of tropical abscesses of the liver is often aseptic, or of slight virulence, even though it is exceedingly foetid. This feeble virulence is the more remarkable " because such is far from being the case in angiocholitic pus, which is essentially septic and infective " (Chauffard).

**Description.**—Acute hepatitis does not always commence in the same manner. It is sometimes preceded by hepatic congestion, which does not

end in suppuration. The congestion shows itself by "pain in the side," with or without fever; the liver is enlarged, and vomiting and diarrhoea appear. A few days later recovery supervenes, but the patient is liable to recurrence or to abscess if he does not leave the country.

Acute hepatitis often commences with a severe rigor and sharp pain in the hypochondrium and the right shoulder. The liver is enlarged, the fever is remittent, and jaundice is seen in a third of the cases. The tongue is dry, and the patient has a typhoid look. In other cases the hepatitis is practically latent at first. The local symptoms are absent, and the general symptoms merely comprise malaise, or attacks of intermittent or remittent fever, readily taken for malaria. The pus forms from the eighth to the twelfth day, and the foregoing modes of onset indicate that tropical abscess may be ushered in by acute symptoms of hepatitis or only by slight gastrointestinal troubles. The abscess may even be latent without fever in one case, and in another case it may reveal its presence by most grave symptoms. Locally the abscess of the liver cannot be recognized unless it is of fair size. The extent of the dullness, the deformity of the liver, and the projection of the organ towards the thoracic or iliac region, depend on the seat of the abscess.

The abscess, once formed, may remain stationary for weeks and months; but most commonly in about three weeks it tries to make its way outwards. Rupture of the abscess into the peritoneum is not always followed, as might be supposed, by rapid and fatal peritonitis. In several cases surprising tolerance of the peritoneum has been noticed, death occurring after some days, with symptoms of prostration. Rupture of the abscess into the intestine sometimes leads to recovery, which may also follow if the abscess opens through the abdominal wall or into the bronchi.

I may mention, as exceptions, rupture of the abscess into the stomach, pelvis, kidney, vena cava, or pericardium. It appears possible that the pus may be absorbed, the abscess being replaced by a cicatrix.

The foregoing description shows that the diagnosis is often difficult. The prognosis is grave, especially because endemic hepatitis is subject to recurrences, and sometimes ends in a chronic form, after long periods of intermission. An opened and cured abscess does not protect against fresh abscesses. No limit to the period of formation of abscesses can be fixed, and persons may leave the tropics for Europe, but yet fresh abscesses may still appear.

**Pathological Anatomy.**—The liver in acute hepatitis is large, reddish, and friable. It is rare for several abscesses to be found, and suppurative hepatitis in three-quarters of the cases only causes one abscess—a marked difference from the pyæmic abscesses, which may be very numerous.

The tropical abscess is most frequently seated in the right lobe of the



liver, and on its convex surface (Dutrouleau). The quantity of pus may amount to 2 or 3 pints. It is yellowish or reddish, thick, creamy, and sometimes stained with bile. It may become foetid from the close connection of the intestine, or in consequence of communication with other organs.

The walls of the abscess are often anfractuous, formed of embryonic tissue. Sloughs often detach themselves from the walls. At a more advanced period the pyogenic membrane becomes surrounded with a fibrous membrane. The abscess commences in the deep parts of the organ, and gradually reaches the surface. When it comes in contact with Glisson's capsule, it behaves differently, according to circumstances. Adhesions are established between the liver and the neighbouring organs. Ulceration takes place, and the pus makes a passage for itself through the diaphragm and into the bronchi (vomica), into the peritoneum, the pericardium, the pleura, or the intestine; but the abscess sometimes opens through the abdominal wall.

**Treatment.**—Bleeding has been recommended (Dutrouleau), and ipecacuanha has been given in an enema (15 grains of ipecacuanha in twenty-four hours) (MacLean). The formation of the abscess must be watched in order to open it without delay.

## XIX. APPENDICULAR LIVER.

I have given the name of **appendicular liver** to the toxi-infection of the liver consecutive to appendicitis. From its situation the liver receives the frontal attack of the toxines and microbes, whose virulence has been increased in the closed cavity. The appendicular veins and the branches of the portal vein carry toxines and microbes to the liver. It is nevertheless important to distinguish between toxic and purulent hepatitis. Soon after the onset of appendicitis the liver may be affected by the toxines, which are carried more rapidly than the microbes; a change in the hepatic cells, which deserves the name of toxic hepatitis, is the result. This early toxic hepatitis does not suppurate. Later, during the second or third week of appendicitis, the microbes are carried to the liver, and cause purulent hepatitis. It is, therefore, indispensable to describe separately toxic and purulent hepatitis.

### 1. Toxic Hepatitis.

I have already described toxic appendicitis, or **appendicæmia**, in which the signs may be jaundice, choluria, urobilinuria, albuminuria, hæmatemesis, etc. I shall here discuss one of these signs, jaundice, with the concomitant lesion of the liver, which I was the first to point out in my communication to the Académie in 1898.

**Clinical Cases.**—A young man of twenty years of age came into my wards, and a student remarked: "There is a man with jaundice." The man had, as a matter of fact, a yellowish tinge, not pronounced on the skin, but well marked on the conjunctivæ. He said that he was suffering from his stomach. Abdominal pain and jaundice naturally aroused at first the idea of hepatic colic. The pain in the belly had commenced four days before, on Sunday morning; towards evening he was taken ill with fever and vomiting, and the abdominal pain persisted all night. On Monday, as the situation did not change and the belly remained tender, the patient came to the

hospital. I asked him to indicate the exact spot where the pains commenced, and also where they were most severe. He at once placed his finger on McBurney's point. Palpation revealed tenderness and muscular resistance; the hyperæsthesia was most marked at this point. The diagnosis was, therefore, appendicitis.

An explanation had still to be found for the jaundice: the liver was neither enlarged nor painful. Was the jaundice a simple coincidence, or was there some relation between it and the appendicitis? My first impression was that it was not a case of true jaundice. Examination of the urine with nitric acid showed no ring of biliverdin or bilirubin, but gave a brownish disc. Spectroscopic examination revealed the presence of urobilin and of brown pigment. The yellow tint of the skin and of the conjunctivæ was, then, not the result of true jaundice. The analysis of the urine likewise proved the presence of **albumin** in fairly considerable quantity. With these symptoms I expressed the opinion that this youth was suffering from **toxic** appendicitis, the appendicular toxine, elaborated in the closed cavity, having caused changes in the cells of the liver and kidneys, whence the urobilinuria, jaundice, and albuminuria.

The diagnosis being given, treatment had to be decided. After having obtained the consent of the patient, and in spite of the apparent mildness of the appendicitis, I requested Marion to operate. He found a collection of foul-smelling pus in the peritoneum; the appendix was gangrenous, and bathed in pus; a large calculus was found in the lower portion of the canal, which was greatly dilated, and converted into a closed cavity. It is easy to see what would have happened had the appendicitis in its **gangrenous** form been allowed to run its course.

This example shows once more that the gravity of the appendical lesions is not always in direct relation to the intensity of the symptoms. The ablation of the appendix removed the primary focus, but the liver and the kidneys were also affected. On Wednesday the situation became critical: the patient was prostrate; his face was drawn and yellowish; hiccough was frequent; the urine was scanty and albuminous; the temperature was 102° F., and the pulse 106. Two injections of serum (1 pint in each) were administered. He passed a better night. Next day, Thursday, the hiccough had almost gone; the temperature was 99° F.; but slight jaundice was still present. Urobilin still persisted in the brownish urine, but, an important point, the **albumin had disappeared**. Two days later the jaundice disappeared in its turn, and the urine contained no more urobilin; a few days later the patient was out of danger. This case, described in one of my lectures on the toxicity of appendicitis, is a proof of this toxicity. The toxine had affected the liver and the kidneys, and the intoxication revealed itself by urobilinuria, jaundice, and albuminuria. The appendix being removed, the albumin disappeared within twenty-four hours, and the urobilinuria ceased three days later.

Routier has mentioned several cases to me. One of them referred to a young girl who was operated on for appendicitis, and cured. From the commencement of the disease she had jaundice, which became more pronounced during the next few days. Another case referred to a man who underwent an urgency operation, and was cured of appendicitis. The jaundice, which was present on the day of the operation, persisted for several days.

Valmont saw a man who had had several attacks of appendicitis. He was taken ill with jaundice during the last attack, and died in three days. One of the patients, whose case I have referred to under Appendicular Vomito Negro, was taken ill, at the commencement of appendicitis, with jaundice, which became general over the whole body.

In some cases the jaundice is obstinate, and may last for weeks and months, until the appendix is removed. Hartmann communicated the following case to me: In September, 1897, a young man was taken ill with clearly-defined pain in the right iliac fossa. Dreyfus-Brissac diagnosed appendicitis. The acute crisis once passed,



the young man still had a somewhat yellow complexion, and the liver was found to be enlarged. Dreyfus-Brissac sent the patient to Bréda for a cure. On October 2, 1898, a fresh attack of appendicitis occurred. This time there was also jaundice, and albumin was found in the urine. Hartmann removed the appendix on October 14; on October 25 the jaundice and the albumin had disappeared.

In August, 1903, in consultation with Segond, I saw a woman who had had jaundice since February, when she suffered from appendicitis. The pain in the right iliac fossa had never completely disappeared. An operation was performed, and ten days later the jaundice disappeared.

A woman was in the Salpêtrière for appendicitis in February, 1903. She remained jaundiced till Segond operated for appendicitis three months later.

**Description.**—This toxic jaundice may appear on the second or third day of an attack of appendicitis, but we must not expect to find marked jaundice. The yellow tint is often slight. It is most pronounced in the conjunctivæ and the face, and it is only in exceptional cases that it becomes general, like true jaundice. The fæces remain coloured. The urine is fairly dark, and true bile pigments may be found in it, but most frequently it contains only urobilin or brown pigment. In some cases the liver is enlarged.

The jaundice is not of long duration, and quickly disappears after appendectomy. Patients have been seen who with each attack of appendicitis had an attack of jaundice. Sometimes, as we have seen in the cases quoted above, the colour remains for weeks and months, and only disappears after ablation of the appendix. In view of these facts, I think that the intoxication of the liver consecutive to appendicitis may play some part in the causation of cirrhosis of the liver.

This form of jaundice is sometimes the sole evidence of the appendicular toxicity, but most frequently it is associated with albuminuria as a second sign. In patients suffering from appendicitis we must always look for jaundice and albuminuria, and the urine must always be examined for pigment, albumin, and casts, because all these signs, formerly, and even now, too much neglected, point to the impregnation of the system by the appendicular toxines. They are another argument in favour of early intervention, so as to remove the focus which produces the poison.

Jaundice in an individual with appendicitis does not simplify the diagnosis. As a matter of fact, there are cases in which the pain of appendicitis, though present at McBurney's point, may radiate as far as the subhepatic region (ascending type of appendix). Let us suppose that in such a case the patient also has slight jaundice. The idea of hepatic colic will naturally present itself. How are we to make a diagnosis? In the hepatic colic true bile pigments are found in the urine, but this is not a pathognomonic sign, because they may also be found in appendicular jaundice. What is more important is that in the case of hepatic colic the painful focus has neither its origin nor its maximum intensity at McBurney's point. In appendicitis,

even when the pain radiates towards the hypochondrium, the maximum intensity of the pain, the muscular resistance, and the hyperæsthesia are in the appendicular region. Hepatic colic with jaundice, therefore, will not be mistaken for the icteric tint of appendicitis.

From the point of view of **prognosis** we must always mistrust toxic jaundice in appendicitis, for it is sometimes the prelude of a terrible general intoxication. We find at first slight jaundice and albuminuria, and later urinary and hepatic insufficiency, hæmatemesis, nervous troubles, and death.

**Pathological Anatomy.**—In a case described more fully under Appendicular Kidney I was able, thanks to the kindness of Letulle, to make a histological examination. Sections of the liver treated with osmic acid, after fixation in 10 per cent. formalin, showed an accumulation of very fine fatty granules in the interior of the liver cells, especially near the centre of the lobule. The remainder of the trabeculæ, however, were not free from fat. One detail was interesting to note: accumulation of fat in the endothelial cells, which showed a fair number of trabeculæ, and in the interior of the capillary vessels a large number of leucocytes charged with fatty granules. In sections stained with hæmatoxylin-eosin the lobulated look of the liver was somewhat remarkable. This disposition was partly due to condensation of the periportal connective tissue and to some islets of trabecular atrophy, with pigmentation of the cells, grouped around the hepatic veins of average size. There was no necrobiosis in the liver cells. In short, I found in the liver lesions of granulo-fatty degeneration of the centro-lobular hepatic cells—that is, lesions due to superacute intoxication.

In another case of toxic appendicitis published by Lorrain the histological lesions greatly resembled those described by Letulle. Certain cells were filled with very fine grains of the colour of rust. “The presence of the ochre pigment in abundance in the cells of the liver indicates deep-seated trouble, probably resulting from acute intoxication.”

In another case, discussed under Appendicular Kidney, the histological examination made by Nattan-Larrier proved that the cells of the organ were undergoing fatty degeneration.

## 2. Infective Purulent Hepatitis.

Toxic hepatitis, as I have said, occurs early, and is accompanied by neither hepatic pain nor violent symptoms. Purulent hepatitis, due to the infection by the appendicular microbes, is quite different. It appears during the decline or convalescence of appendicitis. It is accompanied by sharp rigors, severe fever, pains in the hypochondrium, rapid increase



in the size of the liver, jaundice, etc. The reader will get an idea of it from the following cases taken from my clinical lectures :\*

**Clinical Cases.**—A man came into my wards on March 12, 1898, with febrile icterus. The temperature was 102° F. on admission, and reached 104° F. the next morning ; the pulse was rapid, and the tongue dry and parched ; the yellow colour (though not very intense) extended over the whole body ; the urine was abundant, of a mahogany colour, fairly rich in bile pigment, and Gmelin's reaction gave a very clear ring of bilirubin ; a cloud of albumin was also visible. The fæces were only slightly coloured, but still they had not the whitish, putty-like appearance seen in obstruction of the common bile-duct, when the bile can no longer reach the intestine. The case was, therefore, one of true icterus, with high fever, and as the patient was prostrate, feverish, and answered questions with difficulty, we thought at first of icterus gravis.

Examination of the liver gave valuable information. The hepatic region formed a bulging which extended as far as the epigastrium ; this bulging was so marked that an exploratory puncture had been made, without any result, prior to the admission of the patient, on the hypothesis that it might be a fluid tumour. The liver was enlarged, very tender on pressure, and extended below the false ribs by the width of two fingers. The other abdominal and thoracic organs were healthy, and the entire disease seemed to be confined to the liver. The man told us that, twelve days previously, on February 27, he had been seized with a violent rigor and sharp fever, which was but the prelude of many similar attacks. Rigors and fever had since recurred almost daily, without any periodicity.

According to the patient, acute hepatic pain appeared with the fever ; this pain, though less severe, was still present ; the jaundice had come on some days after the attack of fever.

This information led to various suppositions. The acute attacks of fever, the hepatic pain, and the enlarged liver caused us to think of suppuration ; but what could be the cause ? The hypothesis of a suppurating hydatid cyst was hardly likely, and, moreover, an exploratory puncture had been without result. Had we to deal with a large abscess of the liver, analogous to the so-called tropical "abscess" which generally follows on dysenteric infection ? On the other hand, had we to deal with small miliary abscesses associated with suppurative angiocholitis ? The former hypothesis was hardly admissible in the absence of any cause for a large abscess in the liver. As for a large abscess from dysentery *nostras*, its symptomatology is so masked that it often passes unnoticed. The second hypothesis was debatable, as the clinical picture recalled the symptoms of infective angiocholitis, whether due to gall-stones or not. Angiocholitis, however, does not cause so much enlargement of the liver in a few days.

There is a variety of suppurative hepatitis which must always be remembered in these cases. I refer to hepatic abscesses consecutive to appendicitis. Sharp attacks of fever, hepatic pain, rapid increase in the size of the liver and icterus, form a syndrome which should awaken the idea of hepatic infection consecutive to infection of the appendix. It became, therefore, necessary to know whether the patient had recently had an attack of acute appendicitis. I say *acute* appendicitis, because it is only during the active phase of the closed cavity that such complications can take place. On this point the patient replied that some ten days prior to the attacks of fever he had felt sharp abdominal pain, which had prevented him working. He indicated the region which corresponds exactly with McBurney's point. The appendicular pain had lasted only a few days, but it was undeniable, and had been accompanied by severe constipation, as is customary.

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\* "Le Foie Appendiculaire," "Abcès du Foie Consécutive à l'Appendicite" (*Clinique Médicale de l'Hôtel-Dieu*, 10<sup>me</sup> leçon).

We were, therefore, able to state that the man had at this time an apparently benign attack of appendicitis. Do we not, however, know that appendicitis is never benign, in the true sense of the word? Do we not know that the exaltation of virulence in the closed cavity, **which sums up the entire history of appendicitis**, may favour the emigration of the pathogenic microbes in all directions, and thus, by means of the lymphatics and veins, give rise to remote infections, the most terrible of which undoubtedly is hepatic infection? In consequence I diagnosed purulent infection of the liver, following appendicitis, and gave a fatal prognosis.

During the month that this patient was in my ward we counted fourteen severe attacks of fever, the temperature reaching 105° F., and the attacks being preceded by violent rigors. In a short time the liver formed an enormous abdominal tumour, which was hard and smooth. The jaundice improved at times, and the fæces became more or less coloured. The hepatic region was painful, the pain radiating to the abdomen and thorax. Respiration became difficult, and the râles due to pulmonary œdema were heard at the base of the chest, especially on the right side.

On March 31 facial erysipelas broke out, and disappeared in a few days. During the last days of the disease the liver was still enlarged; the jaundice had, so to say, disappeared; the belly was much distended; and the stools were loose, fœtid, and almost colourless. The patient sank into a typhoid state. He died with a temperature of 104° F. four weeks after his admission, and less than six weeks after the start of the hepatic infection. The appendicitis, which was the cause of the fatal complications, was two months old.

As far as the treatment was concerned, I had not thought of surgical intervention, because surgery is practically powerless in multiple abscesses due to appendicular infection of the liver. A single abscess is absolutely unique.

The post-mortem examination confirmed the diagnosis. The liver weighed 100 ounces. Its surface showed at various points yellowish or brownish projections. At first sight the specimen might have been taken for secondary cancer. The consistency of the organ was soft. Sections of the lobe revealed abscesses everywhere, the gland being riddled with them. From 150 to 200 abscesses, varying in size from a pin's head to an egg, might certainly have been counted, and there was also an abscess of the size of an orange in the right lobe. Some were just beneath Glisson's capsule, others were deeply seated in the parenchyma. They contained fairly thick pus, without fœtid odour, the colour varying from yellow to green. These abscesses were mostly independent, and were separated by septa of healthy or altered hepatic tissue, but they had no proper walls. Other abscesses communicated with one another, forming large anfractuous cavities. On section a fair number of these abscesses had a spongy, areolar aspect, whence the name **areolar abscesses of the liver** (Chauffard).

The extrahepatic bile-ducts were healthy and permeable, and such was also the case with the trunk of the portal vein. The organs of the abdomen were absolutely normal, and, except for a few perihepatic adhesions, we found no peritoneal lesion and no ascites; nothing in the spleen; nothing in the intestine.

The appendix was surrounded by false membranes, which increased its size threefold by uniting it to the posterior surface of the cæcum and to the front of the psoas. Dissection of the adhesions, so as to isolate the appendix, revealed a small peri-appendicular abscess, containing about a teaspoonful of slightly fœtid pus. The veins at the base of the appendix were so enlarged as to form a prominent varicose network, which, after enveloping the appendix, spread over the cæcum, where it blended with the mesenteric veins.

I have never seen such a venous network in a case of appendicitis; it is probable that it only acquires such a development in the case of venous infection. The appendix was not perforated, but the mucosa was ulcerated at three or four points, and towards the tip there was a very small abscess. Let us next consider the histological lesions and



the bacteriological examination. The pus from the small peri-appendicular abscess gave a pure culture of coli bacillus. The veins of the cellular coat of the appendix were, in a large number of instances, attacked by endophlebitis and periphlebitis.

Several of these veins were filled with thrombi, formed in part by endothelial cells and fibrous tissue. By the side of these thrombosed veins, especially under the serous membrane, very dilated veins, which contributed to form the appendicular plexus, were found. Microbes were present in the walls of the thrombosed veins, and in the inflamed tissue round them.

The microbes, after leaving the closed cavity, became engaged in the appendicular veins, giving rise to phlebitis and thrombosis, and reached the liver through the portal vein.

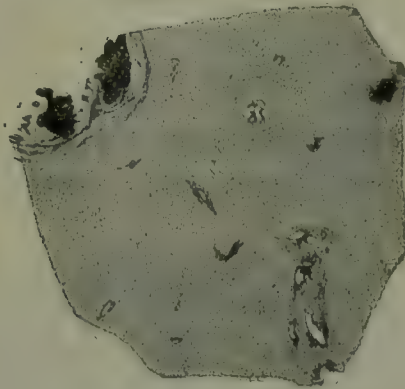


FIG. 50.—FORMATION OF ABSCESS IN LIVER, SECONDARY TO APPENDICITIS.

After reaching the liver, they followed the course of the portal veins—that is to say, they occupied the periphery of the hepatic lobules. It was, therefore, around the lobules that the infection of the liver had commenced. The microbes swarmed there, and embryonic cells sheathed the portal vein like a sleeve.

The process gradually extended, and in the end the liver was transformed into a sort of purulent honeycomb.

**Summary.**—In this case it was possible to follow the organisms in their migration from the appendix to the liver. When we remember that hundreds of abscesses were



FIG. 51.

L, normal lobule; vp, portal vein (normal); a, b, c, abscess in various stages.

formed in the liver in a few weeks, the virulence of the microbes will be understood. The exaltation of the virulence, acquired in the focus of the appendix, allowed the microbic colonies to pass through the appendicular veins, to follow the blood-stream as far as the portal vein, **vena porta malorum**, and rush forward in infecting columns to the conquest of the liver.

Such is the hepatic infection. This complication of appendicitis, which differs from toxic hepatitis, is far from being rare. Bertholin has collected twenty-eight cases in his thesis.

**Description.**—Events usually run the following course : An individual, either a child or an adult, is attacked with severe or slight appendicitis. The physician commences to investigate carefully the area in which the pain has first shown itself. Palpation, pressure, muscular resistance, and hyperæsthesia show that the painful spot is at the centre of a line drawn from the umbilicus to the anterior superior iliac spine. Although the belly may be sensitive in other regions, it is here that the pain commences and reaches its maximum. He inquires as to the onset of the disease, and learns that the patient was in a state of perfect health when he experienced the first symptom. At that time, or a little later, attacks of nausea, and perhaps of vomiting, supervened. The diagnosis of appendicitis is clearly established, and the doctor explains how even slight appendicitis exposes the patient to the greatest dangers, and mentions the many cases where

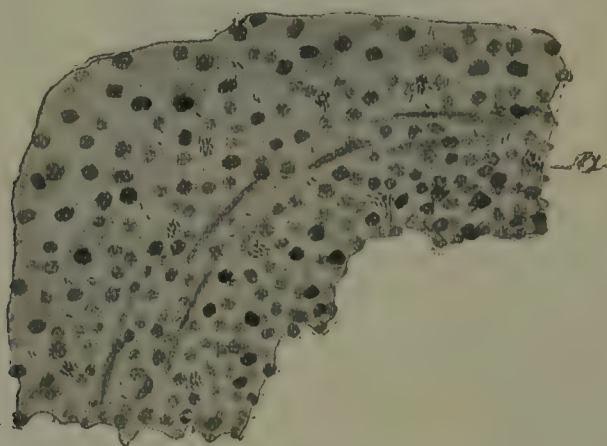


FIG. 52.—INVASION OF LIVER BY THE MICRO-ORGANISMS AT *a*.

appendicitis, looked upon as almost a negligible quantity, has been followed later by peritonitis or suppurative infection of the liver or of the pleura, etc., and in order to avoid such a dire result, he insists on an operation.

If the doctor is a believer in the erroneous doctrine “that there is time enough to see how things will develop,” as the condition of the patient appears to be so little alarming, the fever is so mild, and the belly is so little swollen ; if he remains satisfied with an approximate diagnosis ; or if he only sees in the case typhlitis or typhlo-colitis, which in a few days will yield to purgatives, applications of ice, and injections of morphia, he will consider the case benign, and will reject surgical intervention. As a matter of fact, a few days afterwards convalescence sets in, and seems to justify the treatment. He proclaims aloud that it is wrong to hand over people to the surgeon when they are suffering from appendicitis and are simply in need of medical treatment. The triumph is, however, of short duration. During the few days when the patient is seemingly recovering, the infection is silently continuing its course, and the pathogenic microbes, starting from the appendix, and armed with terrible virulence, have already reached



the liver. After a silent phase of incubation the severe symptoms of infection of the liver—namely, sharp attacks of fever, hepatic pain, rapid enlargement of the organ, and jaundice—appear. The mischief makes speedy headway, and in a week or two the patient succumbs. Let us never forget that the fatal consequences of appendicitis are by no means always in relation to the intensity of the symptoms. I have discussed this question under Appendicitis, and quoted cases of patients apparently suffering from slight appendicitis, and yet an operation performed thirty hours after the appearance of the disease revealed gangrene of the appendix and peritonitis, which would have been fatal if the operation had been postponed.

Similar considerations are applicable to hepatic infection following on appendicitis. The appendicular phase is sometimes very acute and painful, but at other times it may be fairly slight, and so escape the notice of an inexperienced observer. We have seen the small space occupied by this appendicular phase in my patient, who suffered so little discomfort that he had only stopped work for a few days, and yet the consequences were to be fatal. Hepatic infection may supervene in any case of appendicitis, whether the appendix is or is not perforated and whether peritoneal or peri-appendicular lesions are present or absent. It is not the extra-appendicular lesions which constitute the danger. The whole danger comes from the closed cavity where the toxi-infection gathers strength.

We find here a new application of this exaltation of virulence in the closed cavity, which has been so clearly proved by experiment, and which has enabled me to explain the whole history of appendicitis. It is in the closed cavity that the microbes acquire the increase of virulence and the power of expansion, enabling them to traverse the walls of the appendix and establish themselves in the peritoneum, even though the walls are not perforated. It is likewise the increase of virulence and the power of expansion acquired in the closed cavity which facilitate the migration of the microbes through the bloodvessels, and enable them to cause fatal remote infection. Hepatic infection is one of these remote infections peculiar to appendicitis. The microbes—coli bacilli and others—by virtue of their increased virulence, enter the appendicular veins, and reach the great mesenteric vein, the portal vein, and the liver. In some cases, as in my patient, the infected veins form a varicose network. Some small veins are thrombosed; others are not. Phlebitis sometimes affects the larger venous trunks, such as the great mesenteric vein, which receives the appendicular veins, and even the trunk of the portal vein, which starts from the mesenteric vein.

I would remind the reader how this hepatic infection announces itself. The patient has been suffering from appendicitis, and it matters little whether the attack has been severe or mild. Sometimes, indeed, an operation

has been performed (**too late**), and the patient is on the highroad to convalescence. And yet rigors, temperature rising to  $104^{\circ}$  F., and profuse sweats open the scene. The attacks return daily, and the fever is continuous. The attacks of fever are accompanied by hepatic or epigastric pain, jaundice, gastric intolerance, and vomiting. These symptoms are accompanied by rapid swelling of the liver, which may become enormous. The spleen remains normal. Diarrhœa is as frequent as constipation. In one of my cases it was exceedingly profuse. Jaundice may be early or late, slight or intense. The general symptoms are those of the typhoid state. The pulse is quick, the tongue is dry and red, and the attacks of fever persist for one or two weeks. Sometimes the fever improves, and there is a temporary remission, but in the end the patient succumbs in a state of adynamia, collapse, or syncope, or with symptoms of icterus gravis, multiple hæmorrhages, albuminuria, and anuria.

The hepatic infection is always consecutive to the acute phase of appendicitis. It is not to be feared when the active process of appendicitis has been extinct for some time. On the other hand, I do not know of a single example where hepatic infection supervened quickly during the early stage of appendicitis. The migration of the microbes into the veins of the appendix never commences—at least, so I believe—before the fifth or sixth day. Surgery has, therefore, ample time to intervene before the commencement of the appendiculo-portal migration.

This hepatic infection is one of the most terrible complications of appendicitis, because I am only acquainted with two cases of recovery (Kœrte, Loison). The operation was successful because, extraordinary to relate, there was a solitary hepatic abscess. In Loison's case radioscopy gave considerable support to the diagnosis. Summary: Except in very rare cases, medicine and surgery are powerless to avert the evil once it has declared itself. As a matter of fact, however successful the surgeon may be in solitary abscess of the liver, intervention is hopeless in appendicular infection, where abscesses of the liver are almost always counted by dozens. What a lesson, however, for those who say that we must only perform interval operations! Once again I protest against such an assertion. In appendicitis we must never wait, for we can never know what the future has in store for us.

## XX. GALL-STONES.

**Structure and Formation of Gall-Stones.**—The production of gall-stones is one of the most common troubles in the human race (Cruveilhier). They may be formed in the intra- or extrahepatic ducts, or in the liver, but the gall-bladder is the most common situation.



Biliary concretions of all sizes are found in the gall-bladder, from gravel to calculi larger than an egg. They may be solitary, multiple, or indefinite in number. The solitary calculus has no facets, and may be oval or pear-shaped. Multiple calculi are rounded or pyramidal and faceted from the rubbing and pressure of neighbouring calculi. They are elongated when they have been long in the common duct. Their colour is brown, greenish-yellow, or blackish. It is whitish in the case of calculi of cholesterin. Their density, though low, is greater than that of water. When they have not been dried, their structure varies, according as they are simple or compound. The compound calculi have a nucleus and a crust. The nucleus is formed of bile pigment, chalk, epithelial cells, and rarely foreign bodies. It is surrounded by a middle radiating layer, in which crystals of cholesterin predominate. The crust is stratified and composed of cholesterin, bile pigment, or chalk. Calculi without a crust may be met with. Others, again, called simple calculi, are homogeneous throughout their whole thickness. The calculi contained in the same gall-bladder are identical as regards structure, colour, and chemical composition. Agglomerated and fragmented calculi are found. The consistency of the biliary concretions is moderate, and the most resistant ones are formed of pure cholesterin. The calculi are free in the bladder. They may sometimes be adherent to the wall. They are then let into the walls of the gall-bladder which have become alveolar, as Terrier observed when performing cholecystotomy. The chief components of gall-stones are crystalline or amorphous cholesterin, and next the colouring matters of the bile and the calcareous salts. It is curious to note that cholesterin and chalk, which are found in very small quantities in the bile as compared with the other elements, form the chief constituent parts of the calculus. Thus, cholesterin, on an average, forms 70 per cent. of the constituent parts of the calculus, whilst the bile barely contains 2 per cent. The salts of potassium and of sodium, which alone form some calculi, are found in very small quantities in the bile. Throughout the whole thickness of the calculi there is a kind of organic albuminoid web, which indicates the participation of the mucosa in the formation of calculi.

The exact manner in which the stones are formed is still imperfectly known. The substances contained in the bile must be precipitated and agglomerated, and must be kept agglomerated. It is supposed that chalk is a product of the secretion of the gall-bladder (see Catarrh of the Gall-Bladder). Catarrh of the gall-bladder is said to have the power of acidifying the bile, and the acidity of the bile is said to split up the bile salts; by the dissolution of the salts, cholesterin and bilirubin are set free and precipitated—the former in a crystalline form; the latter either in a crystalline form or mixed with the chalk.

It is admitted that microbic infection plays an important part in the process, by favouring cholecystitis, altering the composition of the bile, and contributing to the formation of the nuclei. It may, perhaps, be that typhoid fever and other infectious diseases which affect the gall-bladder are, by reason of the microbes, the remote origin of biliary calculi (Dupré). Dufour was able to collect fourteen cases of patients of various ages who had hepatic colic some months after an attack of typhoid fever, while they had had no symptoms of gall-stones prior to the fever. Several writers accept this pathogenesis of biliary lithiasis (Chiari, Gilbert), and Hanot thus sums up the question: "If the intestinal catarrh spreads to the biliary passages and becomes lithogenous catarrh—that is, if the micro-organism of typhoid fever is, after all, capable of causing biliary lithiasis—the question of a microbic origin is settled. It would, then, remain to be decided which microbes, other than those of typhoid fever, intervene. It may be possible that every microbe, from the very fact that it swarms in the bile-ducts, causes chemical changes leading to the deposit of the mineral principles. Thus, Galippe actually saw the formation of crystals of carbonate of lime in saliva placed in a flask, around masses of micro-organisms which were growing therein."

"Biliary lithiasis would, then, result from the mode of penetration of the micro-organisms into the bile-ducts, from their greater or smaller number, and particularly from the constitution of the mucus, which is more or less readily deposited. If this last theory is true, the soil is more important than the seed, and biliary lithiasis would no longer be an accidental phenomenon, but would remain the expression of a previous state of the organism, of a hereditary or congenital modification, *totius substantiæ*—that is, of a diathesis." My ideas agree absolutely with those expressed by Hanot—that the diathesis is the factor in the pathogenesis of lithiasis.

**Ætiology.**—Biliary lithiasis is more frequent in women; it is most prone to occur in heavy eaters, in fat people who take little exercise, and in individuals past middle life. Often, says Trousseau, the real causes of the disease escape us, and a clear point is that these causes, no matter what they be, are dominated by a predisposition special to the individual. As a matter of fact, biliary lithiasis is often associated with the diathetic conditions that form a part of the group of arthritic diseases—migraine, gout, or rheumatism, urinary lithiasis, obesity, asthma, diabetes, and eczema—which have been described by Bouchard in his work on "*Les Maladies par Ralentissement de la Nutrition*." Urinary lithiasis is closely related to hepatic lithiasis, and these two manifestations may occur successively in the same individual, or exist simultaneously in one family, where they are hereditary. The same remark applies to appendicular lithiasis, and the



numerous cases quoted under Appendicitis show that hepatic, intestinal, renal, and appendicular lithiasis belong to the same pathological family. After the diathetic causes, or side by side with them, come infections of microbic origin, as I have mentioned above.

**Pregnancy—Puerperal Condition.**—Pregnancy plays a large part in biliary lithiasis. I have devoted a clinical lecture to this question.\* Hepatic colic is frequent in pregnant women. In a comprehensive work, the first which has appeared on the subject, Huchard has published several cases. The cases of Depaul, Tarnier, and Pinard are positive evidence. I have reported several cases. According to Leyden's statistics, in one hundred cases of women suffering from hepatic colic, concomitant or anterior pregnancy has been noticed in ninety of them. Authorities are not absolutely agreed as to what period of the puerperal state is most favourable to the appearance of hepatic colic. Some maintain that it is during pregnancy; others that it is during accouchement. Cyr has published the following statistics: In fifty-one women with gall-stones, hepatic colic has been noticed eleven times during pregnancy, four times after miscarriage, and thirty-six times after accouchement. The period between the accouchement and the attack of colic varied from one day to a month in twenty-two cases, and from one to twelve months in fourteen cases. In the statistics sent me by Bouloumié, of Vittel, hepatic colic figures as follows: twenty-two times during pregnancy, and fifty-five times from a day to a year after accouchement. In the statistics forwarded to me by Déléage, of Vichy, hepatic colic figures as follows: fifty-nine times during pregnancy, and forty-five times after accouchement.

Whether hepatic colic appears during pregnancy or after delivery, it is none the less true that the puerperal condition has a great influence in the pathogenesis of biliary lithiasis. Hepatic colic appears in various ways. First variety: A young girl who has never had hepatic colic marries; during her first pregnancy hepatic colic appears, and reappears during subsequent pregnancies; but never apart from the puerperal condition. Second variety: A woman who has never had hepatic colic, either as a girl or later during pregnancy, is seized with hepatic colic some days or weeks after delivery. The colic recurs after subsequent deliveries, and is never present except at this time. Third variety: Hepatic colic appears, either during pregnancy or after labour, and again later, at indeterminate periods, when the woman is not pregnant.

Hepatic colic during pregnancy or after delivery does not differ from the ordinary form. The diagnosis is generally easy; nevertheless, when it appears during pregnancy, it may, for want of clear symptoms, be mistaken

\* "La Grossesse et les Lithiases" (*Clinique Médicale de l'Hôtel-Dieu*, 1898, 15<sup>me</sup> leçon).

for a miscarriage. When it appears after delivery, it may, for want of attention, be looked upon as commencing peritonitis.

Many theories exist regarding the occurrence of biliary lithiasis during pregnancy. Pregnancy is said to favour stagnation of bile in the bile-ducts, and, as a consequence, the formation of calculi. It has been said that pregnancy favours mobility of the kidneys and liver. On account of the flaccidity of the abdominal walls, the gall-bladder, since its means of fixation have been relaxed, tends to swing backwards, and the common bile-duct being dragged on by this swinging movement, becomes narrowed; this narrowing favours stagnation of the bile in the gall-bladder. Pregnancy, according to Heidenhain, is also said to provoke stagnation of the bile, by hindering the play of the diaphragm, especially as the corset fixes the costal insertions of the muscle. This pathogenesis might be classified under the mechanical causes, but the chemical causes (malnutrition), the bacteriological causes (action of the microbes in forming calculi), and the constitution of the patient (arthritism, heredity), have also to be considered. All these theories are possible, but not one seems to me to be sufficient. The undeniable clinical fact is that the puerperal condition exercises a considerable influence on the pathogenesis of gall-stones.

After having shown the influence of pregnancy on the pathogenesis of hepatic colic, let us invert the question and inquire what influence hepatic colic may have on pregnancy and the sequelæ of labour. The prognosis in pregnancy is scarcely affected by hepatic colic; severe and repeated attacks of colic do not interrupt it. The jaundice which in many cases follows hepatic colic is not dangerous to the pregnant woman; it is merely jaundice by retention, which habitually ceases a few days after the colic, and does not injure the cells of the liver. If, however, it lasts too long, as in permanent obliteration of the common duct, the normal activity of the cell may suffer, and formidable mischief may ensue. Hepatic colic and jaundice do not, as a rule, render the prognosis serious, either in the pregnant woman or the recently delivered woman. And though jaundice due to a stone is not, as a rule, dangerous in the pregnant woman, this is not the case in the other varieties included under infective jaundice. It matters little what theory is formulated to explain the affection of the hepatic cell. As soon as the cell participates in the toxic process, it undergoes a change which under other circumstances might not have serious consequences, but which in the gravid condition is always to be feared. Jaundice supervening in a pregnant woman, therefore (I speak of jaundice apart from hepatic colic), is always a suspicious symptom, because it points to possible hepatic insufficiency, with all its consequences. This question will again be referred to under Jaundice, when we shall consider the relations between pregnancy and infective jaundice.



**Symptoms and Complications of Gall-Stones.**—In some cases (in old people particularly) gall-stones cause no symptoms. At a post-mortem examination it is by no means rare to find a calcified and enlarged gall-bladder, containing numerous calculi, in people who have shown none of the symptoms of gall-stones. More frequently, however, the stones cause various troubles and more or less grave complications, divisible into several groups :

1. Migration of the calculi into the large bile-ducts—hepatic colic.
2. Migration and arrest of the calculi in the intestine—obstruction of the pyloric orifice.
3. Persistent obliteration of the large ducts—biliary cirrhosis.
4. Infection of the ducts—angiocholitis, cholecystitis, hepatitis, endocarditis.
5. Passage of the calculi outside the natural passages—perforation of the gall-bladder and bile-ducts, peritonitis, biliary fistulæ.

### 1. Hepatic Colic.

**Anatomy.**—Hepatic colic is the most common complication of gall-stones. In order to understand the mechanism, it will be well to mention certain anatomical facts concerning the ducts through which the calculus has to pass.

In the normal condition the gall-bladder contains about 2 ounces of bile, but its walls are capable of such distension that it may contain more than 2 pints without rupture. The gall-bladder ends in a neck, which is very flexible, and is continuous with the cystic duct. The neck of the bladder presents a swelling, or pelvis. In an opened bladder we see that this swelling is limited above by a valve, which considerably narrows the orifice, and below by a second and less important valve. The gall-bladder is contractile, and possesses muscular fibres, forming a muscularis mucosæ, which becomes hypertrophied in biliary lithiasis. The mucosa shows both temporary and permanent folds. The former disappear as soon as the bladder is sufficiently distended ; the latter anastomose, and circumscribe areolæ of various forms.

The cystic duct is the continuation of the neck of the gall-bladder. It is nearly 2 inches long and  $\frac{1}{8}$  inch in diameter ; it opens into the common duct. On opening the cystic duct we see that its internal walls show projections (the semilunar valves of Heister), which prevent the migration of calculi. The common duct is formed by the fusion of the cystic and hepatic ducts. It is 3 inches long and about  $\frac{1}{4}$  inch in diameter ; it opens into the duodenum at Vater's ampulla, in common with the pancreatic duct. The common duct is not provided with valves, but at its entrance into Vater's ampulla it possesses a sphincter, composed of muscle fibres. As a matter of fact, the real opening is situated at the duodenal opening of Vater's ampulla, and at this point there is a very marked constriction, which is a final obstacle to the calculi coming from the bladder. The cystic and common ducts possess longitudinal muscle fibres, which disappear late in life. They are capable of such distension that they may allow the passage of calculi as big as a hazel-nut. The common duct when distended may be as large as a coil of bowel.

**Hepatic Colic.**—The contraction of the gall-bladder, bile-duct, and abdominal muscles causes a calculus to engage in the cystic duct. If the stone is neither large nor angular, it will pass through the cystic and common ducts

into the intestine without causing colic ; if the stone is larger than the lumen of the bile-ducts, it produces in its migration the symptom-complex of hepatic colic.

In the struggle between the calculus and the ducts, the former is pushed onwards, but causes, in its migration, spasm and contraction of the latter. It meets with numerous obstacles to its passage : in the cystic duct, which is of small calibre, it must pass a series of valves ; and in the common duct, which is larger, it meets, at the last moment, with the narrow orifice of Vater's ampulla. Hepatic colic appears most frequently a few hours after a meal, *post prandium*, probably because at this moment the gall-bladder contracts, in order to empty the bile into the intestine. The onset is sudden, and the subject complains of sharp pain, radiating in several directions : to the pit of the stomach (epigastric point), around the umbilicus, to the right hypochondrium, the right shoulder, and the lower extremity of the scapula on the same side (scapular point). The pains rapidly become very severe, and cause the patient intense agony. They may be continuous or intermittent, following one another at more or less close intervals, and constituting an attack of hepatic colic. The attack lasts, on an average, from six to twelve hours, though it may persist for several days. It is most frequently apyretic, but in some cases fever is also present.

Hepatic colic is, as a rule, accompanied by vomiting, which is at first alimentary, if the colic appears soon after a meal, and subsequently becomes glairy and bilious. As long as the calculus remains in the cystic duct, the vomit may be bilious, because the bile continues to pass into the intestine, whence it regurgitates into the stomach ; if, however, the calculus is arrested in the common duct, the passage of the bile into the intestine is interrupted, and bilious vomiting cannot occur. The same remark applies to the decoloration of the fæces. As long as the calculus remains in the cystic duct, the bile flows freely into the intestine, and the fæces are coloured ; but if the calculus is impacted in the common duct for some time, the fæces become colourless, and the yellow colour of the skin and of the urine reaches its maximum. Generally speaking, the attack of hepatic colic ceases suddenly, and the patient at once feels a delightful sensation of relief. The calculus has, then, gone back into the gall-bladder, after a vain attempt to pass through the cystic duct, or has reached the duodenum, after a painful migration through the ducts. The end of the attack is often accompanied by the passage of much watery urine (nervous urine). During the attack the liver is often enlarged, and the region of the gall-bladder is extremely tender on pressure.

When the colic has been long and severe, the liver remains enlarged and painful for several days ; it reaches below the false ribs, and is so tender on pressure that women cannot wear their corsets.



I have just described a severe attack, but hepatic colic is often much less acute. Many people with gall-stones merely complain of cramp in the stomach, which may be looked on as gastralgia, but is, in reality, rudimentary hepatic colic. The yellowish tint which often follows this so-called cramp in the stomach explains the nature and the origin of the mischief.

Jaundice is a frequent symptom of hepatic colic, provided the calculus blocks the common duct completely and for a sufficient time. The result is jaundice by retention some hours after the colic. If the obliteration of the common duct lasts long enough, the fæces have a whitish look, due partly to the absence of bile and to the presence of non-emulsified fats. The urine is loaded with bile pigment, and has a characteristic mahogany colour. Jaundice is, however, not constant: in forty-five cases of hepatic colic analyzed by Wolff where gall-stones had been found in the stools, jaundice was absent in twenty-five, proving that the stone may traverse the common duct without, however, completely obliterating it. The icteric tint may be very slight, and must be looked for carefully. We must not forget, too, that the seat of the trouble may be the cystic duct, in which case there will be no jaundice. Finally, we must remember that attacks of pain, mistaken for hepatic colic without icterus, may in reality be due to cholecystitis.

If the stools are examined by sifting them, the calculus or the calculi which have caused the complications, will be found, unless the calculus in the cystic duct has passed back into the gall-bladder, or has been pushed back from the duodenum into the stomach and rejected by vomiting. The calculi may not appear in the stools till three or four days after the attack. To find them, the stools must be carefully sifted; otherwise the calculi may pass unnoticed. The syndrome of hepatic colic may likewise be caused by worms or by hydatids impacted in the excretory canals, but these cases are very exceptional.

**Accompanying Symptoms.**—I have given the name of “satellite symptoms” to certain phenomena which sometimes accompany hepatic colic, or which may, indeed, exist without typical colic; in the latter event we have to deal with defaced hepatic colic.

Vertigo is one of these symptoms, and I may say that it is frequent, if trouble is taken to seek it. I do not know the exact pathogenesis, but it is certain that a fair number of persons with gall-stones suffer from vertigo, whereas I have not noticed this symptom in renal calculi. A few years ago I saw a lady in whom the attacks of hepatic colic were ushered in, accompanied, and even replaced, by attacks of vertigo, lasting for several days. The vertigo may be slight or intense, transitory or lasting; it must have frequently been confounded with gastric vertigo, the so-called cramps in the stomach often being abnormal hepatic colic. Vertigo due to calculus (as, by the way, all forms of vertigo) distresses patients, who believe that they are

threatened with "a stroke." Such was the case in a lady whom I saw some years ago with Jacquet. The patient, who suffered from gall-stones, dared not move from the bed to the sofa, because the feeling of vertigo and faintness was so strong; she fancied that she was threatened with "a fit of apoplexy." In the hospital, when questioning patients with hepatic colic, we find that vertigo is a marked symptom.

A tendency to syncope is met with in many cases of gall-stones. And I am not alluding to syncope caused by the excessive pain, but the prostration and distress, satellite symptoms of the passage of a stone, and of hepatic colic, which cause the victims to say that "they are going to faint." A satellite symptom of real importance is rigors and severe attacks of fever. The fever is not due to angiocholitis, cholecystitis, or abscess of the liver; in these cases the fever, which is the result and the index of biliary infections to be discussed later, has generally a grave prognostic signification, and furnishes valuable indications as to surgical intervention. The febrile complications that I am about to describe have another signification, as the following examples will show:

Some years ago I was called in consultation to a lady who was subject to hepatic colic. She had been ill for some days with pain, jaundice, decoloration of the fæces, and vomiting. The attack had so far been normal, but suddenly severe rigors appeared, with a rise of temperature to 104° F. and profuse sweats. The attacks returned every day, but not at a fixed hour. Quinine had no effect, and infection of the bile-ducts or abscess of the liver was feared. I felt justified in giving a favourable prognosis, and made a diagnosis of satellite attacks of fever due to the passage of gall-stones. As a matter of fact, a few days later the patient recovered from the colic and the fever, after having passed about a dozen large stones.

Another example: I had in my ward at the Necker Hospital a patient in whom the diagnosis was not obvious. The liver was somewhat enlarged and painful, and the conjunctivæ were yellowish. The patient was seized with a violent and prolonged rigor, such as occurs in malaria or lobar pneumonia, the attack lasting for fifteen minutes. The slight jaundice attracted my attention, and I questioned him regarding gall-stones. I learnt that he had had hepatic colic, and though the pains were only very slight, I diagnosed satellite fever due to the passage of stones. At the end of a quarter of an hour the temperature rose to 104° F., and the hot stage was followed by profuse sweating, which ended the attack. Two days later two gall-stones were found in the fæces, showing the nature of the case.

I have noticed these attacks frequently, and in hospital, on questioning my patients with hepatic colic, I have obtained a history of severe attacks of fever. I have dwelt on this variety of fever because it is not yet recognized, in spite of the labours of Charcot and Magnin. It is very important to distinguish it from other attacks of fever due to acute infection of the biliary passages.

The distinction between satellite fever and infectious fever has long been known. Pentray, Magnin, Charcot, and Besnier have stated "that the intermittent fever which supervenes in biliary lithiasis appears in two different



conditions, and may indicate two different morbid states." In order to distinguish these two varieties of intermittent fever, Charcot called the former hepatalgic fever, or the satellite fever of hepatic colic ; and the latter hepatic or biliary fever (of toxic origin). Even acute attacks of satellite fever may exist with slight forms of hepatic colic ; they are at times so severe that the colic is defaced and of minor importance. Magnin quoted a case in which the satellite fever, due to the passage of gall-stones, preceded the colic by several months. One attack of satellite fever is rare ; as a rule, we see a series of attacks, which return several days following, without well-marked periodicity. I am not certain of the cause of these satellite attacks. Charcot thought they were the result of slight infection, in which case they might be considered as a kind of bilio-septic fever. It is possible, but I am not convinced, because we see many people who are suffering from gall-stones, but in whom the attacks of satellite fever never end in bilio-septic fever and are never followed by infective lesions of the gall-bladder or of the liver.

The **prognosis** of the attacks is generally good ; although they cause great alarm, they generally end in recovery. We must not, however, be too optimistic, for attacks which have at first presented the characteristics of hepatalgic fever may have a fatal ending, as in the case reported by Besnier—an exceptional case, however, the patient having, in a few days, passed 148 stones. On the other hand, I saw a patient who had fifty-five satellite attacks, in series of six to eight intermittent attacks, with hepatic colic and jaundice, and yet recovered, without other complications occurring.

The **diagnosis** between satellite fever and bilio-septic fever is often difficult, but it may nevertheless be said that satellite attacks are generally associated with hepatic colic, pain and vomiting, while in bilio-septic fever these troubles are often absent. The satellite attacks are almost always followed by the passage of calculi, which is not the case in bilio-septic fever due to cholecystitis and angiocholitis. Finally, satellite attacks are often coexistent with other satellite symptoms due to passage of stones—vertigo, fainting-fits, and syncope—which is not the case in bilio-septic fever. Summary : It cannot be said of hepatic colic that it is always apyretic, whilst I have never noticed satellite attacks in renal colic.

In addition to the attacks of fever just described, we also find fever, due to biliary stagnation, in persistent obliteration of the common duct. This question will be discussed under Persistent Obliteration of the Common Duct.

**Complications.**—Hepatic colic is sometimes accompanied by complications. Even at the commencement of the attack rupture of the cystic and of the common ducts, followed by peritonitis, may occur. Sudden death in an attack is not quite unknown. It is probably due to reflex action, and at the post-mortem examination a large calculus is found impacted in the cystic or the common duct. Hepatic colic may be accompanied by vascular

troubles, which show themselves by pulmonary congestion on the right side, œdema of the lower limbs, and dilatation of the right side of the heart, with tricuspid insufficiency. The reaction of the biliary lesions on the right side of the heart has been pointed out by Potain. Gangolphe has indicated the existence of a murmur in jaundice, and placed it at the mitral opening; but it seems that this murmur should be located in the tricuspid opening. Potain has shown that acute affections of the biliary passages and calculous jaundice in particular may cause transitory dilatation of the right heart, with tricuspid insufficiency and hypertrophy of the ventricle. The dilatation is probably due to excess of pressure in the pulmonary artery, and depends upon diminution in the calibre of the arterioles of the lungs, the diminution being doubtless the result of reflex action transmitted to the bulb, and reflected to the lungs by the branches of the great sympathetic.

We shall see later that biliary lithiasis may cause infective endocarditis, through organisms present in the bile ducts and derived from the intestine.

**Diagnosis.**—The diagnosis of hepatic colic is generally easy. It is distinguished from renal colic in that the liver is not painful; the pain starts from the kidneys, follows the course of the ureters, and spreads to the testicles, the neck of the bladder, and the extremity of the penis, and jaundice is absent. Idiopathic hepatalgia, or simple hepatic neuralgia (which Beau thought so frequent), is very rare, since gall-stones have, after careful search, been almost always found in the stools shortly after the attack of colic. The diagnosis from gastralgia is sometimes difficult; some patients merely complain of cramp in the stomach, but closer examination shows that the so-called cramp means hepatic colic. The urine contains bile pigment, the conjunctivæ become yellow, the liver is enlarged and painful, and the pain reaches the right shoulder. When the diagnosis of biliary lithiasis is difficult, urobilinuria and peptonuria (Bouchard) are in favour of it.

I may mention the diagnosis of renal from lead colic, and I would insist on the diagnosis of defaced hepatic colic, which may be painless, the presence and the migration of the calculus being shown by rigors, fever, vertigo and syncope. The diagnosis from the pain of intestinal lithiasis may present some difficulties. In order to avoid repetition, I refer the reader to the section on Entero-typhlo-colitis.

The diagnosis of hepatic colic from **appendicitis** has been already discussed. Hepatic colic must not be confounded with hepatic pseudo-colic, which is due to adhesive pericholecystitis.

The **prognosis** of hepatic colic must always be reserved—first, because the gravest complications, such as perforation of the bile-ducts, syncope, and sudden death, are possible during the attack; and, secondly, because the calculi may give rise to a series of complications, described in the following sections.



**Treatment.**--The objects in view are--(1) to relieve the pain; (2) to facilitate the expulsion of the calculus; and (3) to prevent the formation of new calculi. In order to ease the pain, aspirin may be given in doses of 30 to 60 grains daily. Subcutaneous injections of morphia are of much service. To these means may be added enemata of chloral and the application of ice-bags to the hypochondrium. Prolonged baths also give good results. In order to facilitate the expulsion of the calculus, large quantities of oil and massage of the hepatic region have been recommended (Pujol). Alkalis have a twofold action: they facilitate the expulsion of the calculi and prevent the formation of new ones. With this object in view, cures at Vichy, Carlsbad, Contrexéville, and Vittel may be described. Durande's remedy, given in perles containing 3 parts of ether to 2 parts of essence of turpentine, was frequently used by Trousseau. The patient must be careful to avoid fat, acid foods, and drinks.

## 2. Migration and Arrest of the Biliary Calculi in the Intestine— Stenosis and Obliteration of the Pylorus.

**Intestinal Obstruction.**--Obstruction of the intestine by gall-stones is not extremely rare, because Dragon has collected 140 cases. In order to cause obstruction, the calculus must be as large as a walnut or an egg. The passage of these large calculi remains to be explained. While large stones can pass through the bile-ducts, when much dilated, the majority of these stones pass into the intestine through a fistula between the gall-bladder and the duodenum.

The reason for the belief that these large calculi have not passed through the bile-ducts is that patients may have previously experienced neither hepatic colic nor jaundice. Some of them, however, have had symptoms of calculous cholecystitis. The gall-bladder forms adhesions with the intestinal coil (pericholecystitis); a large fistula is established, and the calculus then passes from the gall-bladder into the intestine. Sometimes the calculus passes into the intestine without encumbrance; at other times it causes grave mischief. In the latter event the symptoms of intestinal occlusion are sudden, but in a third of the cases recovery supervenes spontaneously, and the calculus or calculi are passed *per anum*. When intestinal occlusion persists, symptoms of peritonitis are often present. The calculi are generally arrested in the jejunum, the ileum, or the lower portion of the rectum. In some cases it has been found that the large size of the calculi was due to the addition of calcareous deposits of fæcal matter. Intestinal spasm is also an important factor in the pathogenesis of obstruction by gall-stones. The following cases will give an idea of these complications:

Merklen: A woman with no previous history of gall-stones was suddenly taken ill with colic, vomiting, arrest of fæces, and flatus, followed by fæcal vomiting, a cholera-

like condition, and lowering of the temperature. After a short respite the symptoms again became acute; hiccough, tympanites, and chilliness were present, and the patient succumbed. The occlusion was seated in the small intestine, and was due to an enormous calculus, measuring more than 3 inches in circumference. It had passed into the intestine through a cystico-duodenal fistula. The gall-bladder contained another large calculus.

**Audry:** A patient was taken suddenly ill with symptoms of intestinal occlusion: sharp abdominal pain, vomiting, absolute constipation, and meteorism. A respite then supervened, but was soon followed by rapid aggravation of the symptoms, and the patient succumbed in an algid condition. At the post-mortem examination recent peritonitis and obstruction of the small intestine, due to an enormous calculus, weighing  $1\frac{1}{2}$  ounces, and measuring  $4\frac{1}{2}$  inches in circumference, were found. The calculus had entered into the intestine through a cystico-duodenal fistula.

**English Cases.**—Maclagan reported to the Clinical Society of London the case of a patient who was seized on four occasions with sharp abdominal pain and constipation. The attacks lasted from three to six days, and after each attack the patient passed gall-stones of the size of a walnut. The patient died, and at the post-mortem examination a cystico-duodenal fistula was found. The gall-bladder contained another large calculus. Broadbent had a case of intestinal occlusion in an old man who succumbed in four days. At the post-mortem examination an enormous gall-stone was found in the small intestine. Ord saw three analogous cases. Enormous gall-stones were arrested at the end of the ileum in the first case, in the small intestine in the second case, and at the anal sphincter in the third case. Harrington published a case of an aged woman with symptoms of intestinal occlusion. She was operated on, and a gall-stone was found in the ileum.

We see that intestinal occlusion, caused by gall-stones, supervenes in old people who have not suffered from hepatic colic or from any symptoms indicative of the passage or the arrest of the calculi in the bile-ducts. These large calculi are slowly formed in the gall-bladder, which becomes infected, and enter the intestine generally through a cystico-duodenal fistula. In ninety-two cases collected by Lobstein, the symptoms of intestinal occlusion were preceded in seventeen cases only by symptoms of cholelithiasis.

The treatment is surgical. According to Lobstein's statistics, out of sixty-one cases which were operated on, twenty-nine ended fatally; and out of thirty-one cases, nineteen succumbed. It must be added that most of the operations in which death supervened were performed on exhausted patients with peritonitis. In order to have the best chances of success, the operation should be performed as early as possible.

**Stenosis and Obstruction of the Pylorus.**—The close relation between the gall-bladder and the pylorus explains the pathological connections of these two organs in calculous cholecystitis, which may react in different ways on the pylorus and its orifice. Sometimes a large calculus from the perforated gall-bladder becomes embedded in the wall of the pylorus, forming a pouch for itself, and obliterating the orifice. In other cases the calculus is juxta-pyloric, and causes fibrous changes in the pylorus, with stenosis. Marchant quotes three such cases in his work.



The contraction of the pylorus may not be due directly to the gall-stone ; it may be due to adhesions and to perigastritis, which is secondary to calculous pericholecystitis.

Summary : Calculous cholecystitis may cause stenosis and obstruction of the pyloric orifice, either directly by the presence of large calculi, or indirectly by adhesions, by retraction of fibrous tissue, or by perigastritis, consequent on pericholecystitis. The more or less marked contraction of the pyloric orifice shows itself by the following symptoms : epigastric pains ; vomiting several hours after the ingestion of food ; considerable dilatation of the stomach, with tympanites and "clapotement" ; constipation ; loss of flesh due to insufficient nourishment ; cachexia ; induration or tumour of the epigastric region.

A patient vomits, loses flesh, and becomes cachectic. He complains of sharp pains in the epigastric region. On examination, we find dilatation of the stomach and pyloric tumour, and we cannot eliminate at first the idea of cancer ; the induration, and particularly the tumour, obscure the diagnosis. After all, vomiting, progressive loss of flesh, cachexia, pain, induration, tumour, and dilatation of the stomach may all be present in pyloric stenosis, whether it is due to ulcer, cancer, perigastritis, adhesions, biliary calculi, etc. ; and if the patient has a history of hepatic colic, jaundice, or signs of cholecystitis, we must think of pyloric stenosis, consequent on calculous cholecystitis, and have recourse to prompt surgical intervention.

### 3. Persistent Obliteration of the Bile-Ducts—Biliary Cirrhosis.

Hepatic colic is only accompanied by a temporary and sometimes incomplete obliteration of the cystic and common ducts. I shall now deal with the persistent obliteration of these canals, and with the many complications resulting therefrom.

When the cystic duct is permanently obstructed by a calculus, the bile no longer reaches the gall-bladder ; the bile already present is absorbed ; the walls of the gall-bladder, as the result of chronic inflammation, become thickened and converted into fibrous tissue ; and the gall-bladder atrophies. In some cases the walls of the gall-bladder become infiltrated with lime-salts. In other cases the bile gives place to a sero-mucous secretion, and the gall-bladder (hydrops) may be much enlarged. The fluid in hydrocholecystitis is mucous, shreddy, whitish, and sometimes rich in muco-pus. As the obstruction of the cystic ducts allows the free passage of the bile into the intestine, this complication is by no means as serious as obstruction of the common duct.

When the common duct is permanently obliterated by one or more calculi, it matters little whether the obliteration occurs at some point of its course or at Vater's ampulla ; the bile no longer passes into the intestine,

and chronic jaundice appears. There are, nevertheless, examples where, in spite of the presence of several calculi (Cruveilhier), the common duct has remained sufficiently patent to allow the bile to pass into the duodenum.

Obliteration of the common duct, as also of the cystic duct, is not always preceded by colic ; the obliteration may come on silently, without pain. Persistent obliteration may last for weeks and months, without other symptoms than chronic jaundice, with clay-coloured stools, distension of the gall-bladder, and enlargement of the liver. Sometimes, also, persistent obliteration of the common duct may at length cause lesions in the liver, gall-bladder, and ducts ; dilatation and suppuration in the bile-ducts and gall-bladder ; fibrosis of the liver (biliary cirrhosis) ; suppurative angiocholitis and abscess of the liver ; and changes in the hepatic cells (parenchymatous hepatitis and pancreatitis).

**Dilatation of the Ducts and Gall-Bladder.**—In consequence of complete and permanent obstruction of the common duct, the cystic and hepatic ducts may undergo enormous dilatation, while the common duct may be as large as an intestinal coil ; the bile accumulates in the gall-bladder, which may, then, descend as far as the umbilicus, and even into the right iliac fossa. The distended gall-bladder may contain several pints of fluid. Cruveilhier in his atlas has figured a gall-bladder descending into the right iliac fossa ; and in Benson's case the gall-bladder was punctured, under the impression that the case was one of ascites, 4 pints of bile being withdrawn. The fluid in obliteration of the common duct does not resemble the thready mucous liquid of hydrocholecystitis due to obliteration of the cystic duct.

The obstruction of the common duct also determines enlargement of the superficial and deep intrahepatic ducts, and the latter assume a cylindrical or ampulla-like form, analogous, says Monneret, to bronchial dilatations. The dilatation is sometimes so general that the parenchyma of the liver resembles cavernous tissue. The retention of the bile causes great enlargement of the liver ; the surface is smooth and of an olive colour. On section, the bile-ducts are seen to be dilated and to exude bile, which is often mixed with muco-pus, sand, and biliary concretions, which have formed owing to the stagnation of the bile. The biliary canals are attacked with chronic inflammation (angiocholitis), which commences in the mucosa, and later involves the entire wall of the duct.

Chronic jaundice, clay-coloured fæces, swelling of the liver which may descend as far as the umbilicus, and distension of the gall-bladder, are the first results of the permanent obstruction of the common duct. The fæces may, however, from time to time regain their colour, if the calculus or the calculi engaged in the common duct do not completely obstruct the lumen, and allow an intermittent flow of bile into the intestine. When these complications supervene in an individual who has had hepatic colic, it is



easy to trace the cause of the lesion ; but when the symptoms of lithiasis are not clearly defined, it is necessary to eliminate prolonged catarrhal jaundice, primary cancer of the bile-ducts, and cancer of the head of the pancreas and of Vater's ampulla. The clinical side of the question will be discussed in Section XXI.

**Biliary Cirrhosis.**—Obstruction of the common duct produces enlargement of the liver, but this hypertrophy is often replaced by atrophic cirrhosis. In no case does this cirrhosis resemble Laënnec's atrophic cirrhosis ; the surface of the liver is smooth, and the parenchyma is but moderately indurated ; it is therefore a special cirrhotic process. Experimental obliteration of the common duct in animals by a ligature, and the pathological obstruction of this canal in man by primary cancer of the biliary passages, or by cancer of the head of the pancreas, cause similar cirrhotic changes. The retention of the bile produces angiocholitis and peri-angiocholitis, the process ending in diffuse interstitial hepatitis. This connective hyperplasia affects at first the large ducts, and then the smaller canaliculi. It shows itself first in the spaces, next in the fissures, and circumscribes the hepatic lobule, without ever ending, like Laënnec's atrophic cirrhosis, in the formation of nodules. Formation of biliary canaliculi, which invade the lobule, pigmentation of the cells, and invasion of the lobule by connective hyperplasia, are also found.

The differences are great between hypertrophic biliary cirrhosis (Hanot's disease) and the biliary liver. The latter is not hypertrophied, but is rather small and contracted ; dilatations of the biliary canals are often found, with or without abscesses, and the angiocholitis commences in the large bile-ducts, which are healthy in hypertrophic biliary cirrhosis.

These lesions may, like the various diseases of the liver, become complicated at any moment by symptoms of icterus gravis, which causes death in a more or less rapid manner. Moreover, no matter what are the lesions of the liver, no matter what are the causes of its anatomical and physiological decay, as soon as it is unable to fight, it is invaded by secondary infections (microbes and toxins), and the complex symptoms of hepatic insufficiency appear.

**Pancreatitis.**—This question is discussed later, in Section VII. of Diseases of the Pancreas.

#### 4. Infection of the Biliary Passages : Angiocholitis, Cholecystitis, Hepatitis, Endocarditis.

In the normal state, the bile-ducts, the gall-bladder, and the bile are aseptic. In animals the bile in the gall-bladder is aseptic. The effusion of pure bile into the peritoneum does not cause peritonitis. Normal bile is, therefore, aseptic and sterile. The bile is not antiseptic, and does not

destroy germs, as has been supposed. As far as the microbes are concerned, it is as favourable a culture medium as ordinary broth, and does not lessen their virulence in any way. The coli bacillus, streptococcus, staphylococcus, etc., grow readily in the bile (Gilbert and Dominici).

How, then, does infection of the biliary passages take place? I said that the canals were aseptic, but the last portion of the common duct forms an exception. Numerous microbes have been found there, especially the coli bacillus; they come from the duodenum, which is very rich in germs (Gessner). The common duct is, therefore, in danger of being infected, and the duodenum is a constant menace to the bile-ducts. In the normal condition, the flow of the bile mechanically maintains the asepsis of the ducts, but any obstacle to the flow favours invasion of the biliary canals by the microbes normally present in the duodenum, and the terminal portion of the common duct. The infection ascends from the ducts to the gall-bladder and to the liver.

By ligaturing the common duct, biliary infection has been reproduced experimentally (Gilbert, Netter, Girode). The ligature produces at the same time arrest of the flow of bile and trauma of the walls, two conditions favourable to the entrance of microbes. These experimental conditions are fulfilled pathologically by the obstruction of the common duct; calculi, by obliterating the canal, prevent the exit of the bile, and cause traumatic erosion of the mucosa; the door is, then, open to infection, and the soil is prepared. In this way angiocholitis, cholecystitis, and abscesses arise.

Calculous infection of the biliary passages generally occurs in people who have had colic, jaundice, swelling of the liver, pains in the hypochondrium, etc. These symptoms may, however, be absent, or may have disappeared a long time before the symptoms of infection appear. Fever is sometimes the prominent feature. We have to deal with true infective fever, which is also intermittent, characterized by more or less violent rigors, with a rise of temperature to 103° or 104° F., and followed by profuse sweating after the attack; these attacks recur every night, or every other night. The apyrexia between the attacks may be complete (intermittent form) or incomplete (remittent form); or, again, the fever may show but slight remissions (continuous form), and is, then, more serious. Intermittent fever may accompany all the localizations of biliary infection, but it is in hepatic infection that it attains its greatest intensity, and it is much less marked or fairly often absent in the case of cholecystitis.

**Angiocholitis—Hepatitis.**—Angiocholitis may attack the extra- and intrahepatic bile-ducts. In the latter case lesions of the liver are the most important. We find post-mortem that the liver is soft, enlarged, and surrounded by adhesions, due to simple or to suppurative perihepatitis. On



section it seems to be converted into a spongy, purulent tissue; the bile flows in abundance through the dilated ducts, and the hepatic parenchyma is riddled with abscesses of various forms and sizes. Some are miliary; others are larger than an orange. Some of them have been well described by Chauffard under the name of areolar abscesses. These biliary abscesses have several origins: some are due to the cylindrical, moniliform, or ampullary dilatations of the biliary canaliculi, and are not true abscesses; while others, true abscesses, are much more common, and arise in the connective tissue around the ducts (suppurative peri-angiocholitis), whether the walls of the duct have been destroyed by the suppurative inflammation or whether the abscess has ulcerated into the bile-duct. The rupture of a dilated bile-duct in the parenchyma of the liver may also become the origin of a large abscess. The pus is whitish, yellowish-green, or brownish, and contains biliary sand and débris of the hepatic tissue; the purulent collection has no limiting membrane, or, if the membrane is found, it is never lined with cylindrical epithelium. If cylindrical epithelium is found in the pus, it indicates communication between the abscess and a biliary canaliculus. Micro-organisms, especially the *Bacillus coli*, abound in the pus from these biliary abscesses.

**Calculous Cholecystitis.**—In exceptional cases the calculi may be formed in the liver, but they are almost always formed in the gall-bladder. The number may be considerable; sometimes there is only one, which may be larger than an egg. The calculi may remain for a long time in the gall-bladder without causing cholecystitis in the true sense of the word. This calculosis of the gall-bladder becomes calculous cholecystitis when the bladder is infected.

Calculous cholecystitis shows itself in different forms. In the first and most common type the gall-bladder diminishes in size; it may even be reduced to "a calculous stump." This condition is due to atrophying sclerosis of the walls. The fibrous walls are sclerosed and retracted on to the calculi. On the inside, the gall-bladder is furrowed with folds and bands, dividing it into partitions, and giving it the look of a beehive. In the alveoli of the hive larger or smaller calculi are embedded in a purulent cavity, and are sometimes very difficult to enucleate. An infiltration of embryonic cells is found in all the layers of the gall-bladder, and numerous micro-organisms are also found. No tumour is found, the gall-bladder being retracted under the liver. As it does not reach below the lower edge of the liver, and as it cannot be felt on exploring the region, it gives no aid in the diagnosis.

In the second type of cholecystitis, which is less common, a tumour is found. The walls of the gall-bladder are "as hard as a cardboard shell," much hypertrophied and thickened, and the cavity is enlarged. The size

of the cystic tumour is not due to the fluid, which is only present in small quantities ; it results chiefly from the enormous thickness of the walls. These cases of calculous cholecystitis make a large projection under the liver ; the tumour can be felt on examining the lower edge of the organ.

Besides the two preceding types, represented by sclero-atrophic cholecystitis, which is very common, and by sclero-hypertrophic cholecystitis, which is very rare, a third type is found ; it is characterized by great dilatation of the gall-bladder, with thinning of the walls and accumulation of much fluid in the cavity. This dropsy of the gall-bladder is generally associated with obliteration of the cystic duct. The liquid is colourless, shreddy, or tinged with bile. The mucosa of the gall-bladder is not alveolar, as in the preceding forms, but smooth, like a true cyst wall (Schwartz). The tumour may be pedunculated, and the obliterated cystic duct forms part of the pedicle ; the tumour descends into the abdomen, and simulates other abdominal swellings.

The infection of the gall-bladder is caused by the mechanism referred to above, whether the obliteration is in the common duct or in the cystic duct ; cases have been recorded where the gall-bladder became infected in the absence of any obliteration. The lesions of calculous cholecystitis are not always identical in these different forms. The liquid in the infected gall-bladder is generally scanty. It may be scarcely clouded with mucus, or it may be purulent, sanious, foetid, and coloured or not by bile (empyema of the gall-bladder). The gall-bladder is the more distended, the more its walls are thinned and the less they are sclerosed. Miliary abscesses are often found in the walls.

The different forms of cholecystitis just described are often followed by pericholecystitis. Adhesions form between the bladder and the neighbouring organs (intestine, pylorus, cystic duct, etc.) ; ulcerations, perforation, and fistulæ appear, and are followed by passage of large calculi into the intestine and intestinal occlusion, calculous peritonitis, stenosis of the pylorus, etc.

**Symptoms and Diagnosis.**—Let us take the simplest case first. A patient has suffered for several years from hepatic colic, verified by the presence of gall-stones in the stools. The pains have later lost the classical characteristics of hepatic colic. The region of the liver has become painful. Sudden movements, jolts, or tight clothing cause pain in the hypochondrium. The digestive functions are disturbed. Sometimes vomiting of bile and bilious diarrhœa are seen. Jaundice is absent, and the urine does not contain bile. Fever, with or without rigors, is common ; the tongue is dry and the appetite lost. At certain times attacks of pain resembling hepatic colic occur, and the patient says : “ A swelling is forming under the liver.”

The patient is examined in the erect and supine positions. I would



specially recommend the erect position, because it is very favourable for the discovery of tumours and deformities of the abdomen. A tumour appreciable to sight and touch, and painful on pressure, is sometimes seen under the edge of the ribs, external to the rectus muscle, at the tenth costal cartilage.

In such a case it is reasonable to diagnose calculous cholecystitis—a diagnosis that may be confirmed by radiography. During the last few years I have seen three similar cases. In the three cases the inflamed gall-bladder formed a tumour below the liver. Notwithstanding recent improvements, radio-diagnosis of gall-stones rarely gives positive results; the main reason for this fact is of a chemical order. Every calculus which contains a sufficient quantity of phosphates or of salts of calcium can be detected by radiography. “In the case of gall-stones, on the contrary, most of them are formed of cholesterine, or of biliary pigments, and are poor in calcium. It is only in exceptional cases that certain calculi of the gall-bladder, following on inflammation of the mucosa, are surrounded by a shell of phosphate or carbonate of calcium. These exceptional calculi are about the only ones which radiography reveals.” To sum up: All gall-stones must contain a notable quantity of calcium, and this is an essential condition, in order to be revealed in the living subject by radiography. The diagnosis of calculous cholecystitis is sometimes very difficult. For example, a patient complains of sharp pains in the hypochondrium, vomiting, anorexia, fever, and loss of flesh. The liver is not enlarged; the lower edge of the organ is painful on pressure, but neither bulging nor tumour in the gall-bladder region is found, because the gall-bladder is hidden under the liver, and is consequently inaccessible to exploration. Doubt exists as to the nature of the disease. We may, however, arrive at a diagnosis if we find a history of hepatic colic, as it is, then, probable that we have to deal with a case of calculous cholecystitis. Pellereau and myself were thus able to make a diagnosis in the case of a woman who was successfully operated on by Tuffier. The calculus was as large as a walnut, and the gall-bladder was hidden under the liver. Finally, the diagnosis of calculous cholecystitis presents difficulties of another kind, when the gall-bladder, by its size or by its displacement, simulates hydatid cyst, cancer of the liver, tumours of the peritoneum or of the kidneys, or an aberrant lobe of the liver. All these mistakes in diagnosis have been made. If the reader will refer to the section on Aberrant Lobe of the Liver, he will find the difficulty in diagnosis discussed.

Calculous cholecystitis may become the cause of a series of complications, such as passage of large calculi into the intestine and intestinal occlusion; obliteration of the pyloric orifice; perforation of the diaphragm, and rejection of the biliary contents through the bronchi; superacute peritonitis, pyelitis, endocarditis, etc.

**Pericholecystitis and Adhesions.**—For fuller details, I would refer the reader to Section XXIII., on Cholecystitis in General. A large number of cases will be found there which prove that, twenty-five times at least out of thirty, the symptoms due to biliary lesions open the scene, and the symptoms of appendicitis appear only secondarily, some days or weeks later. It is essential to know how to make the diagnosis of this morbid association, so as to be able to intervene *secundum artem*.

**Treatment.**—When the diagnosis of calculous cholecystitis has been made, recourse must be had to surgical intervention. The many complications already described show the danger of undue delay. I need not discuss what kind of operation should be undertaken. The progress made in this direction during recent years has been considerable. **Cholecystotomy** is sometimes performed, and consists in opening the gall-bladder, removing the calculi, and establishing an external biliary fistula, which closes later. Sometimes cholecystectomy is performed; this operation consists in resection of the gall-bladder. In other cases cholecystenterostomy is performed (especially in the obliteration of the common duct); it consists in joining the gall-bladder directly to the duodenum or the ileum.

**Pylephlebitis—Aneurisms.**—Calculous obstruction of the biliary passages and the lesions of angiocholitis which it causes are sometimes accompanied by pylephlebitis (infection of the portal vessels). Pylephlebitis may attack the trunk of the portal vein or the secondary branches. The close relation of the branches of the portal vein to the biliary canals explains the possibility of portal phlebitis, consecutive to peri-angiocholitis. In the same way, the inflammation of the portal trunk, secondary to the lesions of the common duct, may be explained.

Pylephlebitis may be obliterating or suppurative. Obliterating phlebitis is very much the rarer; when it affects the portal trunk, we find ascites, swelling of the spleen, and collateral circulation of the abdomen. When the pylephlebitis is suppurative, it is accompanied by attacks of intermittent fever, and multiple abscesses of the liver are found post mortem.

In some cases the arteries of the neighbourhood may be affected by enlargement and ulceration, causing fatal attacks of hæmorrhage (hæmatemesis and melæna).

**Biliary Endocarditis.**—As I have already said, biliary lithiasis favours the infection of the biliary passages: angiocholitis, pylephlebitis, cholecystitis, and hepatitis. The pathological agents being readily transported into the heart, endocarditis is a common result. The left side of the heart is usually affected, especially at the mitral and aortic valves. The symptoms of this endocarditis, which often assumes the ulcerative type, are generally indefinite; the fever and the jaundice that accompany it, indicate the onset of the lesions, which, through lack of attention, may pass unnoticed.



### 5. Peritonitis—Biliary Fistulæ.

Let us now consider cases of partial and general peritonitis, due to perforation of the gall-bladder and bile-ducts and to the passage of micro-organisms into the peritoneum.

**Peritonitis.**—The varieties of peritonitis, which for short I call calculous peritonitis, are of diverse kinds. We find partial peritonitis, which is limited by adhesions, uniting the gall-bladder to the stomach, duodenum, omentum, colon, and abdominal wall. Purulent cloacæ, which may fuse later, are thus formed. The diagnosis is very difficult in these cases. The adhesions are sometimes so thick that they form a tumour around the gall-bladder. Clinically speaking, these cases of partial peritonitis are much less serious than acute general peritonitis, which will now occupy our attention. Trousseau has reported three cases :

A rich shopkeeper of Tours, under Bretonneau's care, was suddenly taken ill during an attack of hepatic colic which had lasted for six days, with uncontrollable vomiting, and every sign of acute peritonitis, which proved fatal in twenty-four hours. The autopsy revealed a gall-stone as large as a walnut in the peritoneal cavity, and in the common duct was the perforation by which the stone and a certain quantity of bile had made their exit.

"About eight years ago," says Trousseau, "I was attending an old notary, who had been subject to attacks of hepatic colic for some time. I was called in one day because the symptoms were more severe than usual. He was vomiting incessantly, the belly was distended, the urine was entirely suppressed, the pulse was scarcely perceptible, and the temperature was subnormal. He had, in fact, all the symptoms of superacute peritonitis. I considered the case hopeless, and, as a matter of fact, the patient succumbed next day. Although no post-mortem examination was allowed, I feel justified in stating that it was a case of peritonitis, caused by effusion into the peritoneum, following rupture of the gall-bladder, or of one of the bile-ducts."

Werner's case : "I was called," says he, "to a patient who had extremely acute hepatic colic. I diagnosed gall-stones, and ordered treatment. Next day, as the pains were worse, and peritonitis had set in, I suspected a rupture of the gall-bladder. The patient died two days later, and at the post-mortem examination I found twenty-five stones as large as hazel-nuts in the gall-bladder ; the bladder was perforated, and the bile had flowed into the peritoneum. A calculus larger than the others blocked the common duct."

Calculous peritonitis is a most treacherous complication. It is due to perforation of the large bile-ducts, and especially to perforation of the gall-bladder. It may come on during an attack of hepatic colic, and as the symptoms in each case are similar (violent pain and vomiting), it may cause serious mistakes. More frequently it appears during the course of obvious, suspected, or latent cholecystitis. The prognosis is fatal, unless speedy laparotomy can be performed. The pathogenesis of these complications has yet to be studied. The peritonitis is due to infection by the bile, fluid, or calculi ; the *Bacillus coli* is the most common and most active agent

in this affection. How does the peritoneum become infected? Is there always a rupture of the biliary passages? and if so, how is this rupture caused? It might be supposed that if the gall-bladder is affected with calculous cholecystitis and ruptures, it is because its cavity is greatly distended and its walls are very thin. Examination of cases proves, on the contrary, that the gall-bladder is habitually contracted, and that its walls are thickened, hypertrophied, and brawny. The mucosa of the gall-bladder, however, presents numerous alveoli, formed by hypertrophied fibro-muscular bands. In these alveoli, which are of various forms and sizes, we often find large or small calculi that are difficult to enucleate, and are, as it were, let into the wall. The alveoli also contain septic fluid; they are, therefore, an excellent culture medium for micro-organisms. The result is an ulcerative process which may lead to perforation and peritonitis. Seuvre and Bouchard have described this ulcerative process in the alveoli of the mucosa, but we now understand the mechanism better, through bacteriological researches. The *Bacillus coli* is the essential agent in the ulcerative process, and in certain cases, just as in appendicitis, the organisms imprisoned in these closed alveoli pass through the walls of the gall-bladder, and spread afar the peritoneal infection, previous perforation of the walls not being absolutely necessary.

**Biliary Fistulæ.**—The process which has favoured adhesions between the gall-bladder and the neighbouring organs frequently terminates in ulceration and perforation, and gives rise to fistulæ which give exit to the contents of the bladder and to the calculi. These fistulæ may be external or internal, and either spontaneous or secondary to surgical intervention. In Murchison's statistics we find eighty-nine cutaneous fistulæ, thirty-six duodenal fistulæ, nine colic fistulæ, and twelve gastric fistulæ; in addition to the foregoing varieties, renal, vaginal, pleuro-pulmonary, and hepato-bronchial fistulæ have been found.

Hepato-bronchial and cystico-bronchial fistulæ explain the rejection, through the bronchi, of bile, pus, and calculi. The close proximity of the pleura explains biliary pleurisy (Gilbert and Lereboullet).

The cutaneous fistulæ have a fairly long, anfractuous, and irregular course; they usually occur in the umbilical region and the right hypochondrium. The formation of the cutaneous opening is usually preceded by cellulitis of the wall, and the orifice gives exit to bile, pus, and often to calculi. These fistulæ generally heal.

The cystico-duodenal are more frequent than the cystico-colic fistulæ, and it may be said that many large gall-stones found in the stools have passed through intestinal fistulæ. The calculi are sometimes so large that they provoke symptoms of intestinal occlusion. The cystico-gastric fistulæ explain how gall-stones may be vomited. It is, however, not impossible for



a calculus to pass back into the stomach during an attack of hepatic colic, and to be vomited later. In the celebrated case of Ignatius de Loyola there was a communication between the common duct and the portal vein.

## XXI. PERMANENT OBLITERATION OF THE COMMON DUCT— DIAGNOSIS OF THE CAUSE OF THE OBLITERATION.

In permanent obliteration of the common duct it is essential to diagnose the cause of the obliteration. Prognosis and treatment are equally concerned. If the duct is obliterated by a calculus, an operation is indicated, and recovery may be expected. If it is obliterated by cancer, the prognosis is hopeless, in spite of the temporary improvement that may follow an operation. By what signs and symptoms can we make this pathogenic diagnosis? How are we to recognize the nature of the obliteration? I shall attempt to answer these questions, utilizing two clinical lectures which I have devoted to this subject.\*

In the first place, every permanent obliteration of the common duct shows itself by an invariable syndrome, no matter whether the obliteration is due to calculus, to cancer of the biliary passages, of Vater's ampulla, or of the head of the pancreas, or to compression by a neighbouring tumour. This syndrome includes jaundice, the presence of much bile pigment in the urine, and clay-coloured fæces. The jaundice may be accompanied by intolerable itching.

Of the lesions causing permanent obliteration of the common duct, some are rare, others are common. Amongst the former, let me mention hydatid cysts of the liver, hypertrophy of the glands of the hilum, cicatrix of an ulcer of the duodenum, bands and adhesions in the neighbourhood, which all cause retention of bile. The most frequent causes are prolonged catarrhal icterus, cancer of Vater's ampulla, and primary cancer of the biliary passages. Of all these causes, the most common are cancer of the head of the pancreas and chronic pancreatitis.

In the pathogenic diagnosis we must first eliminate hypertrophic biliary cirrhosis (Hanot's disease) and chronic icterus, with enlarged liver and enlarged spleen, which is distinguished from the syndrome that we are discussing, by the coloration of the fæces and by the uninterrupted flow of bile into the intestine.

**Permanent Obliteration of the Common Duct by Prolonged Catarrhal Icterus.**—I have given the name of "prolonged catarrhal icterus" to a variety of infective icterus (with or without remissions), which may last two or three months. In prolonged catarrhal icterus we must not forget

\* "Oblitération Permanente du Canal Cholédoque" (*Clinique Médicale de l'Hôtel-Dieu*, 1898, 11<sup>me</sup> et 12<sup>me</sup> leçons).

that, besides the icteric syndrome common to every permanent obliteration of the large bile-ducts (hepatic and common), we may also find anorexia, loss of flesh, and swelling of the liver, and that the disease, in spite of its threatening appearance, generally ends in recovery. The diagnosis is sometimes very difficult. When we see a patient who has been ill for two months or more with jaundice, urobilinuria, clay-coloured stools, enlargement of the liver, and rapid loss of flesh, we naturally think of obstruction of the common duct, due to cancer of Vater's ampulla or of the head of the pancreas. I have often been anxious in cases of this kind as to the outcome of the disease.

**Permanent Obliteration of the Common Duct by Cancer of Vater's Ampulla.**—The small cancer of Vater's ampulla, already described, may cause permanent obliteration of the common duct. The growth remains limited to the ampulla, has no invading tendency, and soon betrays its presence by the "icteric syndrome." It may even be said that jaundice is the first apparent symptom, and is due to obstruction of the orifice of the common duct by the epitheliomatous tumour. It has all the attributes of icterus due to permanent obliteration: yellowish or olive colour of the skin, bile-stained urine, and colourless fæces. At times the fæcal matter may regain its colour and the jaundice become paler, proving that the orifice of the common duct recovers its permeability for the moment. The liver is enlarged; the gall-bladder, though greatly distended, cannot always be felt through the abdominal walls. Pain, either spontaneous or provoked, is a rare symptom; it is, however, seen in some cases. Rendu's patient had acute attacks of pain in the right hypochondrium and the epigastrium. Palpation was so painful at the pit of the stomach and over the left lobe of the liver that a calculus was thought to be impacted in the common duct. The post-mortem examination showed the absence of calculi and the existence of a cancer of Vater's ampulla.

In such a case the icteric syndrome (permanent jaundice, bile in the urine, and clay-coloured stools, with enlarged liver) may persist for months. How, then, are we to diagnose the cause of the obliteration in the common duct? The diagnosis is very difficult. It may be said that in the case of cancer of Vater's ampulla icterus and decoloration of the stools sometimes undergo remissions, but the transient remissions are of no value in the diagnosis, for they are met with in other cases of obliteration. On the other hand, it may be said that pain is an especial feature in gall-stones; but cancer of Vater's ampulla may also provoke pain similar to that of gall-stones, as in Rendu's case. It has been said that diarrhoea is in favour of cancer, but in reality this symptom is of no more value than the preceding ones; it allows us neither to accept nor to reject the hypothesis of cancer of Vater's ampulla. Intestinal hæmorrhage is of more value. When the growth blocks Wirsung's duct, stearrhoea and examination of the fæces by Gaultier's method give useful information (*vide* Section VII., Diseases of the Pancreas).



**Permanent Obliteration of the Common Duct by Primary Cancer of the Biliary Passages.**—A small growth in the hepatic or common duct may block the canal and give rise to intense and prolonged jaundice, bile in the urine, colourless fæces, rapid loss of flesh, and sometimes pain. Primary cancer of the gall-bladder only causes the syndrome in question when it spreads to the large bile-ducts or to the adjacent glands, and thus obstructs the flow of bile; as primary cancer of the gall-bladder is associated with the formation of gall-stones, the result is that in three-fourths of the cases true hepatic colic may be associated with the icteric syndrome—a fact that greatly complicates the diagnosis.

Primary epithelioma of the common or of the hepatic duct is not rare. In either case the icteric syndrome is present, but the condition of the gall-bladder differs. If the cancer is in the common duct, the accumulation of bile in the gall-bladder may cause considerable distension; if the growth is in the hepatic duct (Claisse), the gall-bladder is not dilated. In primary cancer of the biliary canals jaundice is sometimes preceded by loss of flesh. The appearance of jaundice is sometimes sudden, sometimes progressive; when it has appeared, it does not recede, or, at least, the remissions are momentary. The skin assumes a deep yellow or greenish tint. As a rule pain is absent, but I have seen a case of primary cancer of the biliary passages where the pain led me to believe in obstruction by gall-stone; my mistake was revealed at the post-mortem examination. The liver is practically normal in size, and secondary cancer never attacks it. Anorexia is constant, digestive troubles are frequent, and the patient grows rapidly thin, and dies in eight to ten months. The pathogenic diagnosis of the obliterating cause is for several months a matter of conjecture.

**Permanent Obliteration of the Common Duct by Cancer of the Head of the Pancreas.**—Cancer of the head of the pancreas is one of the most frequent causes of permanent obliteration of the common duct. The following cases will give an idea of its evolution:

I was called to an elderly lady suffering from jaundice, which had commenced a few days previously, but was not very marked, though the urine contained bile, and the fæces were quite colourless. The diagnosis of obliteration of the common duct by a calculus was, in my opinion, very improbable, because the patient had never had any sign of hepatic colic; the jaundice had come on without pain, and the region of the gall-bladder was absolutely painless. Although the patient had had no vomiting, no diarrhoea, and no colic, I nevertheless thought of simple catarrhal jaundice, and prescribed mild purgatives, large enemata of cold water, and ingestion of oil; the patient was put on milk diet. Three weeks passed without any improvement in the symptoms, and intolerable itching also appeared. The patient did not consider herself ill, and followed her usual habits. Her appetite, however, grew less. The obstinacy of the jaundice made me anxious, but I tried to fall back on the hypothesis of prolonged catarrhal jaundice. Examination of the abdomen and of the liver gave no information. Other remedies were tried with no better result, and after six weeks the patient, who had grown thin and had a distaste for food, decided to leave for one of the Mediterranean towns.

During the five months of her residence in the South the symptoms were unchanged. She wrote that her colour had now become a brownish yellow, the loss of flesh was making rapid progress, and several times a week, without apparent cause, she had acute attacks of fever, with a temperature of 102° to 104° F. These attacks commenced with a rigor, and ran through three stages—cold, hot, and sweating, just as in malaria. Quinine had no effect. Between these irregular attacks of fever the temperature was normal.

When the patient returned to Paris I was immediately sent for. In six months she had lost nearly 50 pounds in weight, and was literally reduced to a skeleton. I examined her. The anorexia was complete: she could scarcely drink a cup of milk. The fæces were clay-coloured, but at times they showed a slight tint, proving that a small quantity of bile reached the intestine. The extreme thinness of the patient made the exploration of the abdomen very easy, but nothing abnormal could be felt. The liver was of normal size, and the gall-bladder could not be found. It was certainly a case of cancer in the bile-ducts, Vater's ampulla, or the head of the pancreas, unless, by some happy exception, a large gall-stone had become impacted in the common duct without causing pain. In any case an exploratory laparotomy was absolutely indicated, and Routier, who was called in, expressed the same opinion.

The operation was, therefore, performed. No calculus was found. The gall-bladder, which was deep-seated and only slightly apparent at first sight, though much distended, contained about 10 ounces of bile, removed by aspiration. There was no cholecystitis. The liver was almost normal in size. We were convinced, after careful examination, that the obstruction of the common duct was due to cancer of the head of the pancreas, and the operation was concluded by anastomosing the gall-bladder with a coil of small intestine. A few days after the operation the jaundice diminished, the fæces became coloured, the urine was clear and limpid, and the patient was able to take light nourishment. The improvement, however, was not of long duration, and three months later the patient succumbed to cachexia.

This case gives an idea of obliteration of the common duct by cancer of the head of the pancreas, and corresponds to the classical description. In their description of cancer of the head of the pancreas, Bard and Picq thus sum up the question: "The characteristic symptoms of primary cancer of the head of the pancreas are progressive jaundice, enormous dilatation of the gall-bladder, loss of flesh, and rapid cachexia, without notable enlargement of the liver. The last of these four signs, though purely negative in character, is just as important as the other three; their union constitutes the special characteristic of the disease." These conclusions hold good in most cases, but the exceptions, which are far from being rare, must be taken into account. These exceptions show us that the jaundice may not be progressive, the gall-bladder may not be dilated, and the liver may be considerably enlarged.

I said previously that the jaundice may not always be progressive, and in proof thereof I quote the following case, published by Legrand:

A man who was admitted for chronic jaundice had cancer of the head of the pancreas, proved at the post-mortem examination. On admission we found very deep icterus, bile in the urine, and colourless fæces. Furthermore, the gall-bladder was enormous and easily felt. One day a flow of bile occurred; the fæces were of a deep green colour, and the tumour formed by the gall-bladder diminished. Dutil has published a similar



case : A patient was admitted to hospital with jaundice, bilious urine, and colourless fæces. On several occasions, however, it was noticed that bile was present in the stools, and the post-mortem examination revealed cancer of the head of the pancreas.

The following case shows still better how the symptoms of cancer of the head of the pancreas may differ from the classical type :

A man, thirty-six years of age, was taken ill in September, 1891, with digestive troubles and pain, that was especially severe after meals. The pain was most severe in the epigastrium and left hypochondrium, but did not exactly show the characteristics of hepatic colic. It persisted for several months. In February, 1892, progressive jaundice supervened, with increasing loss of flesh, great weakness, and intolerable itching. These symptoms made progress in March, April, and May. At this date Dr. Ferras de Macedo sent the patient to Paris. The condition was as follows : Very intense jaundice, continual itching, colourless foul-smelling fæces, and urine containing bile and albumin. The liver was enormous, measuring 9 inches in the median line and 11 inches in the nipple line ; the surface was regular, of normal resistance, without fluctuation, and the organ formed a marked bulging in the epigastrium. The edge was soft, and palpation did not reveal the gall-bladder. The spleen was normal, and the principal viscera were healthy. The appetite was good, but digestion was painful, and accompanied by a feeling of heaviness and by pain radiating into the left hypochondrium.

It was clearly a case of jaundice from obliteration of the common duct. Was the obliteration due to cancer or to a gall-stone ? The pathogenic diagnosis was most difficult. Bouchard and Terrier favoured the diagnosis of cancer of the pancreas, an opinion shared by Ferras de Macedo. Hanot and myself favoured the diagnosis of a calculus. Reclus, Périer, and Oettinger concluded, and this was also my opinion, that it was necessary to perform exploratory laparotomy, followed by choledochotomy or biliary enterostomy. The patient, wishing for further advice, went to Germany, and finally returned to Paris. Reclus performed the operation. The liver was enormous, and of a very deep brown colour. Under its soft edge the gall-bladder was found ; in size it was larger than two fists. The gall-bladder and the cystic duct were free, but the common duct, near the head of the pancreas, was obliterated by a hard body, as large as a walnut, so firmly and deeply set that its extraction seemed impossible. The gall-bladder was united to an intestinal coil (biliary enterostomy).

The operation was followed by marked improvement, and the patient got up on the fourteenth day. The appetite returned, and on the seventh day the liver measured but 7 inches in the nipple line. Three months later the patient was in perfect health, but a short time after, as the pain and other symptoms returned, he came back to Paris. He underwent another operation, performed by Reclus, who discovered that the hard body found at the first operation was a cancer of the head of the pancreas. The fact was verified at the post-mortem examination. This case proves, contrary to the conclusions arrived at by certain writers, that the liver may become much enlarged in obstruction of the common duct by cancer of the head of the pancreas. It also shows that the icteric syndrome due to the obstruction of the common duct by cancer of the head of the pancreas may be accompanied by pain that is not always easily distinguished from that due to obliteration of the duct by a gall-stone.

**Permanent Obliteration of the Common Duct by a Calculus.**—We have still to make the pathogenic diagnosis between obliteration of the duct by cancer of the head of the pancreas and by gall-stones. The diagnosis is singularly simplified if the obliteration has been preceded by classical hepatic colic. In such a case the cause of the obliteration is obvious. In other cases,

however, the pains due to impaction of a stone are very difficult to distinguish from those due to cancer of the pancreas. Moreover, permanent obliteration by a stone may cause considerable loss of flesh, thus resembling the cachexia of cancer, and rendering the diagnosis doubtful. The following case will illustrate this point :

On August 25, 1897, a woman, sixty-three years of age, was admitted for chronic jaundice, involving the skin and the mucous membranes. The urine, which had an oily look, was of a deep mahogany colour, and contained much bile pigment and traces of albumin. On the other hand, the fæces were clay-coloured. The contrast between the deep jaundice of the skin and of the urine and the whiteness of the fæces clearly pointed to obstructive jaundice ; the bile, being arrested in its course, could no longer flow into the intestine.

The patient told us that she had been yellow for a year. The jaundice had been preceded by pain, that came on about three hours after breakfast. The pain started in the epigastric region, and radiated over the belly, into the back, and between the shoulder-blades ; it was sometimes followed by fainting-fits. The pain was frequently accompanied by vomiting of bile. After the jaundice had appeared the fæces became whitish. The patient still continued to work in spite of the pain. She had been unwell for nearly a year, and although she no longer had the attacks of pain with which the disease began, the pain was, nevertheless, almost continuous, with exacerbations and vomiting of bile. The jaundice increased or diminished at intervals, but never disappeared. On several occasions she had severe attacks of fever, preceded by rigors, and followed by profuse sweating. Pruritus was constant. The appetite had failed, and the stomach had become so intolerant that she could only take milk in small quantities. Her strength had gradually diminished, and she had lost over 30 pounds in weight, and therefore when I saw this feeble old woman with cachexia, chronic jaundice, and œdema of the legs, I could not at first set aside the idea of cancer.

Examination of the patient gave the following results : The belly was slightly distended, but there was no trace of collateral circulation ; ascites and tumour were absent. The liver reached three fingers' breadth below the costal margin, and was very painful on palpation. The gall-bladder, however, was not enlarged, and, even supposing that it had been, it was deeply hidden, and could not be felt. The spleen, heart, and lungs were normal. There was no tricuspid murmur, and the pulse-rate was 75. The urine contained some albumin, but not a trace of sugar—an important point.

Such was the condition on admission. It was now a question of making a diagnosis, and it was necessary to find out the lesion preventing the passage of bile into the intestine. Was it a calculus, or was it cancer ? Although she was old and very thin—both points in favour of cancer—I was compelled to consider the nature of the pains at the onset of the disease in making a diagnosis. These pains appeared to me to be in favour of hepatic colic, rather than of cancer of the pancreas. In consequence, I decided in favour of obliteration of the common duct by a calculus.

The question of treatment remained. As I felt that the obstruction was due to a calculus, and as it had already lasted a year, I thought of surgical intervention. Nevertheless, as I wished to observe the case closely, I prescribed milk, with bicarbonate of soda, capsules of ether and turpentine (Durnade's remedy), and saline purgatives. I tried to relieve the itching by baths of starch and gelatine, alcohol rubs, and inunction of a bromide ointment. From this date the disease ran the following course : The icteric syndrome (icterus, bilious urine, and colourless fæces) was not always regular. At times a small quantity of bile made its way into the intestine, the fæces became slightly tinged, the colour of the urine and of the skin was not quite so deep, and the itching was less troublesome. In view of these slight remissions, I asked myself whether the obliteration might not yield spontaneously. The fæces were strained through a sieve



and examined for gall-stones, without success. Evidently the bile under pressure was able at times to force the barrier, which was not quite insurmountable, but the obliterating body was not dislodged.

The liver extended below the ribs. The patient had no appetite, and took only a few spoonfuls of soup or milk. At times she complained of sharp epigastric and hepatic pains, and vomited fluid containing a small quantity of bile, a further proof that the bile was able to pass through the obstructed ducts. Suddenly **severe attacks of fever** appeared, with violent rigors, rise of temperature to 104° F., and profuse sweating. Apart from these severe attacks of fever, which for some months past had appeared at irregular intervals once or twice a week, the temperature was practically normal, as the following chart shows.

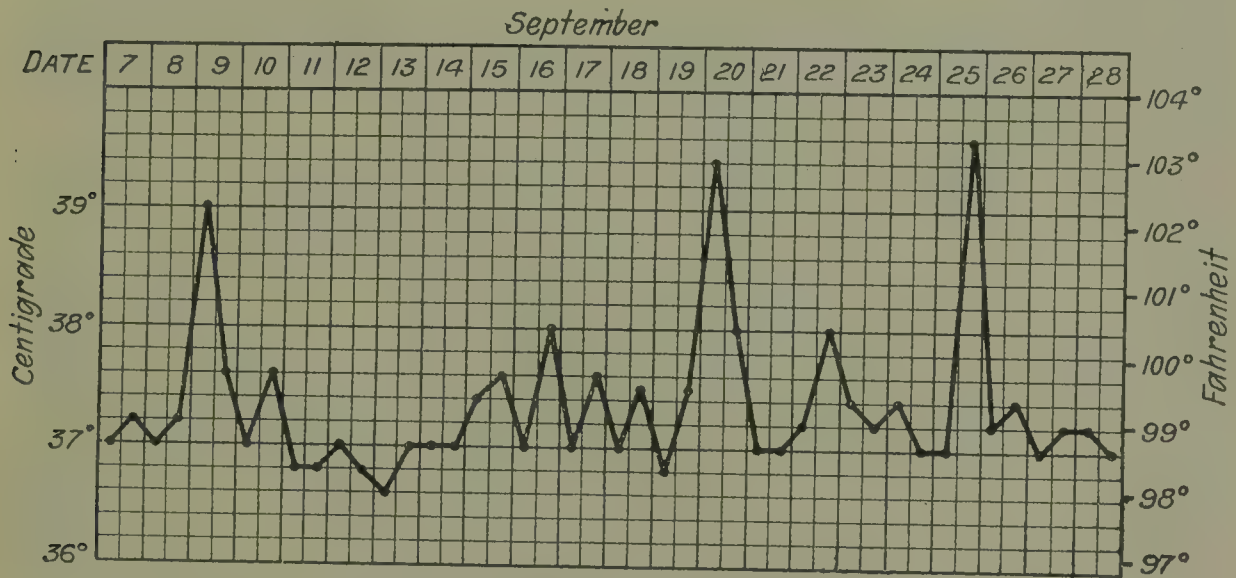


FIG. 53.—TEMPERATURE CHART IN OBSTRUCTION OF THE COMMON BILE-DUCT.

I shall return later to these severe attacks of fever, and shall try to indicate their cause. When fever supervenes in a patient suffering from obliteration of the bile-ducts, we think of cholecystitis or hepatic abscesses, but yet I eliminated the idea of infective fever. The patient continued to lose flesh, the pain did not diminish, the itching was intolerable, the nausea was permanent, and it was time to intervene. The patient readily consented to an operation.

Duplay diagnosed obliteration of the common duct by a gall-stone. The belly was opened, and the anterior edge of the liver was pushed upwards, so as to expose its lower surface. The gall-bladder was not distended; indeed, it could not be felt in the cystic fossa. After the liver had been tilted backwards, Duplay succeeded in feeling near the hilum a deep-seated tumour as large as a hazel-nut, of stony hardness, and covered by a fairly thick soft wall. It was impossible to state precisely whether the tumour involved the common or the cystic duct, but it appeared rather to be at the junction of the cystic and hepatic ducts with the common duct. Digital exploration of the common duct along the free edge of the gastro-hepatic omentum and in its retroduodenal portion showed that the common duct contained no calculi in this portion of its course. The soft parts covering the hard tumour were then incised, and the curette brought out some small fragments, and then a large calculus weighing  $\frac{1}{2}$  ounce.

The diagnosis was therefore verified. The result of the operation was excellent. The icteric syndrome disappeared, the fæces became coloured, the urine gradually regained its normal tint, in a fortnight the jaundice had entirely disappeared, and in three weeks the wound had healed. The itching ceased as soon as the bile flowed into

the intestine ; there was no recurrence of pain or vomiting. The fever disappeared as soon as the bile took its normal course, proving that it was not due to hepatic infection. In less than three months the patient regained her normal weight and appetite. A year later she was in perfect health.

**Analysis of the Symptoms.**—The patient had cachectic œdema and rapid loss of weight. Such wasting in permanent obliteration of the common duct might lead to error and cause belief in cancer, and yet we have just seen that marked loss of flesh is of small importance in the diagnosis of the cause of the obliteration of the common duct ; this fact I have noticed on several occasions.

There is another point deserving of notice. Even before admission to hospital, the patient had suffered from **attacks of fever** that commenced with a violent rigor, and reached 104° F.

In order to discuss the cause and the value of febrile attacks in patients with biliary retention, let us consider the question as a whole.

The migration of gall-stones during hepatic colic is sometimes accompanied by attacks of fever (Charcot's hepatalgic fever). I have discussed this point under the Satellite Symptoms of Hepatic Colic. In this case, however, the attacks of fever could not be attributed to migration of calculi, because an enormous calculus was impacted in the common duct, and prevented any migration. On the other hand, the attacks could not be attributed to infection of the bile-ducts (angiocholitis, hepatitis, cholecystitis), because the fever ceased when the flow of bile became normal. How, then, are we to explain the thirty or forty attacks of fever which the patient had within a few months ? I have no intention of formulating a theory, but, seeing that the attacks lasted as long as the biliary retention, and that they ceased as soon as the biliary retention ceased, it is quite logical to attribute them to the retention and absorption of bile. Is the bile altered in consequence of long retention ? I do not know, but the great clinical fact is that in patients suffering from permanent obliteration of the common duct severe and repeated attacks of fever may occur without appreciable infection of the biliary passages.

Similar attacks of fever were present in a case that I saw with Charrier on July 7, 1895. The patient was suffering from biliary retention due to permanent obliteration of the common duct by a stone. The obstruction had already lasted seven months. The gall-bladder could not be felt, but the liver was enlarged and painful. We recommended an operation, and the patient was taken to the Necker Hospital, where Routier operated. The gall-bladder was of small size, and was not infected ; it contained seven calculi, whilst two others were impacted in the common duct, blocking it completely. No pus was found. The operation was a complete success.

In this case the attacks of fever could not be attributed to an infection of the biliary passages, as the operator did not find any trace thereof, and as the attacks yielded as soon as the operation had re-established the normal flow of the bile. The retention and absorption of bile cause these attacks of fever. I have also noticed similar attacks of fever in obliteration of the common duct by cancer of the head of the pancreas. I



have already mentioned a case of jaundice of eight months' duration following on cancer of the head of the pancreas. The patient had also had attacks of fever, with a rise of temperature to 104° F.; the attacks lasted for several months. In this case also an operation revealed no trace of suppuration; the gall-bladder was not infected, and the attacks of fever ceased as soon as the obstacle to the flow of bile had been removed.

The object of this digression is to emphasize certain varieties of fever in cases of jaundice. First variety: so-called heptalgic fever (satellite fever), associated with the migration of calculi and hepatic colic. Second variety: so-called bilio-septic fever, the result of infection of the biliary passages (angiocholitis, cholecystitis, abscesses). Third variety: fever resulting from the permanent retention of bile. In these three varieties the fever occurs in attacks, with this difference however: that the attacks are distinct and separated by longer or shorter periods of apyrexia in the first and third varieties, whereas they are part of a more continuous febrile condition in infection or suppuration of the biliary passages.

**Diagnosis.**—Let us now consider the diagnosis of the cause of the obliteration. In general terms cancerous obstruction of the common duct causes dilatation of the gall-bladder, while obstruction by a stone causes atrophy. This rule holds in many cases, but as exceptions we find the cases of Cruveilhier, Sabourin, Billroth, Jalaguier, Hanot, Griffon, etc., in which the gall-bladder showed enormous dilatation, consequent on obliteration of the terminal portion of the common duct by a calculus, cancer being absent. I agree, therefore, with Reclus that "dilatation of the gall-bladder is in favour of cancer, because atrophy of the bladder is much more frequent than enlargement in gall-stones." This sign is, however, not absolute, and cannot *per se* decide the question without reckoning on the fact that it is not easy to make out dilatation of the gall-bladder, since there are cases in which, though it is dilated, it is so deeply seated that it cannot be examined.

Enlargement of the liver, formerly considered to be special to calculous obliteration, may exist in the case of cancerous obliteration, as is shown by the enormous size of the liver in the patient I saw with Reclus.

Rapid loss of flesh and cachexia are in favour of cancer. And yet calculous obliteration of the common duct may be followed by considerable loss of flesh and cachexia, as was seen in my patient, who had lost over 30 pounds in a few months.

The comparative analysis of the fæces will be described under Pancreatitis.

Glycosuria has been given as a sign of cancer of the pancreas, but this sign is only of slight value. Glycosuria is found in about a third of the cases, and even then, according to Bard and Picq, it is a secondary symptom, due not to cancer, but to concomitant sclerosis of the pancreas.

**Summary :** The pain furnishes the most important support in the diagnosis of the cause of the obliteration, and even then it is necessary to define clearly the character of the pain. Cancer of Vater's ampulla may cause pain and simulate hepatic colic, as in Rendu's case. Cancer of the head of the pancreas is even more painful; this fact led to an erroneous belief in calculous obliteration. Pain is a valuable sign only when it appears in the form of **classical hepatic colic**, which at more or less distant periods has preceded or accompanied the definite obliteration of the common duct.

I have intentionally omitted in this section obliteration of the common duct consecutive to pancreatitis. This question is so important that I shall deal with it in a special section under Diseases of the Pancreas.

**Treatment.**—Operative interference becomes necessary at some stage of obliteration of the common duct, even in the absence of an exact diagnosis as to the cause. Even in cases of cancer an operation is proper because it re-establishes the flow of bile, relieving the itching and giving the patient some months of hope.

In obliteration by a gall-stone an operation leads to recovery. As my experience has increased, I feel sure that surgical treatment is to be preferred in obliteration of the bile-ducts by a gall-stone, because the obliteration is so often accompanied by cholecystitis. The operation re-establishes the flow of bile, and removes the gall-bladder, which may give rise to complications. It has the further advantage of preventing the onset of severe pancreatitis. I know that the objection will be raised that obliteration by a calculus of several months' duration may yield to treatment at Vittel, Vichy, Contrexéville, Carlsbad, etc.; it is, however, proper to add that many of these cases are liable to relapses, to hepatic colic, cholecystitis, pancreatitis, and other well-known complications.

We must be ready to decide, for it is better to intervene early rather than late. In the case above quoted a late operation did not prevent recovery; the passage of small amounts of bile, however, saved the situation. I feel, however, that more prompt decision is needed. In permanent obliteration of the common duct by a calculus, the most favourable time for operation is within the first two months (*vide* Section VII., Diseases of the Pancreas).

Cases recently published by Lejars and Morestin are in favour of this view, and a case under my care at the Hôtel-Dieu sums up the situation :

A woman was admitted into my wards on May 7, 1907. She had suffered from attacks of hepatic colic for four years, and the present attack was of seven weeks' duration, the icteric syndrome being complete. I made a diagnosis of permanent obliteration of the common duct by a calculus. As medical treatment failed, I asked Terrier and Gosset to operate. The atrophied gall-bladder was excised, and the common duct was catheterized, revealing a rounded stone, as large as a small nut, near Vater's ampulla. The hepatic duct was drained. Complete recovery followed.



## XXII. ANGIOCHOLITIS—CHOLECYSTITIS.

**Pathogenesis.**—Angiocholitis and cholecystitis are always due to microbic toxi-infections. The infectious process is sometimes favoured by stagnation of bile due to compression or obstruction of the bile-ducts; at other times the invasion of the bile-ducts by microbes takes place, without previous obstruction. Biliary calculi realize the type of the obstructive infection; typhoid infection realizes the type of infection without previous obstruction. I would therefore refer the reader to the sections on Biliary Lithiasis and on Typhoid Fever.

In the normal state the bile, the gall-bladder, and the bile-ducts are aseptic, the only exception being the lower portion of the common duct on account of its proximity to the duodenum, in which coli bacilli, streptococci, and staphylococci are normally present. In pathological conditions, however, these microbes enter the bile-ducts. Many other agents, such as the pneumococcus, the *Bacillus typhosus*, the cholera bacillus, diplococci, and liquefying saprogenic bacilli may also infect the biliary passages. In some cases the organisms may even live in a latent state in the bile, which has not the bactericidal properties formerly attributed to it.

The micro-organisms come most frequently from the infected intestine. Why, however, do the biliary passages allow themselves to be invaded? What cause governs the emigration of microbes from an infected intestine to the aseptic biliary passages? The virulence of the intestinal microbes may be a sufficient cause for the emigration, but most frequently this migration towards the biliary passages is favoured by pathological conditions of these passages from obstruction and injury due to calculi, by retention of the bile in the intra- and extrahepatic ducts, or by some previous change in the hepatic cell (alcoholism, gout, or syphilis). In other words, the microbes rush in when the breach is practicable. When the organ is in a condition of morbid receptivity, the microbes and their toxins accomplish their work.

When the biliary infection is established, whether the migration of the organisms takes place with or without the previous assistance of traumatism, of obstruction, or of biliary retention, how does the infection reveal itself? The answer involves the study of angiocholitis and cholecystitis.

**Angiocholitis.**—Angiocholitis, or inflammation of the bile-ducts, affects both the large and small ducts (extra- or intrahepatic), just as bronchitis affects the large and small bronchi.

Angiocholitis of the large ducts, and especially of the common duct, is usually due to gall-stones, but inflammation of the common duct (choledochitis) is not always due to a calculus. Longuet mentions three cases, reported by Terrier, Heltzerich, and Ahlfeld, in which the common duct con-

tained no stone, but yet it was enormously distended, and contained greenish viscid or suppurating fluid.

Angiocholitis of the small ducts is so closely allied to the calculous form that I would refer the reader to the section on Gall-Stones. It may give rise to the areolar abscesses of the liver described by Chauffard, and also to enormous intrahepatic enlargements of the ducts, simulating large abscesses of the liver. Longuet quotes an absolutely characteristic case. Intrahepatic angiocholitis may be accompanied by jaundice, without decoloration of the fæces; swelling and pain in the liver are frequent but inconstant symptoms. Fever is seldom absent; it has been called intermittent hepatic fever (Charcot), or intermittent bilio-septic fever (Chauffard). It exactly resembles malarial fever, with this difference, that the attack is generally vesperal and irregular in type.

In some cases the infection affects the liver and the blood, spreading from the ducts to the portal and hepatic veins, and producing infective endocarditis of the right side of the heart, suppurating infarcts in the lungs and pleura, infective endocarditis of the left side of the heart, suppurative meningitis, etc.

**Cholecystitis.**—I would ask the reader to refer to the section on Calculous Cholecystitis, where certain sides of this question have been treated in detail.

The pathogenesis of the infection of the bile-ducts is applicable to infection of the gall-bladder (cholecystitis). As regards its evolution, however, cholecystitis may be divided into certain varieties—cholecystitis, paracholecystitis, and pericholecystitis.

1. Cholecystitis, whether it is or is not due to gall-stones, follows biliary infection. In both forms there may be obstruction of the excretory canals, either by calculi or “because the inflamed mucosa has formed a plug in the fine cysto-choledochic ducts.” The infected gall-bladder may or may not contain fluid; the fluid may be serous (hydrocholecystitis), sero-purulent (pychocholecystitis), or hæmorrhagic (hæmocholecystitis).

Cholecystitis with effusion causes more or less enlargement, so that the gall-bladder may resemble a cyst with thin and distended walls, or a tumour with thick, hypertrophied walls. In non-calculous cholecystitis the gall-bladder is usually distended; in calculous cholecystitis it is always contracted and shrunken (Terrier).

2. In pericholecystitis the inflammation is localized around the gall-bladder, and does not extend to remote organs. It may be calculous or non-calculous, suppurative or fibrinous. Pericholecystitis is most frequently suppurative in the case of calculous cholecystitis; it is often fibrous and rich in adhesions, whether it is or is not due to gall-stones.

3. Paracholecystitis must be reserved for suppuration more or less



distant from the infected gall-bladder. Longuet divides the abscesses into four types: "Some, anterior and inferior, invade the abdominal wall (flank and right hypochondrium), and give rise to fistulæ. Others spread upwards over the upper surface of the liver, between the diaphragm and the false ribs on the one side and the liver on the other; they form the antero-superior type. If, however, they continue their course as far as the posterior wall of the abdomen, they become postero-superior. Finally, the postero-inferior abscesses leave the roof of the abdominal cavity, in order to reach the posterior wall of the abdomen and the renal and lumbar regions." These *remoté* abscesses are more commonly seen in non-calculous than in calculous cholecystitis; they are comparable with the remote abscesses of appendicitis.

The **symptoms** of cholecystitis have been described elsewhere. Non-calculous cholecystitis, as often as calculous cholecystitis, is complicated by adhesive pericholecystitis. These adhesions may cause very acute pain—a common symptom when adhesions are formed in the abdominal cavity. Often, indeed, the other symptoms pass unnoticed, or are of minor importance, and a patient suffering from adhesive cholecystitis is seized, just as in hepatic colic, with acute pain and vomiting. The attacks of pain recur; the diagnosis of hepatic colic or of calculous cholecystitis is made, and an operation is decided on. No calculus is met with, but, on the contrary, adhesions due to pericholecystitis are found. The adhesions are removed, and the patient recovers from the so-called hepatic colic. He has really been suffering from hepatic pseudo-colic. Fraenkel and Terrier have reported cases of this kind.

Kummel mentions a woman who was subject to hepatic colic with icterus, and passed gall-stones in the stools. As the pains grew worse, an operation was performed. The gall-bladder contained no calculi, but had become adherent to the omentum and to the cystic and common ducts. The adhesions were the cause of the pains.

I have seen two similar cases. The adhesions were removed, and the pains, which had been agonizing, ceased completely.

### XXIII. ASSOCIATION OF APPENDICITIS AND CHOLECYSTITIS.

My communication to the *Académie de Médecine*,\* on the **association of appendicitis with cholecystitis**, is, I believe, the first work published in France on this subject. I have since discussed the question in a clinical lecture.† This association, which is fairly frequent, is not the result of a fortuitous coexistence; the two infections are associated, the one causing the other. Appendicitis and cholecystitis enter into combination, and from this combination, which may be sudden and acute or slow and progressive in its onset, a complex condition results. The present description is founded

\* *Académie de Médecine*, séance du 17 Juin, 1903.

† Dieulafoy, *Clinique Médicale de l'Hôtel-Dieu*, 1906, 7<sup>me</sup> leçon.

on about thirty cases ; it is of importance, because it has as its immediate corollary, to state, as precisely as possible, the indications for surgical intervention.

**Clinical Cases.**—On September 25, 1902, Achard asked me to see an old lady, seventy-eight years of age, who had been taken ill two days before with abdominal pain, nausea, and slight fever. On examining the patient, Achard had discovered a painful spot over the gall-bladder. There was no muscular resistance, and the belly was not distended. Although there was no history of gall-stones or of hepatic colic, the location of the pain led him to suspect cholecystitis. The pain grew worse, and the fever was more severe on the following day.

When we examined the patient two days later, the clinical picture was complete. The belly was distended ; the situation of the pain had changed, acute pain, with muscular resistance, being present over the appendix. We, then, diagnosed acute appendicitis, and as the condition of the patient grew rapidly worse, we recommended immediate operation. Segond was then called in. He also diagnosed acute appendicitis, and operated at 11 p.m.

Before anæsthesia was induced, the pain and the muscular resistance had not allowed us to ascertain certain details. As soon as the abdominal wall was relaxed, Segond found induration over the appendix, and a tumour in the region of the gall-bladder. He therefore made a rather high incision, and found two lesions, appendicitis and cholecystitis. Exudate matted together the gall-bladder, the colon, the cæcum, and the appendix. The gall-bladder was pushed down, and formed a violet and distended tumour ; it contained a turbid and bilious fluid, as well as several calculi. Cholecystostomy was performed. The appendix of the ascending type was adherent and swollen. It was then resected. The operation was followed by complete relief. During the next few days the biliary fistula gave issue to bile and to two calculi. Two months later the patient left for Nice in excellent health. She no longer vomited, and had only a small biliary fistula, which was cicatrizing.

This case shows the benefit of prompt operation, because immediate relief followed. If the operation had been postponed, the double infection in the appendix and the gall-bladder would very probably have proved fatal.

In December, 1902, I was called with Segond to see a man of about thirty years of age. Two days previously the patient had felt indisposed. At first the symptoms of gastric disturbance—loss of appetite, malaise, and slight fever—had not been characteristic ; on the following day the abdominal symptoms were more marked, the pain was worse, and a painful tumour could be felt in the region of the gall-bladder. The patient's physician had diagnosed cholecystitis. The temperature had been over 104° F. on two occasions.

Two days later, when I examined the patient, the clinical picture had changed. The fever persisted, and the most important feature was not the pain in the region of the gall-bladder, but a sharp and characteristic pain with muscular resistance over the appendix. The pain was most marked above McBurney's point, as is common in cases of ascending appendicitis. Careful examination showed that the pain on pressure decreased in proportion as we ascended towards the liver. No tumour was noticed in the region of the gall-bladder. These signs left no doubt as to the existence of appendicitis. Segond formed the same opinion. The urine contained albumin.

The symptoms of appendicitis, therefore, were more marked than those of cholecystitis. The rapid course of the disease, the temperature rising above 104° F., and the presence of albumin gave us much alarm.

In such cases a decision is imperative. Segond and myself were in favour of immediate operation. The patient's physician did not agree with us. As we shall see, the operation was the more indicated in that it revealed peritonitis, appendicitis, and cholecystitis.

The operation was performed at 10 p.m., Segond first attacking the appendix. On opening the peritoneum he found some turbid serous fluid, indicative of appendi-



citis ; a larger quantity of turbid fluid had spread into the pelvis. The appendix, which was inflamed, and which reached up behind the cæcum, was excised. Subsequent examination by Nattan-Larrier revealed ulcerative appendicitis. Below a plug of fæcoid matter were two large ulcerations, the one elongated, the other oval. At the site of these ulcerations the tissues were so destroyed that they were transparent, the wall of the appendix being reduced to a thin sheet. At other points the mucosa was swollen ; elsewhere there was a hæmorrhagic stippling.

Second next turned his attention to the gall-bladder. The incision in the abdominal wall was prolonged upwards, and by means of digital exploration he reached the gall-bladder, which was deeply seated. He found that it was distended, with red and thick walls. He performed cholecystostomy. The incision gave exit to a yellowish liquid, then some creamy pus, and finally to some tinted pus ; no gall-stones were present. The neck of the gall-bladder appeared to be obliterated.

The results of the operation were remarkable. In spite of the triple infection, two hours after operation the temperature had fallen to normal. Speedy recovery followed.

In the next case (Grant) appendicitis and coexisting cholecystitis ended in perforation and death. A man, fifty-three years of age, was said to have previously had hepatic colic. Violent pains, localized to the region of the gall-bladder, suddenly appeared one night. Grant diagnosed cholecystitis. Two hours later a fresh attack of pain with vomiting appeared. Rupture of the gall-bladder was diagnosed. Next morning intense pain over the appendix also appeared. Freeman diagnosed appendicitis and cholecystitis. The operation revealed appendicitis and cholecystitis, which had both ended in perforation. The patient died four days later from general peritonitis.

Adolf Becker has recently collected some thirty cases of coexistent appendicitis and cholecystitis. These cases fall into two groups—in the first group the appendicitis appears to have preceded the cholecystitis, and in the second and larger group the cholecystitis preceded the appendicitis.

**First Group.**—*Kehr's Case.*—A woman, forty-six years of age, who seemed to have had previous attacks of appendicitis, was taken ill with acute hepatic colic. Laparotomy was performed. The appendix was surrounded by adhesions, and fixed to the back of the cæcum and colon. The gall-bladder, covered with membranes, contained turbid liquid and eight gall-stones.

*Kehr's Case.*—In the case of a man, thirty-seven years of age, with appendicitis cholecystitis was also suspected. The operation revealed a thickened appendix, reaching up behind and adherent to the cæcum and colon. The gall-bladder was completely buried in adhesions. The gall-bladder and the appendix were removed.

**Second Group.**—*Müller's Case.*—A woman, twenty-one years of age, had three attacks of pain in the right hypochondrium ; the pain radiated to the shoulders, the back, and the chest ; jaundice was present, and gall-stones were not present in the stools. The diagnosis was cholelithiasis. Acute pains in the right ileo-cæcal region supervened several days later, with marked tenderness over the appendix. Müller diagnosed cholelithiasis and appendicitis. The operation revealed cholecystitis, with an adherent and contracted gall-bladder containing calculi, and appendicitis, with stenosis of the canal at the mouth of the cæcum, thickening of the wall, and purulent exudate.

*Müller's Case.*—A man, forty-six years of age, had for several years past suffered from hepatic colic with jaundice, but no calculi had ever been found in the stools. Later a very painful tumour was noticed in the cæcal region. Müller diagnosed chronic appendicitis, for which he operated. He found the appendix surrounded by adhesions, and dilated in the shape of an ampulla in its lower third. In spite of resection of the appendix the patient still continued to complain of violent pain in the right hypochondrium. A very painful tumour was found in the region corresponding to the gall-bladder. A second operation was performed, and the gall-bladder was found to be surrounded by adhesions and to contain several calculi.

The acute or chronic lesions in these cases of appendicitis associated with cholecystitis are of every kind : muco-purulent or hæmorrhagic fluid, ulceration of the walls of the appendix, stenosis, dilatation, perforation, gangrene, adhesions, pericæcal suppuration, peritonitis, etc. The cholecystitis may or may not be due to gall-stones, and the gall-bladder in different cases is distended, thickened, retracted, and adherent, and contains fluid variable in quantity and in quality.

**Discussion.**—We must now consider the cause of this double infection of the appendix and the gall-bladder. Is it brought about simultaneously by one cause, or is there rather a superposition of the two infections, the one preparing the other ? And, in this case, which is the earlier, the appendicitis or the cholecystitis ?

We might suppose that the two infections are due to a calculous process, causing simultaneously cholecystitis and appendicitis. This hypothesis will not hold in the face of the fact that in this double infection, calculi are very seldom found in the appendix, and are sometimes absent in the gall-bladder. We must therefore look elsewhere for the pathogenesis of the double infection.

Is cholecystitis the result of appendicitis ? We know how readily ascending infections of appendical origin occur. In Section XIX. we saw that the infection starts from the appendix, and is carried to the liver by the venous network that ends in the portal vein and in the liver. This infection through the veins has, however, nothing to do with the infection of the gall-bladder. It is the liver that is attacked, and not the gall-bladder, and the proof is that in the numerous cases of appendicular liver the gall-bladder remains healthy.

We may also ask whether the infection, starting from the appendix, may not reach the gall-bladder by the peritoneal route, through the adhesions which so often unite the appendix, the intestine, and the gall-bladder. This ascending infection causes subphrenic empyema and pleurisy in appendicitis. This question has been discussed under Appendicular Pleurisy. In such a case the infection, starting from the *primum movens* in the appendix, travels up along the cæcum and colon, reaches the hypochondrium, often causing subphrenic empyema ; passes through the diaphragm, which may or may not be perforated ; and invades the pleural cavity. The membranous and purulent tracks stand out as landmarks along the route, so that we can follow the infection from its modest origin in the appendix to its intrathoracic expansion, where the pleurisy is often purulent and putrid. On referring, however, to the numerous cases in which ascending infection has led to subphrenic empyema and pleurisy, it will be seen that cholecystitis was not present, the infection licking the walls of the gall-bladder, but not provoking cholecystitis.

If the gall-bladder escapes the broad track of the appendical infection,



which, as it ascends, spreads along the intestine, around and above the liver, and even into the thoracic cavity, is it reasonable to suppose that it can be infected by the small track which in the present case joins it with the appendix? In the majority of cases cholecystitis causes appendicitis, the infection taking place from above downwards. Furthermore, in twenty-five out of thirty cases the earliest symptoms are due to the biliary lesions, while the symptoms of appendicitis appear some days, weeks, or months later.

A patient, for example, has had undoubted signs of hepatic colic or of cholecystitis, especially pains in the right hypochondrium or in the region of the gall-bladder, the diagnosis of cholelithiasis, hepatic colic, or cholecystitis being made. At a given moment pain appears in the right iliac fossa, and is accompanied by other symptoms, indicating acute, subacute, or chronic appendicitis. As a rule, especially in the acute form, the symptoms of appendicitis, with or without peritonitis, become much more marked than those of cholecystitis. In other cases, especially in the subacute forms, the two foci (the one in the appendix and the other in the gall-bladder) remain sufficiently distinct for the diagnosis of cholecystitis and appendicitis to be made.

Many mistakes must have been made before attention was called to the association of appendicitis with calculous or non-calculous cholecystitis. A physician, having seen several attacks of typical hepatic colic in a patient, may have believed in a fresh attack of colic when the appendicitis broke out in a subintrant form. Another physician, having witnessed the onset of acute appendicitis, may not suspect the previous occurrence of cholecystitis if the symptoms of cholecystitis are effaced, or if the information given is insufficient.

We have, then, a new chapter to add to the history of cholecystitis and appendicitis; the possible association of this double infection must keep us constantly on the alert.

In the future, therefore, it will be necessary to pay attention to the coupling of these two infections. The infection of the gall-bladder is certainly serious, but the toxi-infection of the appendix is even more so. Failure to recognize appendicitis, and to fortify oneself on the ground of cholecystitis only, is a grave error, because it may falsify the therapeutic indications. The belief that we have to fight all the time against infection of the gall-bladder leads to delay, and during that time the appendicitis, which has not been recognized, may rapidly induce toxi-infectious complications, with or without perforation or gangrene, and the life of the patient is compromised by the double or triple infection, because we have not acted in time.

Early surgical intervention is especially indicated in such cases. An

operation for cholecystitis alone when appendicitis is also present means leaving the appendicular toxi-infection to do its worst. Not to operate on either lesion, and to advise delay, is to anticipate the most dire results.

#### XXIV. ABERRANT LOBE OF THE LIVER.

In certain individuals the liver has an extra lobe, which has been called the aberrant or erratic lobe, the hepatic tonguelet, and the floating lobe, as it has a certain mobility. The aberrant lobe arises on the under surface of the liver, close to the quadrate lobe. It is, as it were, hung from the liver, which is not depressed. The aberrant lobe is sometimes directly continuous with the liver substance; at other times it is attached to the liver by a rudimentary or a complete pedicle. The size, form, and length of the aberrant lobe vary; it may reach a length of 7 or 8 inches. The more marked the pedicle, the more mobile and depressed is the lobe; it simulated a floating kidney in the cases quoted by Pichevin and Faure.

**Symptoms.**—The floating lobe of the liver may remain unnoticed. It often causes sharp pains in the right hypochondrium, radiating to the lumbar region and to the shoulder. The pains simulate the crisis seen in calculous cholecystitis or in floating kidney. On examining the belly, the tumour is found. The liver is not depressed. The aberrant lobe descends more or less; it is smooth, fairly mobile, and sometimes painful on pressure. It follows the movements of the liver, unless it has a well-marked pedicle. The most frequent error in diagnosis consists in mistaking the aberrant lobe for a displaced kidney. Sometimes a mobile lobe is found with a mobile kidney, and, then, the diagnosis is complicated.

An interesting feature is that the aberrant lobe of the liver is fairly often associated with gall-stones and cholecystitis. We may even find the tumour formed by the aberrant lobe and the tumour due to cholecystitis side by side. The question was discussed by Riedel (Berlin, 1888), who found six cases of aberrant lobe in women suffering from cholecystitis, with or without cholelithiasis. The first authentic case, as far as I know, was reported by Trousseau. A woman had been suffering from hepatic colic with jaundice and fever. A tumour was felt at the right edge of the liver, and was taken for calculous cholecystitis. The post-mortem examination revealed calculous cholecystitis, but the tumour felt during life was an aberrant lobe of the liver masking the gall-bladder.

This description of the aberrant lobe of the liver shows the difficulties in diagnosis, especially when an aberrant lobe and cholecystitis exist in the same subject.

#### XXV. ICTERUS—JAUNDICE—CHOLÆMIA.

**Description.**—Under certain conditions, the bile pigment, elaborated by the liver, is reabsorbed; it passes into the blood and circulates in the plasma. The organs, tissues, and secretions are then more or less coloured by the bile.



**Icterus, or jaundice,** is the yellow coloration of the skin and of the mucosæ by the bile pigments. Jaundice appears first in the conjunctivæ, and then spreads over the face, the mucosa of the mouth, the trunk, and the limbs. The colour of the skin varies from the palest yellow (subicteric tint) to a deep yellow, which is generally the sign of icterus from retention. A golden-yellow colour is especially seen in acute jaundice; the greenish, olive, or almost black tints are principally seen in chronic jaundice. The jaundice is always general, and there is no such thing as partial jaundice, but it is more or less pronounced, according to the region. The colouring matter of the bile is deposited in the deep layers of the Malpighian network, and the jaundice does not completely disappear until after the desquamation of the coloured cells. The bile pigment is chiefly eliminated by the kidneys, whence the characteristic colour of the urine; but it is also eliminated by the sweat and sebaceous glands, which explains the yellow colour of the linen in contact with the patient's sweat. As the milk may contain bile pigment, a child should not be suckled by a wet-nurse suffering from chronic jaundice. In a pregnant woman suffering from chronic jaundice the yellow colour may be communicated to the fœtus.

When the skin is jaundiced, the urine is always affected. The coloration of the urine **precedes** by some hours that of the mucosæ and of the skin. The bile pigment is eliminated by the epithelium of the tubules; the kidneys are greenish, especially in the cortical region, and the microscope shows the pigment in the epithelium of the tubules. The urine is of high specific gravity, and is generally less copious than in the normal condition; its colour varies according to the quantity of the bile pigment, being orange-yellow, greenish, brownish, or almost black. The green tint is due to the conversion of bilirubin into biliverdin by oxidation. The urine stains the linen, and the more acid it is, the more marked is the green colour.

The analysis of the urine should be made in the following manner: Fuming nitric acid is poured into a wineglass (Gmelin's reaction), and the urine previously filtered is slowly added by means of a pipette. At the junction of the acid and the urine a greenish band will be seen, and above this green zone we see, from below upwards, blue, violet, red, and yellow rings. The green tint predominates, but after some time all these shades are blended in an orange colour.

**Effects of Jaundice.**—I shall only describe the symptoms due to the impregnation of the system by the bile.

1. *Gastro-intestinal Troubles.*—The fæces in obstructive jaundice are often clay-coloured, foetid, and rich in fatty matter—a proof that the bile under normal conditions promotes the absorption of fat. The clay colour of the fæces is due as much to the excess of fat as to the absence of bile. In obstructive jaundice we must carefully look for a return of colour in the fæces, because

this is an indication that the obstruction has a tendency to yield. The patient usually has a distaste for food ; the digestion is imperfect, the tongue is coated, and there is a bitter taste in the mouth. According to Murchison, the bitter taste is due to the passage of taurocholate of soda into the blood.

In certain patients the fæces are not colourless, and there may even be an excess of bile ; the fæces are greenish and liquid, and we then speak of polycholia or pleiochromia. The liver secretes an excess of bile, which is partly evacuated, partly absorbed.

2. *Poisoning*.—When the intestinal digestion is deprived of bile, less fatty matter is absorbed, and the patient suffers in consequence. He may lose flesh, or he may become poisoned by the products of fermentation absorbed through the intestine, and by the bile salts absorbed by the liver. This double cause of auto-intoxication is not marked in most cases, because the healthy liver cells destroy the poison, while the healthy cells in the kidney eliminate it. If, however, the cells of the liver and of the kidney are affected, symptoms of intoxication may result. In chronic jaundice the kidneys assume a greenish colour ; the epithelium of the canaliculi becomes pigmented, and sometimes undergoes fatty degeneration.

3. *Circulatory Troubles*.—The pulse is generally slow ; it may fall below 30 a minute. Lowering of the arterial tension is also noticed. The slowing of the heart-beats and of the pulse is due to the action of the bile salts in the blood. The salts, and especially the colouring matter of the bile, are cardiac poisons. I have described elsewhere the mitral and tricuspid murmurs which sometimes accompany acute and chronic jaundice. Jaundice often causes epistaxis, especially from the right nostril (Galen).

4. *Changes in the Blood*.—After numerous and contradictory experiments, the following conclusions have been arrived at : Acute jaundice (I am not referring to icterus gravis) causes no change in the blood, except that the serum is tinted by the colouring matter of the bile. Chronic jaundice causes diminution of the red corpuscles and increase of the fatty matter and cholesterin. The red corpuscle, in order to resist attack, modifies the normal conditions of its permeability.

5. *Cutaneous Troubles*.—Jaundice is often accompanied by pruritus. The itching is particularly severe in the feet and in the hands, and is sometimes intolerable, depriving the patient of sleep. Urticaria and lichen may also occur in jaundice.

In chronic jaundice we sometimes notice an eruption called **xanthelasma** (from ξανθος, yellow ; and μελασμα, a black spot), a name that is preferable to “yellow patches on the eyelids,” because in many cases the eruption is general. Xanthelasma confined to the eyelids often exists apart from jaundice, but general xanthelasma is almost always associated with chronic jaundice. The eruption of xanthelasma shows itself in the shape of patches



of the colour of chamois-leather (Rayer); they are slightly prominent, but non-indurated, and the edges are clear or irregular. Another form—**xanthelasma tuberosa**—is characterized by firm reddish-yellow nodules. Xanthelasma commences at the inner canthus of the eye, extends to both eyelids, and finally spreads over the palms of the hands, the soles of the feet, the elbows and the knees, showing a well-marked tendency to symmetry. Xanthelasma, from the anatomical point of view, is characterized by chronic hyperplasia of the derma, with fatty infiltration of the newly-formed elements; there is no tendency to ulceration.

**Pathogenesis—Ætiology.**—Gubler divided jaundice into two classes—hæmapheic, or hæmatogenous, and bilipheic, or hepatogenous. We shall see the meaning of these two varieties.

Bilipheic jaundice is due to retention of bile, and its production is readily understood. When an obstacle is opposed to the free flow of bile, biliary stasis in the liver and consequent absorption occur. The glands, says Bernard, are, or may be, organs that are as active in absorption as in secretion. The intrahepatic absorption of bile is carried out by the lymphatics rather than by the veins. The obstacle to the circulation of the bile may be situated in the liver, in the intrahepatic canals (catarrh of the bile-ducts, cancer of the liver, cirrhosis, abscess, appendicular liver, etc.), in the large excretory ducts, and at the duodenal orifice. To this last category belong obstruction by calculi, catarrh of the common duct, worms, hydatids, cicatricial or inflammatory constrictions of the common duct (ulcer of the duodenum), primary cancer of the biliary passages or the head of the pancreas, aneurysms, and tumours situated in the hilum of the liver.

In obstructive jaundice the colour of the skin is very pronounced, and the fæces are colourless if the large ducts are quite blocked. Furthermore, the urine contains biliverdin, as shown by nitric acid, and the blood-serum assumes a greenish-yellow tint.

Such are the characteristics of bilipheic jaundice. In other diseases, however, the skin and mucous membranes present a more or less deep subicteric tint, although the urine does not contain bile pigment. We may treat this urine with fuming nitric acid, and obtain an old mahogany tint, but not Gmelin's reaction. This variety is Gubler's hæmapheic jaundice. His explanation is as follows:

In the normal condition the red corpuscles destroyed in the system give rise to a substance called hæmaphein, which is converted into bile pigment in the liver. If the liver, in consequence of lesions or functional troubles, can no longer bring about this conversion, or if the destroyed corpuscles are in such numbers (pyrexia, toxæmia) that the liver cannot convert their débris, an excess of hæmaphein is formed, and is eliminated by the kidneys, giving special characters to the urine. So far there is no colouring of the skin, but if the kidneys do not eliminate the colouring matter, the tissues take on a yellow tint, and hæmapheic jaundice results.

Gubler's ingenious theory is no longer accepted. In the first place, hæmaphein has never been isolated, either from the urine or from the serum. In the second place, the blood pigment does not play the part assigned to it by this theory; the chief factor is the liver cell, on which the evolution of the hæmoglobin and the formation of the pigments depend. The colour reactions in the so-called hæmapheic urine are due to urobilin and a red-brown pigment, its chromogene (P. Tessier). Urobilin (Jaffé) belongs to the series of iron-free pigments derived from hæmoglobin, and while it is not found in normal urine, it is present in the fæces, which owe their ordinary colour to it. Hayem, who has carefully studied this question, found that urobilin dissolved in water and in urine gave with the spectroscope a dark band of absorption between the green and the blue. Urobilin, according to Hayem, is the pigment of the diseased liver: when the liver cell is attacked by organic and physiological decay, it elaborates urobilin and other modified pigments instead of normal bile pigment.

Urobilin is really the result of a change in the hæmoglobin; this change takes place in the liver, and not in the blood-serum. Urobilin, when reabsorbed, gives urobilinæmia, and its excretion by the kidney causes urobilinuria.

Urobilin, however, is not the only modified pigment. Winther has isolated a red-brown pigment of supreme importance. These modified pigments, which are sometimes associated with the normal but masked bile pigments, give rise to the so-called hæmapheic jaundice. These various kinds of jaundice, however, are not absolutely distinct; they may follow one another or may occur together, and may be classified in the following manner (Hayem):

1. Bilipheic jaundice, with normal bile pigments in the urine and in the serum of the blood. On examining the urine, we obtain Gmelin's reaction. The fæces are colourless when the retention of the bile is sufficient. Urobilinuria may or may not be associated with this form of jaundice.

2. Gubler's hæmapheic jaundice. In the urine presence of urobilin and modified bile pigments; absence of true pigments. In the serum, bile pigments and a trace of urobilin; fæces without special characters. In reality hæmapheic jaundice does not exist, because urobilin has an extremely feeble staining power. It is therefore more correct to say that there are varieties of jaundice, with urobilin and other modified pigments.

3. Slight jaundice, in which the urine contains urobilin alone, whilst the serum contains urobilin and bile pigments.

4. Jaundice in which the urine and the serum contain urobilin alone. Custom has now prevailed: the name "hæmapheic icterus" has been abandoned, and we describe two varieties—true or bilipheic jaundice, and jaundice with urobilinuria. These ideas are of much importance in prog-



nosis; in bilipheic jaundice the hepatic cell is healthy; in urobilinuric jaundice it is diseased (Hanot, Hayem). Urobilinuria and jaundice indicate degeneration of the hepatic cell.

In urobilinuric jaundice the urine stains the linen with salmon-coloured spots. In the test-tube the colour varies from amber-yellow to red-brown, but the greenish sheen observed in true jaundice does not occur. Nitric acid gives an old mahogany colour, and never the green tint due to bile pigment.

The spectroscope is indispensable in the examination of the urine, because it shows whether the brown colour of the urine is due to true bile pigment or to urobilin. The use of the small hand spectroscope makes this examination easy, even at the bedside of the patient. The method is as follows :

We commence by looking through the spectroscope, directing its extremity towards the light. The normal solar spectrum is, then, seen, the colours succeeding one another from left to right in the following order : red, orange, yellow, green, blue, indigo, violet. The instrument must be carefully focussed until the edges of the spectrum are clearly defined. The cleft in the spectroscope is, then, carefully adjusted, so that the normal lines of the spectrum are clear. Two lines, B and C, are seen in the red ; one line, D, in the orange ; one line, E, at the edge of the yellow and the green ; and one line, F, in the green.

A small glass reservoir, containing the urine, is then placed between the light and the end of the spectroscope. Normal urine does not affect the solar spectrum to any appreciable extent ; such is not the case when the urine is charged with bile pigments or with urobilin. Urine containing bile pigments blots out the whole right side of the spectrum, and the large dark band commences a little distance to the right of the E line, leaving between this line and the left extremity of the dark zone only a narrow luminous band of greenish-yellow.

Urine charged with urobilin gives two dark bands in the spectrum : the first one completely obscures the violet and the indigo, and darkens the right part of the blue ; the second is a black band, situated in the green, and completely masking the F line. If a few drops of ammoniacal chloride of zinc are added to the urine containing urobilin, it becomes dichroic, rose-coloured by direct light, and green by reflected light. The dark band which obscured the F line is displaced towards the left, and the F line becomes visible to the right of this band. This displacement of the band of absorption and the reappearance of the F line on addition of a few drops of ammoniacal chloride of zinc are characteristic of urobilin.

The distinctions just laid down in the pathogenesis of icterus are often met with clinically, but there are cases (in the bilious fevers, for example, in malaria, or in certain intoxications of the liver) in which the causes and the varieties of jaundice are associated.

**Appendicitis** may cause either variety. In Section XIX. I have described a case of early jaundice with urobilinuria of **toxic** origin, and a case of jaundice associated with purulent infection of the liver. These cases of appendicular jaundice are important in clinical medicine.

The **prognosis** must always be reserved, because **icterus gravis** sometimes

commences like the simple catarrhal form. Jaundice accompanied by fever or by adynamic symptoms must be looked upon with suspicion. We shall see in the following section that pregnancy is a grave factor. Chronic jaundice is often the index of formidable lesions. There is no **treatment** for jaundice *per se*. The treatment must be directed to the causative lesions. The kidneys must be watched; milk diet and mild diuretics must be prescribed.

**Cholæmia.**—In addition to jaundice, which is the complete clinical expression of the impregnation of the organism by bile, we see mild attenuated bilious conditions. Sometimes the skin is slightly jaundiced, but no pigment is found in the urine; this condition is known as acholuric jaundice. Sometimes the skin is normal, the urine contains no bile, and the only sign of biliary impregnation is the presence of bile pigments in the blood; this condition is known as cholæmia. This morbid syndrome (cholæmia) has recently been isolated by Gilbert and Lereboullet, who have made important contributions to the subject.

The principal symptom of cholæmia is furnished by the examination of the serum, which generally assumes an olive-yellow tint when it is collected in a vessel. With the spectroscope, it shows absorption bands in the green part of the spectrum; with nitric acid it gives Gmelin's reaction. The urine contains urobilin, but not true bile pigments.

According to the authors above quoted, cholæmia may be accompanied by dyspepsia, pain in the stomach, and diarrhoea; renal troubles—albuminuria and hæmoglobinuria; subacute or chronic biliary pseudo-rheumatism; cutaneous troubles—erythema, urticaria, pruritus, bullous eruptions, lentigo, xanthelasma, pigmentation, and melanoderma; visceral and cutaneous hæmorrhage; cardio-arterial phenomena, intermittence, and hypotension; psychical troubles—melancholia, hypochondria, and neurasthenia. The predominance of these troubles in a given case will allow us to classify the forms as dyspeptic, hæmorrhagic, or nervous.

Cholæmia is said to predispose to canalicular infections, and may be complicated with acute or chronic hepatic troubles (lithiasis and biliary cirrhosis).

Cholæmia may supervene as a symptom in many primary and secondary affections of the liver; it may also exist as a kind of idiopathic disease—*i.e.*, simple cholæmia and family cholæmia.

The anatomical substratum, variable in the first case, seems in the second case to be a discrete and perhaps infective angiocholitis. The diagnosis is probable when abundance of urobilin is present in the urine, and certain when bile pigments are found in the serum. The treatment is practically identical with that of other hepatic affections.



## XXVI. ICTERUS GRAVIS—ICTERUS AND THE PUERPERAL STATE.

**Nature of the Disease—Pathogenesis.**—Transient icterus is in itself a benign symptom. With few exceptions, the tissues may be impregnated by bile without the least danger resulting. We meet people who have hepatic colic or catarrh of the biliary passages, with temporary obliteration of the common bile-duct, and experience no serious inconvenience from jaundice that has lasted for several weeks. We also see people suffering from hypertrophic biliary cirrhosis with persistent jaundice, and yet they preserve their appetite, their strength, and their appearance of health. In these different cases the relative benignity of the icterus arises, on the one hand, from the healthy condition of the kidneys, which eliminate the pigment and the bile acids (toxic substances), and, on the other hand, from the capital fact that the hepatic cells preserve their functional activity. The danger of certain kinds of icterus, then, does not come from the icterus itself, and here, as elsewhere, the icterus is little more than a symptom. The danger, as we shall see later, comes from the destruction of the hepatic cells, and the name **icterus gravis** answers to a somewhat ill-defined morbid group, the principal characters of which are as follows :

An individual is in perfect health. After a few days of fever, lassitude, headache, muscular pains, and prostration, symptoms that simulate the invasion of influenza or typhoid fever, or after some gastro-intestinal trouble which might easily be taken for a simple catarrh of the digestive passages, slight or pronounced icterus supervenes. The fever is more or less high. Epistaxis, hæmorrhage from the gums, hæmatemesis, melæna, hæmaturia, purpura or ecchymoses, then appear, and are accompanied or followed by nervous troubles, such as vomiting, hiccough, dyspnœa, delirium, convulsions, or coma, and in the majority of cases the patient is carried off within two to three weeks, with ataxo-adynamic symptoms and hyperthermia or hypothermia.

In the foregoing example **icterus gravis** strikes the individual in the midst of good health, appearing as a primary disease, resembling typhus, yellow fever, or some other infectious disease, and breaking out at times in an epidemic state. In addition to primary icterus gravis, we find a series of cases in which icterus gravis does not appear as a morbid entity, but as a **secondary condition** in the course of some other disease (malignant syphilis, typhoid fever, or pneumonia), in pregnancy, or in the course of a disease of the liver. Thus, in an individual suffering from cancer of the liver, venous or biliary cirrhosis, gall-stones, hepatic tuberculosis, or hepatic syphilis, symptoms of icterus gravis appear, and the icterus in such a case is only an **episode** in the course of the hepatic disease.

We must not speak of one but of several forms of icterus gravis (Jaccoud) ; there are also cases in which the syndrome of icterus gravis is due to acute intoxication (phosphorus), and in this section we shall have to separate each of these forms.

Former observers, struck by the course and the symptoms of this disease, gave it the name of **icterus gravis** (Ozanam), in opposition to icterus benignus ; this name implied malignancy, without in any way specifying a distinct morbid entity. On the other hand, the predominance of certain symptoms gave the disease several names : **typhoid icterus** (Lebert), **fatal icterus**, **hæmorrhagic icterus** (Monneret).

Later, our ideas concerning the disease were modified. Icterus gravis was no longer considered a syndrome, and writers wished to make it a distinct morbid entity, with its own anatomical character. Rokitansky had already announced that icterus gravis was the result of acute atrophy of the liver. Frerichs confirmed his researches, and several writers called it **acute yellow atrophy of the liver**. Subsequent researches proved that the liver is not always atrophied, that the lesions of the cells do not consist solely in atrophy (Robin), and that irritative lesions have a certain part in it, and the disease for a time received the name of **diffuse parenchymatous hepatitis**.

It may be said that the nature of icterus gravis is now established. Icterus gravis, as Bright, Budd, and Trousseau saw it, is an infective disease which in some features resembles yellow fever. It has its origin in the rapid anatomical and physiological decay of the cells of the liver, with or without atrophy of the organ. The liver is not only attacked in its histological elements, but also in the chemical constitution of its parenchyma (Quinquaud) ; the hepatic cell may be physiologically destroyed, while the morphological changes appear to be slight. In short, the multiple and important functions of the liver are suppressed, glycogenesis is abolished, and, as a consequence, there is rapid decay of the nervous, vascular, and muscular systems. The hepatic cell is no longer able to arrest the passage of the poisons and pepto-toxines brought by the portal vein ; the products of digestion undergo incomplete elaboration ; the extractives, in the form of urea, no longer undergo their final oxidation ; the bile acids are incompletely elaborated, or, in a word, the hepatic function tends to be suppressed, and the liver "is no longer the citadel advanced against the infection," but, on the contrary, becomes the starting-point of secondary infections. To these causes of infection and hepatic intoxication are often joined causes of uræmic intoxication, as the renal epithelium may be attacked.

Another question now arises : we say that icterus gravis is due to destruction of the hepatic cells. What agent produces this change ? Is it a micro-organism or a toxic substance ? Bacteriological researches have shown



that various microbes may be associated with the lesions and symptoms of icterus gravis. The *Staphylococcus aureus* has been found by Legall in two cases of primary icterus gravis, and by Girode in one case of icterus gravis during pregnancy.

In the cases reported by Hanot various microbes were found. A young woman died of icterus gravis. Streptococci were found in the small ducts and veins of the liver, and in the serum from the lung and the spleen. An alcoholic youth was carried off by icterus gravis, and the *Bacillus coli* was found in the hepatic cells and capillaries. A man suffering from cancer of the biliary passages died of icterus gravis, and the *Bacillus coli* was found in the bile and in the blood of the cardiac cavities.

Boix reports the following cases: In a patient suffering from icterus gravis, with hypothermia, Hanot and Boix found the *Bacillus coli* in the blood and in the liver four hours after death. In a patient suffering from icterus gravis, with hyperthermia, the same authors found the *Staphylococcus albus* in the blood twenty-four hours before death, and the same staphylococcus in the liver half an hour after death. Babès has published four cases of icterus gravis terminating rapidly in death, and associated with the streptococcus. Hanot has communicated two cases of icterus gravis that supervened in pneumonia and in secondary cancer of the liver. In the first case the pneumococcus was found in the liver, and in the second case the pneumococcus was found in the blood of the cephalic vein five hours before death. Achard has published two cases of primary cancer of the liver ending in icterus gravis; in each case the *Staphylococcus albus* was found in the liver during life. Bar and Rénon have reported the case of a new-born child who died of icterus gravis complicating hepatic syphilis. The *Proteus vulgaris* was found in a pure state in the blood of the umbilical vein, in the liver, the spleen, and the heart.

This enumeration proves that icterus gravis may be associated with the *Bacillus coli*, streptococcus, staphylococcus, pneumococcus, and *Proteus vulgaris*. There can, therefore, be no question of a specific disease, and we have proof that the hepatic cell, under certain conditions of morbid receptivity, may be attacked by various pathogenic agents, and undergo changes which produce the syndrome of icterus gravis.

The hepatic lesions of icterus gravis are, however, chiefly due to the toxins. The poison comes in some cases, perhaps, from ptomaines absorbed by the liver. This hypothesis of a poison attacking and destroying the hepatic cell is realized in phosphorus-poisoning, which exactly reproduces the syndrome of icterus gravis: steatosis, destruction of the hepatic cells, icterus, hæmorrhages, nervous symptoms, and hypothermia.

*Summary:* Whether the pathogenic agent acts as an infective or as a toxic agent, we may say that the pathogenesis of icterus gravis is being cleared up. The only difference between primary and secondary icterus gravis is that the former attacks an individual whose liver is apparently healthy, whereas the latter attacks a person whose liver has been already damaged. It is possible that the difference between the primary and secondary forms is not so pronounced, for it is not proved that in the so-called primary forms the liver has not been surprised in a condition of morbid imminence, as a *locus minoris resistentiæ*. The varieties of icterus

gravis would then all belong to one class, though distinct from one another, according to the scale of gravity, in the same manner as cases of benign icterus may be grouped in another class, having only the scale of benignity as their distinctive sign.

The so-called primary icterus gravis, which so closely resembles **yellow fever nostras**, must not be completely abandoned. It is much rarer than secondary icterus gravis, but its symptoms are more clearly pronounced, and it will serve best as a type for our description.

**Pathological Anatomy.**—In icterus gravis the liver is generally small; the atrophy may be such that the liver may weigh only 15 to 20 ounces. Glisson's capsule, having grown too large for the atrophied parenchyma, is furrowed. In some cases, if the disease is very rapid, the liver remains of normal size, and the lesions are only appreciable with the microscope. The organ is hypertrophied when icterus gravis occurs as a secondary trouble in a person whose liver was already enlarged (malaria, alcoholism, or hypertrophic cirrhosis). Most frequently the yellow colour of the liver is not uniform, but presents mottlings and reddish islands (red atrophy). The parenchyma is softened, friable, or sometimes diffuent, and in a section the lobular appearance is no longer seen. The liver contains but little blood and bile, and there is also but little bile in the gall-bladder and the large bile-ducts.

Under the microscope the following lesions are seen: At the parts most affected the liver cells have disappeared, and are replaced by an amorphous veinstone, infiltrated with embryonic cells, hæmatoidin, bile pigment, and fatty granules. Elsewhere the cells remain, but have undergone complete disintegration; the protoplasm has disappeared, and the cell is infiltrated with fatty granules and bile pigment. "The cloudy condition of the cells and the infiltration with bile is the first phase in the degeneration, which ends finally in fragmentation and destructive softening of the elements." This destructive process has been explained in various ways. According to some writers, it is simply a degenerative process; according to others, it is an irritative one. The irritative process seems at times to be limited to the cells, passing over the connective tissue (parenchymatous hepatitis); at other times the connective tissue is also affected (Frerichs). The hepatitis is both parenchymatous and interstitial, and the bloodvessels participate in the process.

In certain cases, if the patient dies so rapidly that the lesions have not time to develop, or if, for other reasons, the changes in the liver are less marked, the cells show cloudy swelling, and the parenchyma encloses a quantity of extractives. Chemical analyses have shown that in some cases the extractives have increased twofold. The bloodvessels, and especially the hepatic veins, are changed; they are almost bloodless, and contain much



leucin and tyrosin. The bile-ducts are almost always involved; we find angiocholitis of the small canals, which are obliterated by cellular detritus (Bamberger). This lesion is probably the cause of the icterus, unless we admit, with Frerichs, that the icterus is due to the compression of the biliary canaliculi by exudation into the periphery of the lobules. Cornil, in one case, found the interlobular canaliculi dilated and filled with cells, and the intralobular network abnormally developed. This intra-acinous network, which has been noticed by other authors, is said to appear in the last stage, when the fatty detritus of the parenchyma is absorbed. It is probable that the biliary canals are intact in the very rare cases in which icterus is almost absent. The **kidneys** are always affected, and show an early stage of parenchymatous nephritis. Many convoluted tubes are blocked by large cloudy, granular, fatty, and deformed cells. A certain dependence has rightly been found between the lesions of the kidneys and of the liver. According to some writers, the lesion of the kidneys, following that of the liver, is said to be caused by the icterus and by the elimination of the bile; according to others, the renal and hepatic lesions are said to be dependent on the same cause. Both theories are accepted.

The **blood** shows the abnormal fluidity found in cases of pyrexia and in the infectious diseases (Vulpian). It has little tendency to coagulate, being diffuent, blackish, and tarry. It is deficient in red blood-corpuscles, and is loaded with leucin, tyrosin, and xanthin, due to incomplete oxidation of the proteids. According to some writers, it is said to contain less urea. The hæmoglobin affected by the changes in the extractives is said to absorb less oxygen than in the normal condition (Quinquaud). The lungs, pleura, capsule of the liver, spleen, and kidneys may show hæmorrhages. The spleen is usually enlarged and softened. The heart is soft and fatty, endo-pericarditis being common (Jaccoud).

**Symptoms.**—I shall first describe so-called primary icterus gravis, and then briefly mention the secondary forms. The onset is, as a rule, insidious. The disease may at first resemble the simple catarrhal form, the symptoms of icterus gravis appearing a few days later. Sometimes the disease begins with a violent rigor, accompanied by extreme prostration, rachialgia, and vomiting; but this sudden and acute onset which recalls the outburst of certain pyrexias is more rare than a gradual onset. For some days the patient complains of lassitude, pains in the limbs, prostration, and low spirits, resembling the invasion of influenza or of typhoid fever. At other times gastro-intestinal troubles open the scene, and form the chief feature. A diagnosis of gastric trouble is at first made. The malaise, however, increases, fever appears, and from the third to the eighth day icterus sets in. From this moment three classes of symptoms dominate the situation—viz., icterus, hæmorrhage, and nervous troubles.

The **icterus** varies from the lightest yellow colouring of the conjunctivæ and of the skin to a green or bright yellow colour. There is, however, no relation between the gravity of the trouble and the degree of the icterus, and if I insist on this point, it is because an old theory assigned to the absorption of bile an important part in the production of the trouble. The icterus is due to divers causes. When it is the result of absorption of the bile, nitric acid gives the well-known green reaction in the urine; but there are cases in which the urine, treated with nitric acid, does not reveal pigment of hepatic origin, and is coloured a dirty brown. Gubler called this condition **hæmaphic icterus**. These two forms may alternate or may occur together.

As the disease progresses, the icterus may disappear—a bad sign, proving the decay of the hepatic cell (Jaccoud). The fæces are lighter than in the normal condition.

**Hæmorrhages** are, so to say, constant, appearing in the course of the disease or in its last stage. Epistaxis, which may be seen from the commencement, purpura, hæmorrhage from the gums, hæmatemesis, and melæna, are the most common; hæmaturia, hæmoptysis, and metrorrhagia are much rarer. These hæmorrhages are favoured by the fluid condition of the blood. An attempt has been made to explain them by the passage of the bile into the blood; this theory must be abandoned. Experiments have decided the point; and, furthermore, the same hæmorrhagic diathesis is found in other diseases, such as black smallpox, purpura, and typhoid affections, and the explanation does not require the presence of bile in the blood. The hæmorrhages, as in phosphorus-poisoning, depend on the destruction of the hepatic cell, and Galen was correct in saying, *Hepar sanguificum*. The **nervous troubles** consist in phenomena of excitation: slight delirium, dyspnœa, hiccough, insomnia, tremor of the lips, subsultus tendinum, convulsive movements; and in phenomena of depression: sleepiness, stupor, and coma. These troubles of excitation and of depression occur together, or follow one another without a fixed rule. The symptoms of depression often open the scene, and the symptoms of excitation appear later, being followed by coma.

The **pulse** and the **temperature** present marked variations. The pulse may be quick or slow. The temperature may rise to 103° or 104° F., or else it is lowered from the commencement of the disease, and remains low until its last stage (Wunderlich). There is, therefore, no fixed relation, from the point of view of prognosis, between the febrile condition and the gravity of the general symptoms, but some relation may be established between the temperature and the nature of the infective agents. Recent researches have proved that the *Bacillus coli* and its toxine cause hypothermia (Dupré, Netter, Boix); it is certain, as experiments and clinical medicine prove, that, in a general way, the intoxications and infections



caused by the *Bacillus coli* produce a subnormal temperature, whilst the infections and intoxications of other microbes—streptococcus, staphylococcus, or pneumococcus—raise the temperature, often causing considerable hypothermia. These same distinctions have been formulated by Hanot, as regards the diseases of the liver, and especially of icterus gravis. Boix has collected thirty-four cases of icterus gravis, in which the temperature, curve, and nature of the pathogenic microbe are indicated. He shows that in hepatic infection by the streptococcus, staphylococcus, or pneumococcus, etc., the temperature rises to 104° or 105° F. (hyperthermia); on the other hand, in hepatic infections by the *Bacillus coli* the temperature is lowered to 95° F. (hypothermia). There are, of course, mixed and intermediate cases.

The two morbid types of icterus gravis, hyperthermic and hypothermic, are seen, as Boix has pointed out, in two morbid species which are dissimilar as regards pathogenesis, but which both end in the syndrome of icterus gravis—*i.e.*, yellow fever, with its high temperature (hyperthermia); and phosphorus-poisoning, with its low temperature (hypothermia).

The examination of the **urine** furnishes information of great value. The urinary secretion is generally diminished. Cases have even been quoted where the anuria was complete for several days. The icteric tint of the urine is more or less pronounced. It is generally a case of bilipheic icterus; in some cases, however, the reaction of the so-called hæmapheic icterus has been noticed. The urine is often albuminous; it contains hardly any urea at an advanced period of the disease, but, on the other hand, leucin and tyrosin are found in quantity in almost all cases. The urea is diminished because, according to certain writers, the liver being the principal producer of urea, this salt is less when the liver is destroyed, or when its functions are suspended. The urine contains leucin and tyrosin, because the liver no longer fulfils its normal functions; the combustion of the proteids is incomplete, and, instead of producing uric acid and urea, which are the last stage in the oxidation of the proteids, it only produces less oxidized products, such as leucin, tyrosin, and xanthin.

The examination of the liver does not always cause pain; it reveals the atrophy of the organ. The spleen is enlarged. The skin shows such eruptions as erythema, roseola, miliaria, and urticaria.

**Course, Duration, Prognosis.**—I have already dwelt on the insidious and variable onset of icterus gravis. According to the predominance of hæmorrhage or of adynamic symptoms, hæmorrhagic and typhoid forms have been described; but this division is not, in my opinion, suitable, the mixed form being the more frequent. The **prognosis** is extremely grave; in some exceptional cases the patient succumbs in two or three days, and the course is lightning-like. The final symptoms generally supervene from the sixth

to the tenth day. However, icterus gravis is not absolutely fatal, and the name of **fatal icterus**, given by some English writers, is, happily, not justified. There are some undoubted cases of cure, and they are, indeed, more frequent than would at first be supposed, considering the custom of looking upon icterus gravis as a sentence of death. In truth, it may well be asked why this so-called essential icterus gravis, or **yellow fever nostras**, should not be cured like other infectious cases; therefore I do not see why this should be a reason for making a special variety, and for grouping the recoveries under the name of icterus pseudo-gravis. **Recovery** is sometimes announced by a **crisis** of polyuria, increase of urea, abundant diaphoresis, and profuse diarrhoea. The urinary toxicity, which is very much lowered during the disease, exceeds its normal coefficient at the moment of the crisis. The condition of the kidneys is of great importance in the prognosis; the danger is less when the secretion of urine is free. Parotiditis (suppurative inflammation) has several times coincided with recovery.

**Diagnosis.**—It is very difficult to make a diagnosis at the commencement of the disease; later, when the principal symptoms have appeared, icterus gravis somewhat resembles other diseases accompanied by icterus, such as ulcerative endocarditis, purulent infection, poisoning by phosphorus, and yellow fever. Icterus is very rare in infective endocarditis; hæmorrhage is absent, and auscultation of the heart reveals the murmurs of endocarditis. Purulent infection originates from a wound or injury, and is characterized by repeated rigors, by violent attacks of fever, and by pain in the hepatic region, due to pyæmic abscesses in the liver. Poisoning by phosphorus resembles icterus gravis so closely that, in the absence of any history or of traces of the poison in the vomit, it would often be very difficult to make a diagnosis. We shall see later the complete analogy existing between **vomito negro** and icterus gravis, which has sometimes been called **yellow fever nostras**.

Another difficulty presents itself: for some few days a patient has icterus supposed to be catarrhal. Fever is present, repeated epistaxis occurs, and purpura appears. How are we to know whether these symptoms are due to benign or grave icterus? The analysis of the urine, the discovery of albumin, and the quantity of urea give insufficient information. Diagnosis and prognosis often remain in doubt, and it is a case of saying, with Trousseau: "In icterus, as in pleurisy, we never know how it may end."

**Ætiology.**—The so-called primary icterus gravis is far less common than secondary icterus gravis. It sometimes appears suddenly in an apparently healthy subject; at other times it develops through predisposing causes, such as pregnancy, alcoholism, excesses in eating, or gastro-intestinal auto-infection. A person, although, at the time being, in good health, has previously suffered from hepatic colic, symptoms of biliary lithiasis, or jaundice,



and the affected liver has not quite lost all traces of the disease. Another individual, though, for the time being, in good health, has some hereditary predisposition, and, by a combination of circumstances which make pathological heredity a very complex question, an organ, such as the brain, the kidneys, the heart, or the liver, is less resistant to the repeated attacks on life. Under the influence of conditions with which we are still imperfectly acquainted, icterus gravis has been epidemic, but it must be stated that the epidemic remains limited. The focus does not extend beyond a barracks, prison, or ship. It is not rare to see relatively benign cases which resemble catarrhal icterus during the same epidemic by the side of serious cases. Kelsch is of the opinion that the infective agent is of telluric origin.

**Secondary Icterus Gravis.**—This form supervenes during the course of some disease (typhoid fever, cholera, or pneumonia), which has placed the liver in a condition of receptivity. Pregnancy has this effect, because it renders the liver liable to infection by steatosis of the hepatic cell. Any disease of the liver may be complicated by the hæmorrhagic and nervous troubles of icterus gravis. I quote as such : alcoholic, biliary, cardiac, and tubercular cirrhosis ; cholelithiasis ; cancer ; permanent obstruction of the biliary canals ; hydatid cysts ; and hepatic syphilis. In these various lesions the door is opened to the secondary infections and toxines, the affected liver cells being no longer in a state of defence. Some of these cases refer to patients already suffering from icterus, and in them the disease takes the name of aggravated icterus, which is often “ only the last act in the morbid process.”

The symptomatic picture of secondary icterus gravis is almost the same as that of the primary form ; it is, however, less marked, and the course is less rapid, because the destruction of the hepatic cells is from the first not so general as in essential icterus gravis.

The course and gravity of the trouble depend much on the previous condition of the patient. Sometimes the complications of aggravated icterus hasten the end fairly quickly ; at other times they follow one another slowly in a subacute form, and they may recover.

**Icterus and the Puerperal State.**—As I have already remarked, icterus due to gall-stones is not, as a rule, serious in a pregnant woman ; such is not the case in the other varieties comprised under the term of “ infective icterus.” Whatever theory is put forward to explain the changes in the hepatic cell matters little ; as soon as the cell participates in the toxi-infectious process, it undergoes an adulteration which, under other conditions, might not have serious consequences, but which in the gravid condition is always to be feared. When icterus appears in pregnancy, it is always of serious import (I am speaking of icterus occurring apart from hepatic colic), because it indicates a hepatic lesion that may lead to insufficiency. Hepatic

insufficiency is sometimes accompanied by albuminuria and renal insufficiency, the epithelium of the kidneys being directly affected by the toxic-infective agent, or suffering injury from the elimination of poisonous bile.

Le Masson has collected thirty-nine cases of icterus during pregnancy and thirteen cases of icterus after confinement. The thirty-nine cases show thirteen recoveries and twenty-six deaths. The thirteen cases occurring after accouchement show three recoveries and ten deaths. These figures indicate the gravity of puerperal icterus.

The icterus of pregnancy appears chiefly after the third month, and it is generally impossible to find any factors other than the gravid condition. The icterus is sometimes preceded by nausea, vomiting, gastralgia, and colic; in other cases it appears without any previous manifestation. The jaundice may be slight or intense, from a subicteric tint to deep and general icterus. The urine contains bile pigment and urobilin in unequal proportions; albumin is often found. The fæces are clay-coloured. The fever is slight or severe. The liver tends to be enlarged, and is sometimes painful. The other symptoms include anorexia, headache, diarrhoea, or constipation, and are extremely variable. After a duration of a fortnight to a month, the symptoms improve, the icterus disappears, the pregnancy follows its course, and the patient recovers, without further disturbance. This is the benign form.

In other cases, if the hepatic lesion is more severe or more prolonged, the situation becomes serious, and the disease ends in miscarriage, premature confinement, or even in the death of the mother. Finally, in some cases the disease assumes the characters of icterus gravis, with hæmorrhages, epistaxis, purpura, ecchymotic patches, violent headache, delirium, restlessness, dyspnoea, and tendency to coma. "In these grave forms, miscarriage, premature confinement, or even accouchement at term, generally occurs whilst the syndrome is at its height, from two to five days after the onset of icterus, and very often on the second day; but there is occasion to insist on their suddenness. Physicians sometimes discuss the question of intervention when the woman is about to be confined, as if, after such a termination, it were permissible to hope for recovery! The relative calm and feeling of well-being might lead us to form a favourable prognosis. This calm is, however, deceptive, and the amelioration is of short duration; the symptoms have only lulled, to reappear some hours later, when they cause death with terrible rapidity" (Le Masson).

Icterus appearing after the confinement is much more often grave than benign, since in thirteen cases it was followed in ten by death. It is, furthermore, almost always associated with puerperal infection. Puerperal icterus is sometimes epidemic. The following summary of seven epidemics is taken from Le Masson's monograph:



Epidemic of Lüdenscheld in 1879 ; epidemic of Roubaix, described by Carpentier ; epidemic of Saint-Pierre de la Martinique in 1858, described by Rouillé and by Saint-Vel (" out of thirty women attacked by icterus, twenty succumbed to coma after miscarriage, or premature confinement ") ; epidemic of Limoges, described by Bardinet in 1860 ; epidemic of the Maternité and of the Hôpital des Cliniques in 1870-1872, described by Hervieux, Depaul, and Meunier ; epidemic of Saint-Paul in 1873, described by Smith ; epidemic of Neusenstamm in 1876, described by Vinay.

In 1867 Hervieux tried to explain these troubles " by the presence of a special morbid ferment or toxic principle." We now speak of toxi-infection, the cause of which it is often possible to find. In short, no matter what pathogenesis is invoked to explain the icterus of pregnancy (leaving aside icterus due to gall-stones) it is none the less true that in the pregnant woman the liver cell no longer has the same antitoxic properties, so that toxi-infections readily bring about decay. The gravity of this condition, of which the icterus is one of the signs, must lead us to reserve our prognosis.

We must obviously divide icterus occurring in pregnant women into two great classes. If it is associated with cholelithiasis or occurs with hepatic colic, the prognosis is almost always good, because the liver cell is not affected. If the pregnant woman has neither hepatic colic nor gall-stones, or if the icterus supervenes as a toxi-infection of the liver, with or without participation of the kidney, the situation may become perilous.

**Treatment.**—Saline purgatives and diuretics must be administered at the commencement of icterus gravis. Milk diet is absolutely indicated. I have several times noted the utility of large and repeated injections of artificial serum ; two of my patients recovered. Intestinal antisepsis has been recommended. The hæmorrhages, vomiting, and nervous troubles demand symptomatic treatment. Most of these troubles unfortunately resist the best-directed measures.

## XXVII. CATARRHAL ICTERUS—PROLONGED CATARRHAL ICTERUS.

**Pathogenesis.**—Under Icterus Gravis we saw that there was a scale of gravity in its various forms, and so, too, benign icterus has a scale of benignity. Some are infectious, others are not, and those which are infectious are not in the same class.

Emotional icterus, for example, is not of infective origin. It does occur, as in the case quoted by Potain, of a man about to be shot ; in Rendu's case, of a young girl excited by catheterism ; and in Chauffard's case, of a man seized with a violent fit of temper. Emotional icterus, which may appear in less than an hour, is probably due to an excess of biliary secretion and to its absorption.

Under Angiocholitis I quoted several varieties of benign icterus, such as

syphilitic icterus of the secondary period, icterus from pigmentary hypercholia in the newly-born, and icterus associated with angiocholitis of biliary origin. These various kinds of icterus, which are generally benign, have already been described. I shall now especially deal with the condition described as catarrhal icterus.

Primary catarrh of the biliary passages, still called simple catarrhal icterus, because the icterus is the most apparent symptom, may invade the intrahepatic and also the extrahepatic bile-ducts. If the catarrh attacks the intrahepatic canals alone, icterus appears, because the bile is absorbed *in situ*, from the obliterated canals; but the stools are not colourless, because sufficient bile continues to pass into the intestine. On the contrary, if the catarrh attacks the common bile-duct, so that it is obstructed by the inflammatory products, obstructive icterus occurs, and is associated with more or less complete decoloration of the fæces. This last form is the type of so-called catarrhal icterus.

In the very rare cases where a patient with catarrhal icterus has succumbed to an intercurrent disease, inflammation of the common duct has been found post mortem; the inflammation may be limited to the intestinal portion and to the duodenal orifice of the duct (Virchow). The walls of this portion of the common duct, the connective tissues around it, and Vater's ampulla, show oedematous swelling, which constricts or blocks the orifice. "It will readily be understood that this oedematous swelling may obstruct the flow of the bile and produce icterus, when we remember that coryza prevents nasal respiration in consequence of the swelling of the mucosa." Moreover, the common duct at this level is blocked by a mass of epithelial cells, forming a kind of plug, which, unstained by the bile, completes the obstruction of the duct and opposes the passage of the bile into the intestine (Vulpian).

In a post-mortem examination performed by Müller the common duct was obstructed, the mucosa being swollen and covered with petechiæ; catarrhal obstruction of Wirsung's canal was also present.

The affected parts of the common duct preserve their normal dimensions (about 6 millimetres in diameter), and are not impregnated with bile, whilst the biliary passages above the obstacle are dilated. This obstacle to the flow of the bile causes icterus by retention, the constant and prominent symptom of catarrh of the biliary passages. Such is, in general terms, the chief lesion in catarrhal icterus. How is this lesion produced? According to an old theory, angiocholitis of the common duct was said to be associated with gastro-intestinal catarrh, and, in consequence, connected with the causes of this catarrh (high living, chill). Some individuals, it is true, suffer from gastro-intestinal troubles and icterus, following on orgies, acute alcoholism (and we know the influence of alcohol on the liver), or drunkenness, whence



the old name of *icterus a crapula*. This theory only takes account of a local process; it is founded on the spread of the catarrh from the duodenum to the common duct, with obliteration of the lumen.

Other theories are opposed to, or associated with, this theory of the disease. Catarrhal icterus is considered to be an infectious disease, due to internal or external infection. That catarrhal icterus is frequently a general disease, or a variety of bilious fever chiefly limited to the biliary canals, follows from the careful study of cases. It will thus be perceived, as Chauffard points out, that the disease commences with pre-icteric symptoms of fever, lassitude, anorexia, vomiting, epistaxis, insomnia, albuminuria, labial herpes, and sometimes with foetid bilious diarrhoea, indicating hypersecretion of the liver. The icterus appears three or four days after this period of invasion, and is soon followed by decoloration of the fæces. "The secretion of the urine and the excretion of the urea run a parallel course." In the first phase the urine is scanty, bilious, and poor in urea; in the second phase at the moment of the crisis the urine is abundant and rich in urea. In this form catarrhal icterus has the aspect of a general disease, and takes this aspect from its mode of appearance. Catarrhal icterus, as a matter of fact, is often, like certain fevers, a disease of certain seasons, whence the old name of vernal or autumnal icterus. It is epidemic, and attacks two, three, or more persons in the same house or in the same locality. These cases have been frequently noticed in the army.

Through these considerations, certain forms of catarrhal icterus are classed with general infective diseases, and side by side with icterus gravis, of which they are sometimes an attenuated form. The discord, however, arises when it becomes a question of stating the cause of the infection.

Chauffard is of the opinion that catarrhal icterus has its origin in the putrid poisons formed in the intestine. These poisons, or ptomaines, normally absorbed *in situ* in order to be eliminated by the liver, may, under certain conditions, increase their noxious properties and cause auto-infection, according to the expression of Jaccoud. Kelsch does not consider the ptomaines as the cause of the evil. "Is chemistry going to take us back to the humoral theories which time and experiments seemed to have killed, and which, besides, are so little encouraging to prophylaxis?" The infective agent is said to be exterior. "The ground appears to us to be the generating focus *par excellence* of this agent. The slimy bottom of ditches or of ponds seems to be the most favourable medium for the preservation and the multiplication of this agent." All these views are acceptable—a proof that infective icterus may result from multiple causes, and show slightly different aspects.

**Symptoms.**—In some cases the disease commences with gastro-duodenal catarrh. The patient suffers for three or four days from loss of appetite,

nausea, vomiting, constipation, furred tongue, pain in the epigastrium, prostration, headache, and fever. At this moment icterus appears. The yellowish tint first affects the conjunctivæ, forehead, and neck. Within a day or two the fæces become clay-coloured, and the icteric tint grows deeper and extends over the whole body. The urine, rich in bile pigment, takes on a saffron colour, and on the addition of nitric acid the characteristic greenish tint appears. In other cases the gastro-intestinal catarrh attracts less attention, and the disease from the first presents symptoms more like those of general disease. Lassitude, aching of the muscles, prostration, epistaxis, fever, bilious diarrhœa, scanty and albuminous urine, are the symptoms of the period of invasion, and recall the commencement of typhoid fever. Even when the icterus appears we are far from being assured as to the prognosis; we wonder whether we are not face to face with a case of icterus gravis, and we recall the phrase of Trousseau: "In icterus, as in pleurisy, we never know how it may end."

Finally, in some patients icterus is, from the first, the chief symptom of the disease; the urine contains bile and the fæces are colourless, but the patient has neither fever nor distaste for food. Catarrhal icterus may, therefore, present itself in various forms.

The clay-coloured stools are caused, on the one hand, by the absence of bile pigments, and, on the other, by the accumulation of fat in the intestine (steatorrhœa). As in the absence of bile the fat is no longer emulsified and does not pass into the lacteals, the fæces take on their putty-like appearance. As obliteration of the pancreatic duct accompanies that of the common duct, it has been maintained that the steatorrhœa is due to the absence of the pancreatic juice. Müller does not hold this view; he admits that the pancreatic juice alters the quality but not the quantity of the fat in the fæces, and it seems impossible to take sides for or against the bile or the pancreatic juice.

The **liver** is often enlarged, the hypertrophy being considerable if the disease is of long duration.

**Prolonged Catarrhal Icterus.**—In catarrhal icterus the common duct generally becomes permeable in the second or third week. The appearance of bile in the intestine indicates recovery; the fæces regain their colour; the urine increases in quantity, and gradually loses its bile pigment. There is often a crisis of polyuria and azoturia (Chauffard); the toxicity, previously normal, rises suddenly (Roger), but the coloration of the skin lasts for two or three weeks, until the renewal of the epidermis. The pulse is slow during the entire disease.

Such is the usual course of catarrhal icterus, but numerous exceptions occur. In several cases I have seen an attack of catarrhal icterus last more than two months, and I have, therefore, called this variety **prolonged catarrhal**



**icterus** ;\* it must be recognized in order to avoid mistakes in diagnosis. When we see prolonged catarrhal icterus and an enlarged liver in an elderly man, we are always tempted to think of cancer. We find analogous cases in the writings of various authors. According to Niemeyer, the "disease may drag on for weeks and months. The icterus becomes intense, the patient grows very thin, and the liver shows evident swelling." According to Frerichs, catarrhal icterus may last two or three months. Amongst the cases which I have seen, two occurred simultaneously in two members of the same family, and I attributed them to the ingestion of high game. The disease was marked by successive outbursts: the icterus, decoloration of the fæces, and yellow colour of the urine improved, and reappeared on several occasions, and the liver became very much enlarged. In one patient the disease lasted two months, and in the other three months, with epistaxis, while the liver remained enlarged for a long time, and only became normal after a season at Vichy. I saw at the Necker Hospital a case of prolonged catarrhal icterus lasting for seven weeks. These cases of relapsing catarrhal icterus have been designated by the name of Weil's disease.

**Diagnosis.**—The diagnosis of catarrhal icterus is easy if the disease announces itself with symptoms of simple gastro-intestinal catarrh, followed by icterus and clay-coloured stools. If, however, the general symptoms are severe from the first, loss of strength, epistaxis, albuminuria, and fever being the symptoms of invasion in the midst of which icterus appears, we naturally think of the possibility of typhoid fever or of icterus gravis. The diagnosis and the prognosis must, for the time being, be reserved.

Cholelithiasis also provokes obstruction of the common duct, accompanied by symptoms of icterus, bilious urine, and colourless fæces, greatly resembling those of catarrhal icterus. But the patient with cholelithiasis in most cases suddenly feels the more or less sharp pain of hepatic colic, with bilious vomiting, vertigo, and rigors that so often accompany the passage of the calculi. On the other hand, he has not, as a rule, the symptoms which in catarrhal icterus form the pre-icteric phase.

**Syphilis**, in its secondary stage, sometimes causes catarrhal icterus; the pathogenic diagnosis has been given under Syphilis of the Liver.

When catarrhal icterus is prolonged and the liver is enlarged, the diagnosis is especially difficult. We must differentiate it from hypertrophic biliary cirrhosis, cancer of the liver, and cancer of the head of the pancreas, compressing the common duct. Hypertrophic biliary cirrhosis and prolonged catarrhal icterus have, as common symptoms, persistent icterus and an enlargement of the liver, but the decoloration of the fæces seen in catarrhal icterus is not met with in hypertrophic biliary cirrhosis; at least, if it is

\* Dieulafoy, "De l'ictère Catarrhal Prolongé" (*Semaine Médicale*, July 11, 1888).

observed, it is not so marked or so persistent as in catarrhal icterus. Furthermore, the spleen is not hypertrophied in the case of catarrhal icterus.

Secondary cancer of the liver and prolonged catarrhal icterus may present, as common symptoms, icterus and enlargement of the liver, and even decoloration of the fæces, if the common duct is compressed by the cancer (glands of the hilum); but the swelling of the liver is uniform in the case of catarrhal icterus, while it is accompanied by nodules, and often by ascites, in secondary cancer. I am not speaking here of massive cancer of the liver, because icterus is absent in this variety.

The diagnosis between prolonged catarrhal icterus and cancer of the head of the pancreas, compressing the common duct, is, in my opinion, a most difficult problem. In both cases we find persistent icterus, enlarged liver (biliary plethora), and decoloration of the fæces and steorrhœa. Theoretically, it might be thought that the obstruction of the common duct by cancer of the head of the pancreas would be gradual and much slower than the obstruction of the duct in the case of catarrhal icterus; practically it is not always so, and, to quote examples, in three patients in my wards the cancerous obstruction of the duct was almost immediate, and at an advanced stage of the disease a copious flow of bile followed a long period of retention. It might likewise be supposed that the examination of the fatty matter in the intestine might give useful information as to the absence of the pancreatic juice, but we have already seen that, according to Müller, the diagnosis cannot be based on the study of steorrhœa; and, again, the pancreatic duct may be obliterated in both cases. Rapid loss of flesh and sugar in the urine are not constant in pancreatic cancer; diarrhœa and cutaneous pigmentation are in favour of cancer. According to Salhi, salol, which is a combination of salicylic acid and phenol, is decomposed in the intestine into its two elements by the pancreatic juice. If, therefore, salol is given to a patient whose pancreas is healthy, salicylic acid and phenol will appear in the urine. If they are not found, the salol has not been decomposed, because there is no pancreatic juice in the intestine. This procedure, supposing it were exact, would not be enough in the present case to make the diagnosis positive, because Wirsung's duct may also be obliterated in catarrhal icterus. It is only the course of the disease which will remove the doubts and permit us to affirm the existence of prolonged catarrhal icterus.

The diagnosis between prolonged catarrhal icterus and cancer of Vater's ampulla presents for several weeks the greatest difficulties. The **prognosis** of catarrhal icterus is generally benign, but yet in the presence of icterus, even of the most simple kind, we must always make some reserves, because **icterus gravis** may commence like benign infective icterus; and we see in the



same epidemic very benign cases of catarrhal icterus, terrible cases of icterus gravis, and mixed or intermediate cases.

**Treatment.**—When angiocholitis is associated with gastro-intestinal catarrh, we should first prescribe a saline purgative. The patient is dieted and is given bitters, extract of quinine, sweetened with syrup of orange-peel (Jaccoud), alkaline beverages, and Vichy water.

Some years ago Krüll employed a new method of treatment for catarrhal icterus. He gave daily a cold enema of water at a temperature of 60° to 65° F. After a few enemata, the bile generally appeared in the intestine, the duct became permeable, and recovery took place in a few days. I have not obtained such good results. The ingestion of large doses of oil has likewise been extolled, but few patients are willing to submit to this treatment, the results of which, by the way, are open to discussion.

### XXVIII. HÆMOLYTIC ICTERUS.

**History.**—Side by side with icterus of hepatic origin, due to a cellular lesion or to an obstacle to the excretion of the bile, there is a special variety of icterus, due to the exaggerated formation of bile pigments by the destruction of the red corpuscles. It was to this form of icterus that Gubler gave the name of hæmapheic icterus and which is, at present, described as **hæmolytic icterus**. Normal blood serum contains a certain quantity of bile pigments which are derived from the hæmoglobin, freed by the wear and tear and the destruction of the red corpuscles. If this hæmoglobin be freed in a greater quantity than normal, the impregnation of the integument by the pigments thus formed will be intense enough to provoke real icterus. The principal character of this icterus is that it is not accompanied by choluria and that it presents a blood formula which, whilst preserving its special physiognomy, is the only means by which a diagnosis can be made.

This idea of hæmolytic icterus was first propounded by Mirskowski, in 1900, in a case of chronic, acholuric, hereditary icterus. It was, however, proved to exist by Chauffard, who saw that there was, in certain cases, a fragility of the corpuscles which thus accounted for the hæmolysis. By a new technique, Widal and his pupils proved the fragility of the corpuscles in certain cases of icterus which are not congenital, but acquired, and they completed the history of hæmolytic icterus.

This variety of icterus has then, at present, its autonomy and its clinical characteristics. The examination of the blood is, however, necessary in order to diagnose it. The nature of this icterus can be affirmed only after the hæmolysis has been noted. Before studying the various clinical aspects under which it may be met with, it will be well to recall the methods of procedure which enable us to prove the existence of hæmolysis.

**Examination for Hæmolysis.**—The red blood corpuscles have a certain power of resistance as regards fluid media, the maximum of which is realized by the molecular concentration of the blood serum. If they be introduced into a fluid the concentration of which is inferior to this plasma, the corpuscles swell, and, finally, dissolve, giving up their hæmoglobin. There will, then, be hæmolysis. Normally, however, the red corpuscles show a resistance which varies within known limits and, by measuring the “resistance” of the corpuscles, the degree of their “fragility” can be appreciated. It is this fragility, then, the causes of which are still obscure, that presides over hæmolytic icterus. In proving the fragility of the corpuscles, the hæmolytic nature of the icterus is demonstrated. The degree of resistance can be measured by putting a drop of blood into a series of tubes containing solutions of chloride of sodium which gradually diminish in strength, starting from the normal concentration of the serum of the blood. These tubes are compared with one another and, at the moment when the resistance of the corpuscles is overcome, that is to say when hæmolysis takes place, the liquid above the residue assumes a yellow tint. We can, thus, ascertain to what degree of molecular concentration the limit of resistance of the corpuscles corresponds, and, if it be below the normal, we are right in stating that fragility of the corpuscles is present (Vaquaz and Ribierre).

In some cases of acquired icterus, this technique is insufficient to demonstrate the hæmolysis. Widal, Abrami, and Brulé have simplified the method by operating not on the blood fluid, but on the hæmatites, separated from their serum, which seemed to mask their fragility by giving them the normal power of resistance. This method of using deplasmated hæmatites brought out the hæmolytic nature of certain cases of icterus in which the fragility of the corpuscles might have passed unnoticed. Measuring the resistance of the corpuscles is, thus, the basis of the diagnosis of hæmolytic icterus, and it was necessary to refer to this fact first, before reviewing the clinical characteristics of this form of icterus.

**Description.**—There are two principal types of hæmolytic icterus: Congenital icterus (Chauffard) and acquired icterus (Widal). Each of these varieties has its idiosyncrasy, but they have common characteristics which will be described. Three symptoms dominate the clinical picture: Icterus, splenomegalia, and the modification of the blood. The icterus is, generally, acholuric. Gemelin’s reaction reveals no trace of biliary pigment, but, on the other hand, urobiline is most frequently met with. Retention of the bile never exists. The fæces may be even hypercoloured. The yellow tint of the skin may undergo variations of intensity, according to fatigue, work, and gastro-intestinal troubles. Finally, and this is a clinical detail of importance, no signs of biliary intoxication,



pruritus, bradycardia, or wasting, and no nervous symptoms are present. Such is the clinical aspect of this form of icterus, the dominant features of which are absence of choluria and of biliary retention.

The second element of this syndrome is the increase in the size of the spleen, which can usually be felt on palpation, but which is not painful. This splenonchus is not accompanied by an increase in the size of the liver, contrary to what is found in hepatic icterus. The changes in the blood play a preponderant rôle in this syndrome and give it its special physiognomy. The number of red corpuscles is greatly diminished and often falls to about 3,000,000. The hæmoglobin may fall as low as 55 per cent. of the normal amount. The specific characteristic of this form of icterus, however, is the diminution in the resistance of the corpuscles, which is proved by one of the methods indicated. In a patient with normal resistance, hæmolysis hardly appears in the test tube which contains a 0.44 per cent. solution of chloride of sodium, whilst in hæmolytic icterus the fragility is manifest as soon as the tube contains a 0.68 per cent. solution, and the diminution of the corpuscular resistance peculiar to this variety of icterus is thus accounted for. Another characteristic is the presence in the blood of granular hæmatites in the proportion of, at least, 10 per cent. They are never found in normal subjects. They may, it is true, be met with in a large number of cases of anæmia, but they are most frequent in hæmolytic icterus (Chauffard and Fiessinger).

Diminution of the resistance of the corpuscles with all its consequences—hæmolysis and a yellow colouring of the serum, presence of granular hæmatites—are the hæmatological characteristics which are common to every case of hæmolytic icterus and which give it its idiosyncrasies. In the picture which we have presented on broad lines, some varieties are met with which, while retaining the principal symptoms described above, present, nevertheless, some peculiarities as to their origin and evolution, which make real clinical forms of them. Let us now, compare congenital and acquired icterus with one another.

(1) **Congenital hæmolytic icterus** (Neinkowski, Chauffard) constitutes a remarkably fixed type as to its symptomatology. Often running in families, it commences, most frequently, in infancy. In this case, the icterus is essentially chronic and sufficiently accentuated to be easily recognized. It is not influenced by errors in diet, but by fatigue and by anything which can accelerate the destruction of the muscular hæmoglobin. It is remarkably constant and does not proceed spasmodically as acquired icterus does. It is not accompanied by signs of biliary intoxication, or by discoloration of the fæces. The spleen, which is always very large, projects beyond the false ribs and may, sometimes, fill the entire left flank, whilst the liver remains normal. The hæmatological signs present

are:—Very pronounced corpuscular fragility and the presence, in great quantity, of granular hæmatites. The anæmia is little accentuated and not accompanied by any functional troubles. The hypoglobulia is very moderate. The dominating feature of the clinical picture is the icterus and, according to Chauffard: "The patients are rather icteric than pale. The evolution is, essentially, chronic, and the prognosis is in nowise severe."

(2) **Acquired hæmolytic icterus** presents a less perfect unity than congenital icterus does. Its causes are multiple. Its evolution varies and its symptoms are sufficiently constant to offer only the slightest variations from one ætiological type to another.

The icterus is, in this case, less pronounced than in the congenital form. "The patients are rather pale than icteric." Anæmia is the dominant symptom, the pallor of the skin and of the mucosæ is visible under the icterus. A few functional troubles—want of breath and palpitation—are noticed. In opposition to the congenital form, the number of red corpuscles is considerably diminished, and may fall to 2,000,000 and to even less. The splenomegalia is not as considerable as in the preceding form, and, on the other hand, the size of the liver is generally increased.

It is interesting to study the reaction of the blood. Here also, a manifest diminution in the corpuscular resistance is noted, but the hæmolysis is appreciable only by employing deplasmatised hæmatites, which explains why this form was not individualized at the same time as the congenital form. Furthermore, this variety has a character of its own, viz. the auto-agglutination of the hæmatites which is realized *in vitro* by placing the patient's red corpuscles in contact with his own serum in the proportion of one to ten, which does not exist in congenital hæmolytic icterus. The granular hæmatites are very abundant. Leucocytosis is also noticed and signs of blood regeneration, characterized by elements of medullary origin, viz. nucleated hæmatites, megaloblasts, myelocytes.

Such are the various signs of congenital hæmolytic icterus, and it will be seen that, if the icterus be somewhat in the background, the hæmatological characteristics are, on the contrary, preponderant. The peculiarity of this form of icterus is that it spreads by successive outbursts of deglobulization, interspersed by phases of repair. Most frequently, it is curable. Based on the nature of its evolution, it has been found possible to distinguish an acute form supervening as an epiphenomenon during the course of an infectious disease, and ending at times in icterus gravis; a sub-acute form, which we have taken as our type and which is the most interesting, as its pathology lends itself readily to discussion.

**Ætiology—Pathology.**—In what ætiological and pathogenic conditions may this hæmolytic icterus supervene? If congenital icterus be always



identical, notwithstanding its, otherwise, obscure ætiology, acquired icterus, on the contrary, may show itself in fairly diverse forms. This is the variety in which, it seems, we ought to include the, so-called, idiopathic icterus of the newly born, the idiopathic nature of which is, now, proved; the icterus of infectious diseases, pneumonia, typhoid fever, paludism, syphilis, the icterus of intoxications, the icterus of certain forms of cirrhosis and of anæmia, and certain cases which Gubler has called hæmapheic.

The mechanism which produces this icterus seems to depend on two processes: In certain cases, we have to deal with a special corpuscular fragility in which the hæmatites show the reaction of auto-agglutination and in which they are hæmolyzed by the salt water, whilst the serum has, of itself, no hæmolyzing action. In other cases, the serum is said to contain special substances, hæmolysines, which are said to destroy the red corpuscles and to set their hæmoglobin free. It is not exactly known where the destruction of the red corpuscles takes place. Is the liver (Hagen), or the spleen (Chauffard) to be incriminated, or are the red corpuscles destroyed in the circulating blood (Widal)?

**Pathological Anatomy.**—We have, by the way, sufficiently indicated the histological characteristics of the disease, so that it is unnecessary to dwell on its pathological anatomy. Hypertrophy and congestion of the spleen, hyperactivity of the osseous marrow, absence of lesions of the hepato-biliary apparatus are its positive and negative characters which were easy to foresee. The infiltration of the ochre pigment in the spleen, the liver, and the kidneys (Castaigne) constitutes a stigma peculiar to hæmolysis.

**Diagnosis.**—The result of this description shows that the diagnosis of hæmolytic icterus is, sometimes, fairly delicate. If the congenital type be sufficiently peculiar to attract attention, acquired icterus, on the other hand, borrowing the mask of common infectious icterus, runs the risk of not being traced to its proper cause. The absence of choluria and of hepatic signs, the intensity of the anæmia, hypertrophy of the spleen may already carry conviction. The examination of the blood remains the basis of the diagnosis, and it alone can give certainty. By noticing the diminution of the corpuscular resistance and the presence of the granular hæmatites with a lowering of the number of the red corpuscles, we can affirm the diagnosis and differentiate the acquired from the congenital forms. This point being established, it is possible, by more complete methods, to discover whether the case be one of icterus caused by corpuscular fragility, or one of hæmolytic icterus. The treatment of hæmolytic icterus is purely symptomatic. The corpuscular destruction and the anæmia must be fought by prescribing iron in the form of protoxalate, in doses of from 0.20 to 0.40 centigrammes a day. Administered alone, it has given appreciable results, and in certain cases brought about a cure.

## CHAPTER VIII

### DISEASES OF THE PANCREAS

#### I. GENERAL SURVEY OF THE DISEASES OF THE PANCREAS.

THE pancreas may be the seat of irritative, destructive, or neoplastic changes that interfere with the normal functions of the gland. The resulting symptoms in some cases indicate the lesion. The physical signs here are of small importance, as the pancreas is too deeply seated for palpation to be possible; only large tumours of pancreatic origin can be felt. Nevertheless, in certain affections of the pancreas the epigastrium is very sensitive to pressure, and acute pains of a neuralgic kind are sometimes felt. Mirallié, who has carefully studied these pains, attributes them to neuralgia of the coeliac plexus, which is in direct contact with the upper edge of the pancreas.

As physiologists have proved the importance of the pancreatic secretion in intestinal digestion, it might reasonably be thought that changes in the pancreas would induce characteristic digestive troubles. Nothing of the kind is the case. The distaste for fatty and proteid substances, the difficulty of digesting fats, and intestinal meteorism, are banal symptoms.

**Pancreatic vomiting** is said to supervene several hours after meals, and to consist of viscous and thready liquid. **Pancreatic diarrhoea** is said to be characterized by drops of fat which has not undergone emulsion; these little drops are whitish and soluble in ether (steorrhoea). Diabetes is an important symptom of extensive changes in the pancreas. Removal or experimental destruction of the whole pancreas causes severe glycosuria (Mering and Minkowski). As a matter of fact, the pancreas pours into the blood an internal secretion, which prevents glycaemia (glycolytic ferment, Lépine). The preservation of a small fragment of the pancreas suffices to prevent glycosuria. **Pancreatic diabetes** (Lancereaux) has some special characteristics—viz., sudden onset, acute course, loss of flesh, and rapid cachexia. The termination by galloping consumption is frequent in pancreatic diabetes, but even in the absence of this complication, the disease is of short duration, and the patient dies in five or six months from cachexia or diabetic coma. Diabetes may also be due to syphilis of the pancreas.



## II. CANCER OF THE PANCREAS.

The pancreas may be invaded by cancer in a neighbouring organ. More often it is a case of primary cancer of the head, body, or tail of the pancreas. Cancer of the head of the pancreas is the most common form, and is also the easiest to diagnose.

The relations between the common duct and the head of the pancreas explain the obliteration of this canal by cancer. As I have discussed this question in detail, I shall merely mention in this section cancer of the pancreas, which is not accompanied by icterus, the explanation being that the neoplasm is in the body or the tail of the organ.

In such a case the symptomatology is very obscure: loss of flesh and of appetite, distaste for fatty substances, and dyspeptic troubles. Obstruction or ascending infection of the excretory ducts of the pancreas may cause fibrosis and diabetes. When the growth involves the solar plexus, it causes a bronze coloration of the skin. Cancer of the pancreas may invade the prevertebral glands, the vena cava, the portal vein, and the superior mesenteric artery. When it obstructs the vena cava, it causes cyanotic œdema of the subdiaphragmatic half of the body; when it compresses the portal vein, it causes ascites and collateral circulation. Thrombosis of the superior mesenteric artery brings about intestinal infarcts, followed by bloody diarrhoea and fatal peritonitis.

The disease usually spreads to the liver. The cancerous nodules are multiple, lenticular, and transparent, like grease-spots. Sometimes they form large white nodules, softened in the centre, which Gilbert calls coconut cancer.

The duration of cancer of the pancreas is short, and death generally supervenes in five or six months; I have, however, seen a patient in whom the affection lasted twenty-one months.\* In Section VII. we shall see that hypertrophic pancreatitis may simulate cancer.

## III. CYSTS OF THE PANCREAS.

I do not refer here to dilatation of the pancreatic canaliculi met with in chronic pancreatitis and lithiasis, nor to cysts consequent on the encystment of an intraglandular hæmorrhage. I shall only discuss the large cysts, which may reveal themselves clinically. These cysts are sometimes multiple, and constitute cystic disease of the pancreas. Sometimes (and, indeed, most often) a large unilocular cyst develops, and exceeds in size the head of an adult. It is formed of a fibrous wall, smooth externally, irregular and anfractuous internally, and filled with a limpid and colourless fluid.

\* *Clinique Médicale de l'Hôtel-Dieu*, 1897-1898, p. 212.

As the cyst starts from the pancreatic region behind the stomach, it can only grow by insinuating itself between the organs situated in front of it. Sometimes it develops between the stomach and the liver, pushing the stomach downwards and coming in contact with the anterior abdominal wall; it is covered by the distended gastro-hepatic omentum. More commonly it pushes the stomach upwards and the transverse colon downwards, lodging in the large omentum, which it doubles up as it grows. In some rare cases, adhesions of the large omentum prevent the doubling-up, and the cyst can only grow by insinuating itself under the colon.

The cyst, on account of its situation, produces early troubles of compression—viz., uncontrollable vomiting, acute paroxysmal pain in the epigastrium, and intermittent intestinal obstruction. These troubles have nothing characteristic, and it is only when the tumour is present that we can make a diagnosis. When the cyst develops downwards (subcolic type) it may readily be taken for a cyst of the mesentery or of the ovary. In cysts which grow upwards the special prominence formed by the cyst above the umbilicus sometimes permits of a diagnosis. The tumour pulsates, because the aortic pulsations are transmitted to it, but is not expansile. It is generally separated from the liver by a resonant zone. The only treatment is surgical intervention. Puncture of the cyst gives deplorable results. Laparotomy, on the contrary, cures the patient in most of the cases. In many cases adhesions necessitate incision and marsupialization.

#### IV. HÆMORRHAGE OF THE PANCREAS.

The rarity of hæmorrhage is so great that we do not think of it. It presents, however, a fairly constant symptomatology. In most cases the patient is alcoholic or a sufferer from gall-stones. Without any appreciable cause, he is seized with sharp epigastric pain and rigors, and he has an anxious look. Examination at this moment would furnish no explanation for this condition, except tenderness of the epigastrium on deep pressure. The following day the condition becomes worse; the facies is peritoneal, the pulse is small and frequent, the belly is tender in the upper part, and nausea or vomiting occurs. The extremities grow cold, the temperature falls, the voice is lost, and the patient dies of collapse twenty-four to forty-eight hours after the onset of symptoms.

At the post-mortem examination blackish blood is found behind the omenta and around the pancreas, which is partially or entirely transformed into a blackish diffuent mass, having the look of a clot of blood. There is difficulty in recognizing the lobules pushed aside by the blood. In some places the blood is brownish, as if digested, and this transformation seems, indeed, to be the result of auto-digestion by the pancreas. This auto-



digestion may go so far that the organ becomes apparently gangrenous. Auto-digestion has also been noted when hæmorrhage was the result of epigastric traumatism. The hæmorrhage is often associated with cyto-steatonecrosis (*vide* Section VII.).

## V. PANCREATITIS.

Most of the infectious diseases and of the intoxications may cause pancreatitis. The acinous structure of the pancreas is then destroyed; the epithelial cells are destroyed, and affected with cloudy swelling, necrosis, and fatty degeneration; bands of interlobular or intralobular fibrous tissue break up the parenchyma. These lesions are found in typhoid fever, pneumonia, dysentery, diphtheria, malaria, and, with some variants, in poisoning by phosphorus, mercury, and alcohol. Changes in the pancreas may also be found in diseases of the kidneys, the liver, the heart, and the stomach. Infective and toxic pancreatitis have been reproduced experimentally.

The lesions of pancreatitis only acquire an interest when they are sufficiently acute to cause suppuration, or deep enough to produce the entire degeneration of the pancreas and fibrosis of the gland.

**Suppurative Pancreatitis—Abscess of the Pancreas.**—Abscesses of the pancreas may be the result of infection through the blood, as in Macaigne's case, where several pneumococcal abscesses developed in the pancreas after bronchopneumonia. The condition is more frequently a local trouble due to infection of intestinal origin, which spreads along the canaliculi. This infection is favoured by lesions of the excretory canals of the pancreas. Suppurative pancreatitis is frequently seen as a complication of neoplasms of the head of the pancreas and of pancreatic calculi. Pancreatitis is often caused by perinephritic abscess.

**Sclerous Pancreatitis—Sclerosis of the Pancreas.**—In certain cases of pancreatic diabetes we find at the post-mortem examination that the pancreas is atrophied and indurated.

## VI. PANCREATIC CALCULI.

We may find in the excretory canals of the pancreas concretions of carbonate of lime, rounded like a pea or elongated like a grain of wheat. They are often multiple. They induce secondary dilatation of the pancreatic duct and the formation of small retention cysts. They are one of the principal causes of suppuration in the pancreas and of pancreatic fibrosis. They cause crises of epigastric pain (pancreatic colic), which is very difficult to diagnose and to ascribe to its true cause,

Pancreatic colic exists in three out of four cases of pancreatic calculi. It is accompanied by vomiting, and in some cases by a tendency to syncope. The pains radiate towards the first lumbar vertebra, between the shoulders, to the groins, and into the deep parts of the abdomen.

Diabetes is very common, having been found in thirty-six out of eighty cases of pancreatic calculi. The quantity of sugar varies from a few grains up to 15 ounces in the twenty-four hours. It is, as a rule, permanent, but it may be intermittent, occurring only during the attacks of colic. Lazarus quotes two cases, in which the glycosuria was alimentary and not spontaneous.

Eickhorst has often noticed indicanuria, which he considers an indication that the diabetes arises from calculi in the pancreas.

Examination of the fæces shows excess of fatty matter, intact muscular fibres, and much fatty acid in crystals, all pointing to pancreatic insufficiency.

Pancreatic calculi have been found in the stools. The stones may be rounded, coral-like, or prickly. They vary in size from a pin's head to a pea; they are friable, and readily dissolve in water. Their weight varies from 3 to 30 grains. Their chemical composition is : carbonate and phosphate of lime, 72 parts to magnesia, 4; and organic matter, 3. Dieckhoff has found the *Bacillus coli* and streptococci in the centre of the calculi.

X-ray photographs of these calculi have been made by Lazarus. They are not transparent to the rays and give a clear shadow.

## VII. RELATION BETWEEN PANCREATITIS AND GALL-STONES — PANCREATICO-BILIARY SYNDROME — CYTOSTEATONECROSIS AND PANCREATICO-PERITONEAL HÆMORRHAGE.

This section deals with the relations that may exist between pancreatitis and gall-stones. This question is of great importance in medicine and surgery, and we shall see that pancreatitis is beginning to occupy a most important situation. Much of the matter in this section is taken from ten of my clinical lectures on this question at the Hôtel-Dieu.

Those who suffer from gall-stones may be attacked by the following complications : acute and chronic pancreatitis; fibrous pancreatitis, compressing the common bile-duct and Wirsung's duct; cirrhosis of the pancreas, with overgrowth of tissue, simulating cancer of that organ; suppuration and gangrene of the pancreas; pancreatitis simulating acute peritonitis and perforation of the abdominal viscera; sudden onset of cytosteatoncrosis and pancreatiko-peritoneal hæmorrhage.

I have thought it better to discuss this question under Diseases of the Pancreas, and not under Gall-Stones, first giving a résumé of a case at the Hôtel-Dieu :



On April 3, 1906, a man was admitted for severe jaundice of some six weeks' duration. The urine was of an orange colour, and nitric acid gave the characteristic reaction of bile pigment. The fæces were quite colourless, and contained much fatty matter.

Fever was absent, itching severe. The liver was slightly enlarged, but was not painful. Neither tumour nor pain over the gall-bladder. Examination of the supra-umbilical region, above and external to the navel—the area corresponding to the head of the pancreas and the course of the common duct—revealed no swelling, induration, or pain. The patient had been suffering from obstructive jaundice for six weeks. It remained to find out the cause of the obstruction; everything pointed to a gall-stone in the common duct.

The patient had suffered during the past eighteen months from typical hepatic colic, and on February 15, 1906, the jaundice had appeared. On March 15, 1906, after a very bad attack of colic, Dr. Silvy found a cylindrical gall-stone, which must, from its shape, have increased in size while in the common duct. Moreover, cylindrical calculi in the common duct are fairly common, because Jordan has collected some fifty cases. Millard has found several specimens, and Chauffard has written an interesting monograph on "Lithiasis of the Common Duct." He found, on making a median longitudinal section of a calculus, a series of concentric and ascending stratifications of cholesterin, showing the growth of the calculus at the upper part.

The patient had obviously been suffering from jaundice, caused by a gall-stone in the common duct. The problem was to explain the persistence of the jaundice after the stone had been passed two weeks previously. Either the duct contained other gall-stones, or else it was compressed by fibrous pancreatitis, which is often associated with gall-stones.

The jaundice showed no change for some weeks after the patient's admission under my care; the stools were colourless, and the urine contained bile pigment. As the patient lost nearly 2 stones in weight, I began to doubt the diagnosis of chronic pancreatitis; and if I had not seen the gall-stone, I should have suspected cancer of the head of the pancreas. I handed the patient over to Terrier for operation.

Gosset operated on April 23. At this date the jaundice had lasted for sixty-seven days (thirty-seven days after the expulsion of the gall-stone). The abdomen was opened by Kehr's bayonet incision. The liver was pulled upwards and backwards, in order to examine the gall-bladder. It was small, retracted under the liver, and partly hidden by omental adhesions; no calculi were present. Cholecystectomy was then performed.

The common duct was explored, first with a No. 10 olivary bougie, and then with a curette, which passed over an obstacle at the ampulla of Vater into the duodenum. The duct and the ampulla contained no calculus.

The hepatic duct was next examined by a lateral incision in the common duct up to the hepatic duct. No stone was found.

Examination of the head of the pancreas showed marked induration in Desjardin's triangle of infection; the tissue was so hard that there was a question of the presence of a calculus. Puncture with a needle showed no calculus, but fibrous pancreatitis. The hepatic duct was drained.

In a fortnight the bile found its way into the bowel; the jaundice disappeared; the urine and the stools became of normal colour. Six weeks after the operation, the patient having gained some 30 pounds in weight, left the hospital cured.

It is easy to reconstruct the history of the case. The patient had been suffering from a single gall-stone in the common duct. The passage of the stone put an end to the hepatic colic, but the symptoms of biliary obstruction still persisted, being due to stenosing pancreatitis, that prevented the passage of the bile. Drainage of the hepatic duct and withdrawal of the bile externally cured the pancreatitis, and rendered the common duct permeable.

**OTHER CASES.**—Before studying as a whole the relations between pancreatitis and gall-stones, I think it desirable to quote several cases.

**RIEDEL'S CASE.**—A woman had been subject to hepatic colic for a long while. Six months before, severe jaundice appeared after an attack of colic. The liver was enlarged. Below the liver a tumour as hard as iron and as large as the fist was felt. It was thought to be the gall-bladder, filled with calculi. An operation was performed. After separation of the adhesions between the liver, pylorus, and duodenum, the gall-bladder was found to be contracted and filled with calculi. The tumour, however, was formed by the head of the pancreas. The common duct was as large as the thumb; it contained six calculi, which were removed through an incision. Cholecystectomy and drainage. In three weeks the patient was cured; in six months the pancreatic tumour had diminished in size, and in eighteen months it could no longer be felt.

**MAYO ROBSON'S CASE.**—A lady, fifty years of age, had suffered for several years from hepatic colic, followed by jaundice. For some months the attacks had been more severe, and the jaundice had become permanent. Vomiting was frequent. The patient had become very thin and weak. Fifteen stones were removed from the common and cystic ducts. The head of the pancreas was invaded by a large nodular mass. The gall-bladder was anastomosed with the duodenum. Mayo Robson and his colleagues were in favour of cancer. The tumour was, however, really due to chronic hypertrophic pancreatitis associated with gall-stones. Recovery was uneventful; ten months after the operation she was in excellent health.

**QUÉNU'S CASE.**—A young woman had suffered for five years with hepatic colic, followed by jaundice. For three months the jaundice had been permanent, and the patient had lost a stone in weight. Abdominal palpation was negative. Quénu performed cholecystostomy. The gall-bladder contained two large calculi, but the common and cystic ducts contained none. The obstruction to the flow of bile was caused by induration of the head of the pancreas. The results of the operation were normal; after a month the bile passed into the intestine, the biliary fistula closed, and recovery was complete in ten weeks.

**KEHR'S CASES.**—1. A woman had suffered for fifteen years from attacks of pain in the region of the gall-bladder, with vomiting and jaundice. In the last two years the attacks became more frequent; the patient had lost 2 stone in weight in three months. The steps of the operation were: bayonet incision; removal of the gall-bladder, which was small, full of calculi, and adherent to the omentum. The head of the pancreas, affected by interstitial pancreatitis, was as hard as a stone. The cystic and common ducts were free. Kehr drained the hepatic duct. The pain ceased, and the patient recovered. Examination of a fragment of the pancreas, removed at the operation, showed chronic sclerous pancreatitis.

2. A patient had complained for some years of hepatic colic. The last few attacks were very painful, and were followed by severe jaundice. Kehr diagnosed obliteration of the common duct by a gall-stone. The steps of the operation were: bayonet incision; freeing of adhesions between the gall-bladder and the omentum. The pancreas was enlarged and as hard as a stone. A calculus found in the common duct was pushed back into the gall-bladder. Cholecystectomy. Drainage of the hepatic duct. Complete recovery in six weeks.

3. A woman suffered from hepatic colic, with jaundice and clay-coloured stools. Examination under chloroform revealed near the umbilicus an indurated tumour, which appeared to be the head of the pancreas. Operation: bayonet incision. The gall-bladder was distended and adherent to the pylorus; several stones could be felt in the gall-bladder, and one in the cystic duct. Removal of the gall-bladder. No calculi in the common duct. Examination of the pancreas showed that the tumour was the



hard and thickened head of that organ. The hypertrophic pancreatitis had obstructed the flow of bile through the common duct. Drainage of the hepatic duct. Three months later the patient was in good health.

In all these cases the pancreatitis was associated with the presence of gall-stones, either in the gall-bladder or in the ducts. In other cases, however, the pancreatitis still develops, and causes obstruction to the flow of bile through the common duct, although the calculi have passed through into the bowel.

In some instances, indeed, we can follow the evolution of the pancreatitis both in the calculous and in the non-calculous stages. This feature was well shown in the first case quoted. The pancreatitis persisted after the expulsion of the calculus, until the patient was operated upon.

Korte speaks of a similar case :

He performed cholecystectomy in a woman suffering from cholelithiasis. As the pain and jaundice reappeared, he feared that he had left a stone in the ducts and operated a second time. He found hypertrophic pancreatitis, preventing the flow of bile through the common duct. Drainage of the hepatic duct. Complete recovery.

MAYO ROBSON'S CASE.—A woman was suffering from hepatic colic and jaundice. Under chloroform, a hard tumour was felt; it had irregular edges, and was situated between the umbilicus and the costal margin. On opening the abdomen, adhesions were found between the stomach and the liver. The pancreas contained a tumour of malignant appearance; the prognosis, therefore, appeared hopeless. As a matter of fact, it was a case of hypertrophic sclerous pancreatitis. The patient ate a chop three days after the operation; in two weeks the jaundice had disappeared, and some weeks later the patient went home well. Four months later she was in good health; the pancreatic tumour had disappeared.

#### PANCREATICO-BILIARY SYNDROME—PANCREATITIS STENOSANS AND PANCREATITIS EXUBERANS.

The foregoing cases would have been formerly called "jaundice from obliteration of the bile-ducts by calculi." The pancreatitis was usually unnoticed, or called cancer. The progress made in the surgery of the bile-ducts has rendered the close study of pancreatitis possible, giving it an important place in the group of affections due to gall-stones.

In 1905 Quénu and Duval collected 118 cases of pancreatitis associated with gall-stones. To these figures I have been able to add some fifty more cases. Kehr, from a systematic exploration of the pancreas in every operation he has performed on the bile-ducts, says that pancreatitis is present in 33 per cent. These figures show the frequency of the morbid association, which I shall call the "pancreatico-biliary syndrome."

What share does the pancreas take in this association? As we have seen in the preceding cases, the lesion is usually limited to the head of the pancreas, the remainder of the organ being free. The head is cirrhotic, and sometimes as hard as a stone. The cirrhosis has in most cases a hyper-

trophic tendency. The hypertrophy is at times so marked as to give rise to a hard, nodular, and ill-defined tumour, which even at the operation resembles a malignant growth, and lends itself to errors in diagnosis. This morbid process demands our attention. I have proposed for these varieties of pancreatitis the terms "pancreatitis stenosans and pancreatitis exuberans." The former variety surrounds and flattens the common duct, and in some cases Wirsung's canal as well, preventing the flow of bile and of pancreatic juice into the intestine. The latter variety causes stenosis, but it also forms a tumour, which may compress the duodenum.

Histological examination shows interstitial and parenchymatous lesions. Opie has noted the following varieties: interstitial pancreatitis, interlobular cirrhosis, and peri-acinous intralobular cirrhosis. The remarkable feature is the tendency to cause stenosis and overgrowth. The head of the pancreas may be three or four times its normal size. The hypertrophy, however, does not go on indefinitely; atrophy may occur at a later stage.

What is the explanation of the occurrence of pancreatitis in the course of cholelithiasis? Many theories have been formulated. We know that the common duct lies in a kind of gutter on the posterior surface of the pancreas, and also that the common bile and pancreatic ducts open side by side at the ampulla of Vater. One theory says that the pancreatic tissue is infected by reason of its close proximity to the common duct; another theory says that the infection, of biliary or duodenal origin, passes upwards through Wirsung's duct into the pancreas.

It must not be thought, however, that the development of chronic pancreatitis is favoured only by calculi in the common duct or in the ampulla of Vater. In Quénu and Duval's statistics we find that pancreatitis usually coexists with calculi in the common duct, but in many cases the common duct and the ampulla of Vater are both free, the calculi being in the gall-bladder or in the cystic duct. Sometimes the calculi have been expelled, and yet the pancreatitis continues to develop. In short, cholelithiasis may, in the long-run, cause pancreatitis, and calculi in the common duct are most favourable to this complication. Moreover, calculi are not the only factor, and account must be taken of the more or less septic condition of the bile. We may ask if infection starting from the intestine may not be the origin of ascending hepato-pancreatitis, causing, on the one hand, lithiasis, and, on the other, pancreatitis (Desjardins). In fact, the pathogenesis of these varieties of pancreatitis is not yet cleared up.

**Symptoms and Diagnosis.**—It is not easy to state the symptoms of pancreatitis. The first point is that the mischief only occurs in persons who have suffered for a long time from cholelithiasis. For years they have had hepatic colic and jaundice, and have been under treatment at Vichy, Carlsbad, Vittel, or Contrexéville. Others have suffered from calculous



cholecystitis, with or without jaundice. In some patients with pancreatitis the symptoms of biliary lithiasis—pain, hepatic crises, and more or less prolonged jaundice—have been present for eighteen or nineteen years. Apart from some exceptions, pancreatitis only supervenes when cholelithiasis is of long standing.

It is not common to find the onset of pancreatitis marked by acute pain. The condition is usually insidious in its development. And even supposing that pain occurs; as the patient has already suffered from gall-stone colic, it is difficult to know how much of the pain is due to the pancreatitis. Even when the pain is most marked at the pancreatic point (2 inches above and external to the umbilicus), it is not always easy to say whether this pain is due to pancreatitis or to calculi in the common duct.

Obstructive jaundice does not help in making a pathogenic diagnosis, because it may be caused by a calculus blocking the common duct or Vater's ampulla, in the absence of pancreatitis. On the other hand, obstructive jaundice may arise from pancreatitis stenosans, even though no calculus is present in the common duct or at Vater's ampulla.

Rapid wasting has been given as a sign of stenosing pancreatitis. It is said to be due to the fact that the pancreatic juice cannot pass into the intestine because Wirsung's duct is blocked. The statement is true; but this wasting is not special to pancreatitis, being seen in cancer of the head of the pancreas, and also in permanent obliteration of the common duct by a calculus.

Finally, we have enlargement of the head of the pancreas. The tumour is more or less bulky, painful, and indurated. Its situation is about 2 inches above and to the right of the umbilicus. It appears at first sight that this tumour might be of much value in diagnosis, and yet, as we have already seen, it is a frequent cause of error, being often mistaken for cancer of the head of the pancreas or for calculous cholecystitis.

The difficulties in diagnosis, therefore, are evident. How can we say whether a patient is suffering from cholelithiasis and also pancreatitis? Both the prognosis and the treatment are concerned. Much has been expected from the analysis of the urine according to the methods of Sahli and of Cammidge. The results given by these authorities are, however, not conclusive (Chauffard). Glycosuria is exceedingly rare.

Chemical and microscopical examinations of the fæces give the most valuable information. Stearrhoea, from deficiency of the pancreatic juice, is a marked feature. The fatty stools vary in appearance. "Sometimes we find small masses of fat, as large as a pea or a small nut; at other times the fat floats on the surface, forming oily drops, which adhere to the sides of the chamber. The hard masses of fæces are coated with fat on their surface" (Français).

Gaultier has shown that stearrhœa is of the highest importance. By quantitative analysis of the fat contained in the fæces, he has found that in cases of pancreatic deficiency the utilization of the fat (apart from the emulsified fats) is lowered to the extent of 15 per cent. ; and he has demonstrated by qualitative analysis that the saponification of the fats is much decreased. The total utilization is even smaller when both biliary and pancreatic deficiency are present.

Gaultier has described his technique ("Précis de Coprologie Clinique"; Paris, 1907), and the value of his methods has been repeatedly proved in my clinic at the Hotel-Dieu.

Another and, perhaps, as precise a method has been extolled by Carnet. It consists in enclosing salol, or methylene blue, in wax or in keratine, substances which are attacked only by the proteolytic ferment of the pancreas. The absorption of one of these pills and the ultimate finding of the salol in the urine, by means of perchloride of iron, furnishes one of the simplest and most exact of methods.

Let us now consider the question of prognosis. The association of pancreatitis with biliary lithiasis is sometimes serious, from the progressive course of these cases.

Chronic pancreatitis affects the excretion and the secretion of the pancreatic juice—the excretion by blocking Wirsung's duct ; the secretion because the fibrosis spreads to the glandular elements throughout the organ. The pancreatic deficiency, together with the absence of the bile, forms the pancreatico-biliary syndrome. Permanent jaundice and stearrhœa are the cardinal symptoms. We find also gastro-intestinal symptoms : anorexia, vomiting, colic, and offensive diarrhœa ; glycosuria is very rare ; fever is not constant ; hæmorrhages sometimes occur. The patient becomes cachectic, and the case resembles cancer, the more in that the nature of the pancreatic tumour is often wrongly diagnosed. This condition ends in death.

The tumour formed by pancreatitis exuberans may give rise to complications, which also lead to errors in diagnosis. Thus, when the duodenum is compressed, the symptoms are those of pyloric stenosis.

**Treatment.**—Surgical intervention is the only effective method of dealing with these cases. A patient has been subject for a variable period to attacks of cholecystitis, hepatic colic, and jaundice. In spite of medical treatment, the symptoms have shown no improvement. The jaundice is often permanent, the wasting is marked, the stearrhœa is quite evident, and analysis of the fæces shows pancreatic deficiency. In such a case early operation is advisable ; nothing is gained by delay. To wait too long is to expose the patient to the risks of acute pancreatitis or to pancreatico-biliary cachexia.



The operation best suited to these cases provides for the removal of the gall-bladder, in which gall-stones are formed and biliary infection arises. It allows the exploration of the large ducts and the removal of the calculi present in them, and renders possible free drainage of the hepatic duct, and diverts the bile. After some weeks this diversion of the poisonous bile leads to remarkable results.

The common duct again becomes permeable, so that the bile can pass freely into the bowel. The signs of biliary and pancreatic retention disappear. The jaundice fades away, the urine is no longer loaded with bile pigments, the fæces become normal, and in a few weeks or months the patient regains the weight lost, and complete recovery is assured.

The fibrous lesion in the pancreas retrocedes, the tumour disappears, and recovery follows when free drainage is given for the poisonous bile, which is capable of keeping up the cirrhosis of the pancreas.

#### CYTOSTEATONECROSIS—PANCREATICO-PERITONEAL HÆMORRHAGES.

**The Pancreatic Drama.**—I have so far considered chronic pancreatitis associated with cholelithiasis, and ending in the pancreatico-biliary syndrome. Fatal mischief may, however, occur suddenly in these cases, even during an apparently quiet stage, and in the absence of jaundice. The patient is seized with acute pain in the umbilical, epigastric, or hypochondriac regions. The suffering is terrible, and is accompanied by vomiting, prostration, and syncope. The abdominal hyperæsthesia is general, and the constipation is absolute. Under these circumstances it is natural to think of acute peritonitis, poisoning; perforation of the stomach, duodenum, or gall-bladder; appendicitis, or intestinal obstruction. None of these conditions are the cause of this episode, which I have called “the pancreatic drama,” in order to distinguish it from other conditions. And, either at the operation or at the autopsy, we find none of the lesions above mentioned, but the signs of this pancreatic drama: white islets (candle-grease spots) of fatty necrosis, which are often associated with pancreatico-peritoneal hæmorrhages, these lesions being due to an attack of acute pancreatitis, which is almost always grafted on chronic inflammation of the pancreas.

Cholelithiasis is not indispensable to the evolution of these troubles, but I shall in this section limit my remarks to cases in which biliary infection due to calculi and pancreatitis are associated. Before describing the clinical features of the pancreatic drama, it will be well to study the chief signs—cytosteatoncrosis and pancreatico-peritoneal hæmorrhage.

**Cytosteatoncrosis.**—We always find, either at operation or autopsy, in the peritoneal cavity white islets, resembling candle-grease spots. They project slightly, are about as big as a pin's head, and may be discrete or

confluent. At the post-mortem examination we find the spots in the pancreas, omentum, mesentery, appendices epiploicæ, the subperitoneal fat, and even in the mediastinum. The appearance of these white spots is most characteristic.

The condition is due to necrosis of the adipose tissue. I have called it "cytosteatonecrosis," for reasons to appear later. How are these white spots produced? Hallion has shown that two stages occur—steatolysis and steatonecrosis. The former consists "in the splitting up of the fat droplet in the adipose cell, either into fatty acid crystals or into insoluble soaps." The fatty acids form, in the cell, crystals, having the shape of fine-pointed tufts, or, by their union with lime salts, an insoluble soap, filling the cells in a homogeneous mass.

The fat of the cell, then, is converted into crystals of fatty acid or into insoluble soap. Another feature, just as important, in my opinion, is the disappearance of the cell nucleus. For this reason I have proposed the term "cytosteatonecrosis."

Histological sections show clearly the details of this process. They are well shown in the following figure:

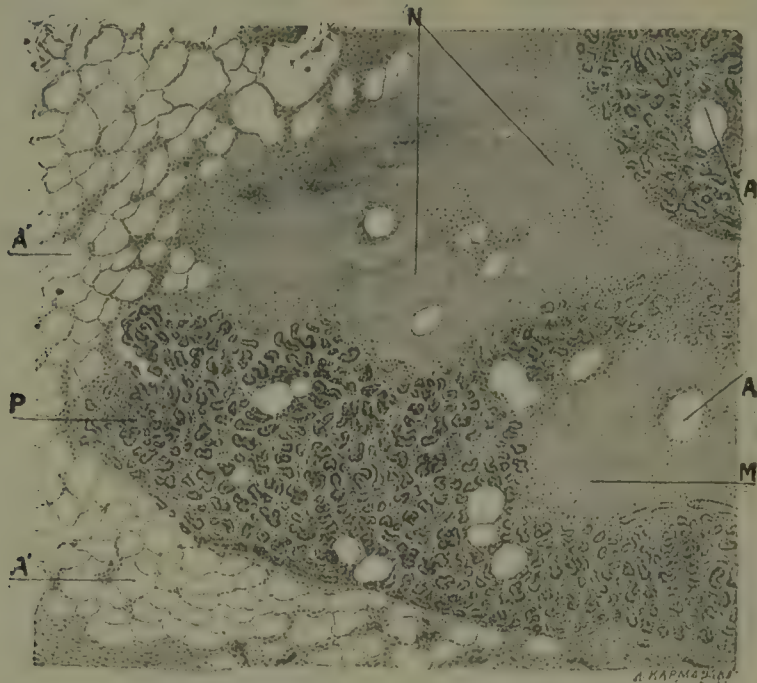


FIG. 54.—CYTOSTEATONECROSIS.

*A*, Normal intralobular fat cells; *A'*, normal peripancreatic fat cells; *N*, necrotic fat cells; *M*, zone of peri-adipose necrosis in the pancreatic tissue; *P*, healthy pancreatic acini. (Letulle.)

The steatolysis and cytosteatonecrosis are due to extravasation of the pancreatic juice, the steapsin having the power of splitting up the fat into fatty acids and glycerin. Both these processes have been experimentally produced in animals (Opie, Langerhans).

What causes this diffusion of the pancreatic juice into the peritoneal cavity in cases of pancreatitis? How does the juice provoke cytosteato-



necrosis in the deep parts of the tissues? Why does the cytosteatonecrosis affect the fat in the thoracic cavity? An answer is difficult.

Upon what does the extreme gravity of cytosteatonecrosis depend? Death is not due to the fact that many islets are scattered through the pancreas and the peritoneal fat. On the other hand, death is not due to peritonitis, for there is no evidence of peritonitis, and we do not find polynuclear cells in the foci of cytosteatonecrosis.\*

The cause of death has been put down to the action of the pancreatic juice upon the solar plexus. Another theory is that death is due to absorption of the diffused trypsin. At all events, we can say, from the clinical point of view, that the finding of "candle-spots" during the operation renders the prognosis very grave.

**Pancreatico-Peritoneal Hæmorrhage.**—In the pancreatic drama cytosteatonecrosis is not the only lesion. We also find hæmorrhage into the pancreas and the different regions of the peritoneal cavity. In many cases we find cytosteatonecrosis alone, but pancreatico-peritoneal hæmorrhage is always accompanied by cytosteatonecrosis.

On opening the peritoneal cavity, bloody fluid often flows out. Circumscribed or diffuse, superficial or deep, foci of hæmorrhage may be found in the pancreas, the subperitoneal tissue, the omentum, the mesentery, the fatty capsule of the kidney, etc. The condition is one of hæmatomata in all these parts.

I have called these hæmorrhages "pancreatico-peritoneal" in order to distinguish them from certain hæmorrhages in the pancreas, associated with suppuration, gangrene, or calculi, and described under the general term "hæmorrhagic pancreatitis," which includes every variety.

What is the explanation of these hæmorrhages? They are not due to any infective process, because no evidence of infection is found. The mischief arises from the action of the trypsin in dissolving the proteids and the walls of the vessels.

\* This does not exclude the possibility of the presence of infection in the lesions under discussion. In some cases we find suppurative pancreatitis and abscesses, especially in the head of the pancreas. In Quénu and Duval's paper I have found 12 cases of suppuration in 114 cases of pancreatitis associated with biliary lithiasis. The calculi were situated in Vater's ampulla, the gall-bladder, and the head of the pancreas. In these twelve cases the suppuration coincided three times with cytosteatonecrosis alone and three times with hæmorrhage and cytosteatonecrosis. The six patients, suffering from suppuration, hæmorrhage, and cytosteatonecrosis, died, whether they were or were not operated upon; the six patients who suffered from cytosteatonecrosis alone, with suppuration, were operated upon, four recovering.

Gangrene of the pancreas is very grave. The death of tissue, which may be rapid and extensive, is probably due to the action of anaerobic micro-organisms. Gangrene usually complicates the suppurative process.

The result is that the steapsin and the trypsin pass into the peritoneal cavity, and carry on a pathological digestion of the fatty tissue and of the vessels of the pancreas, the peritoneum, and its adnexa. The noxious action of the pancreatic ferment produces necrosis of the fat cells and of the vessel walls, whence arise the cytosteatoncrosis and the hæmorrhages. The real cause of the gravity of the prognosis and of death is not absolutely cleared up.

**Cases.**—After the discussion of cytosteatoncrosis and hæmorrhage—the two anatomical signs of the pancreatic drama—I will quote cases of cytosteatoncrosis, either occurring alone or in association with hæmorrhage. Wiener has collected twenty-six cases in which cytosteatoncrosis occurred without hæmorrhage.

**THAYER'S CASE.**—A man had suffered for sixteen months from hepatic colic with jaundice, when acute peritonitis apparently supervened. Acute pains in the epigastrium and left hypochondrium; collapse and death in thirty-four hours. Post-mortem: no peritonitis; whitish islets of fat necrosis, resembling spots of candle-grease. Pancreas enlarged. Over one hundred calculi in the gall-bladder, and a gall-stone blocking the terminal portion of the common duct.

**FRAENKEL'S CASE.**—A woman forty-eight years of age, who had suffered from hepatic colic and jaundice, was suddenly taken ill with acute pain, vomiting, and meteorism simulating peritonitis from perforation. Death in five days. Post-mortem: no peritonitis, but whitish islets of fat necrosis around the pancreas and in the omenta. Stones in the gall-bladder, and one at the ampulla of Vater.

The pancreatic lesion, accompanied by islets of steatoncrosis, is nearly always fatal: in twenty-six cases collected by Wiener, only one recovered, thanks to operation. A woman was suddenly seized with acute epigastric pain, radiating into the hypochondriac regions and the back. Nausea, but no vomiting; constipation. Two days later the pains were more severe; the whole abdomen was hyperæsthetic; the face was pale and anxious; the pulse was 125, and the temperature 101.5° F. Peritonitis, cholecystitis, and appendicitis were all thought of; the operation revealed whitish islets of cytosteatoncrosis in the omentum; peritonitis and appendicitis were absent. The pancreas was indurated. Cholecystitis was found, and the cystic duct was blocked by a large stone. The gall-bladder was removed; the bile-ducts were explored, and the hepatic duct was drained. The symptoms rapidly improved, and the patient was well in eighteen days.

I shall now give a summary of cases in which pancreatico-peritoneal hæmorrhage was associated with cytosteatoncrosis. Most of these cases are labelled "acute hæmorrhagic pancreatitis." More careful inspection shows, however, that the acute attack supervened in the course of chronic pancreatitis.

**DOBREAUEUR'S FIRST CASE.**—A man with slight jaundice was seized during the night with very acute peri-umbilical pain, followed by bilious vomiting and complete arrest of fæces and flatus. Pulse small and irregular; temperature normal. Diagnosis: *intestinal obstruction*. Operation: no obstruction; hæmorrhages in the neighbourhood of the pancreas. Death. Post-mortem: whitish islets of cytosteatoncrosis in the sub-peritoneal tissue; hæmorrhages in the pancreas; gall-stones in the gall-bladder and the common duct.



DOBREAUEUR'S SECOND CASE.—A man had acute abdominal pain, most marked at the umbilicus. Vomiting and symptoms of *intestinal obstruction*. Operation: no obstruction; islets of cytosteatonecrosis in the great omentum; pancreas enlarged. He died next day. Post-mortem: subperitoneal hæmorrhages and islets of cytosteatonecrosis; pancreas enlarged, hard, and infiltrated with foci of fat necrosis; hæmorrhages around the pancreas; gall-stones in the gall-bladder and the common duct.

DOBREAUEUR'S THIRD CASE.—A man was taken ill with violent abdominal pain and repeated attacks of vomiting. Epigastrium distended and painful. Pulse quick; temperature 103° F. Diagnosis: acute hæmorrhagic pancreatitis. Operation: omentum crammed with foci of cytosteatonecrosis; pancreas enlarged. Death from broncho-pneumonia. Post-mortem: cytosteatonecrosis in the great omentum and the transverse mesocolon: pancreas surrounded by hæmorrhage; a hundred small stones in the gall-bladder.

HALSTEAD'S CASE.—A man was taken ill with acute abdominal pain and nausea. During the next few days the epigastrium was very tender; gall-stones suspected. Operation: on opening the belly, blood-stained fluid flowed out, and numerous islets of cytosteatonecrosis were seen. The tissues around the pancreas were infiltrated with blood. Patient died. The autopsy showed hæmorrhages in the pancreas, which was enlarged. Disseminated islets of cytosteatonecrosis in the fatty tissue of the peritoneal cavity. A small gall-stone was found in the ampulla of Vater.

BORG AND EHRENPREIS'S CASE.—A young woman was admitted to hospital for appendicitis. She had been seized on the previous evening with acute abdominal pain and vomiting. Pulse almost imperceptible. Marion operated. On opening the abdomen, blood-stained fluid escaped. Appendicitis was absent, but a part of the omentum covered with islets was revealed by the incision. Marion then diagnosed pancreatitis. Death. Post-mortem: white islets of cytosteatonecrosis present in the omentum, the mesentery, and even in the subpleural tissues. The head of the pancreas, which was adherent to the neighbouring organs, was hard and much enlarged; the tail of the organ was replaced by a blackish sanguinolent pulp. The gall-bladder contained six calculi. In short, acute hæmorrhage and cytosteatonecrosis in the course of chronic pancreatitis.

BUNTING'S CASE.—A man was suddenly taken ill with acute epigastric pain and ballooning of the abdomen. Collapse soon followed. The operation showed the absence of intestinal obstruction, which had been suspected. Death. Post-mortem: islets of cytosteatonecrosis in the base of the mesentery; pancreas enlarged, studded with hæmorrhages, and affected with interstitial sclerosis; calculi present in the gall-bladder, the cystic duct, and the ampulla of Vater.

LEGUEU'S CASE AND LAUNAY'S CASE.—These two cases are absolutely remarkable. I owe them to the kindness of their authors, and have published them in my clinical lectures at the Hôtel-Dieu.

In the above cases the patients all had gall-stones. These cases show the gravity of pancreatitis with steatonecrosis and hæmorrhage.

Out of thirty cases in Lenormand and Lecène's paper only six recovered, thanks to early operation. I give a résumé of one of these cases:

A man who had had biliary colic some years before was suddenly seized with acute abdominal pain, vomiting, and absolute constipation. The belly was tympanitic; the epigastric region was distended and extremely tender. Diagnosis: necrosis of the pancreas, or perhaps peritonitis from perforation or acute intestinal obstruction. A supra-umbilical incision gave exit to abundant bloody fluid. A second incision below the umbilicus gave exit to similar fluid. Numerous islets of cytosteatonecrosis were present in the appendices epiploicæ, the mesentery, and the parietal peritoneum. Drainage

through both incisions. Two days later the patient had a stool, the pains disappeared, and recovery followed.

**Symptoms—Diagnosis—Treatment.**—I shall not dwell long on the symptoms and diagnosis of the pancreatic drama, as I have mentioned them throughout the course of this section. At first sight the diagnosis surely tends to the hypothesis of acute peritonitis, with or without perforation, or to that of acute obstruction. The sudden and acute abdominal pain, the vomiting, and the collapse, may help in making a diagnosis. We must, however, bear in mind pancreatitis, carefully inquiring for any previous history of gall-stones, jaundice, and hepatic colic. We must also define the maximum point of the pain and muscular rigidity, which in pancreatitis is a little above and to the right of the umbilicus.

Even when the diagnosis is doubtful, we must remember early surgical intervention. We risk, it is true, an error in diagnosis; but what does an error matter if the patient's life is saved? And even if we have wrongly suspected peritonitis, perforation of a viscus, appendicitis, or acute obstruction, the mistake will not harm the patient, because operation is indicated in all these cases. In the pancreatic drama the patient's only hope lies in operation.

Furthermore, the operation at once gives the diagnosis. The bloody fluid and the candle-grease spots in the omentum or elsewhere show that the pancreas is affected. It is, then, necessary to drain the pancreas and the peritoneal cavity. Timely operation gives an excellent chance of success.

#### REFLECTIONS ON CANCER OF THE PANCREAS AND OF VATER'S AMPULLA AND PANCREATIC LITHIASIS.

Whatever may be the part played by biliary lithiasis and the part played by pancreatitis, it is certain that the association of these two factors favours the appearance of the pancreatic drama.

I have asked myself whether lesions of another kind, ending in retention of bile and of pancreatic juice, might not favour the pancreatic drama. Take, for example, cancer of the head of the pancreas. The almost constant obliteration of the common duct, the fairly frequent obliteration of Wirsung's duct, and the resulting pancreatco-biliary syndrome, being given, it is reasonable to suppose that such ætiological conditions might be propitious to the production of the pancreatic drama. Nothing of the kind happens. I have collected forty-five cases of cancer of the head of the pancreas, verified by post-mortem examination, and yet the pancreatic drama never appeared.

The same remark applies to cancer of Vater's ampulla. This lesion often provokes obliteration of the common bile-duct and of Wirsung's canal, in much the same way as does impaction of a gall-stone. In both cases we find



retention of bile and pancreatic juice ; in both cases the pancreatico-biliary syndrome is seen. In the eleven cases of cancer I have collected, post-mortem examination did not show cytosteatonecrosis or pancreatoperitoneal hæmorrhages. The patients died from the cancer, and not from the pancreatic drama. In these cases neither stenosing nor hypertrophic pancreatitis was seen.

As regards pancreatic lithiasis, I have been somewhat surprised to find that pancreatic calculi, obliterating Wirsung's duct, and causing retention of bile and of pancreatic juice, produce neither hypertrophic nor stenosing pancreatitis. Islets of cytosteatonecrosis and pancreatoperitoneal hæmorrhages do not occur in these cases.

Jaundice is mentioned in three cases of pancreatic calculi. In Lazarus' case a calculus was found in the ampulla of Vater. In Gould's case the operation showed calculi in the head of the pancreas and in Vater's ampulla.

In some cases of pancreatic calculi, steorrhœa, intact muscular fibres, and crystals of fatty acids have been found in the stools. This fact merely shows some degree of pancreatic insufficiency.

In a case of pancreatic lithiasis reported by Von Simpson, the symptoms and lesions of the pancreatic drama are mentioned. The patient was taken ill with violent pain in the belly and constipation ; he died three days later. The autopsy showed pancreatic lithiasis and foci of cytosteatonecrosis in the mesentery and the appendices epiploicæ.

The interesting point is that the gall-bladder was full of calculi.

In all the cases I have been able to collect of cancer of the head of the pancreas or Vater's ampulla and of pancreatic calculi, I have only found one case in which the pancreatic drama appeared, and even in this case gall-stones were found in the gall-bladder.

END OF VOL. I.

















